PEPTIC STRicture OF THE OESOPHAGUS

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1971.
This Thesis is a clinical, illustrated account of peptic stricture of the oesophagus. It is based on 146 cases. Additional cases are introduced where they illustrate points in differential diagnosis. The text is illustrated by 154 radiographs, photographs of pathological specimens, and diagrams.

The age and sex incidence and the symptomatology are dealt with.

The radiological and oesophagoscopic appearances are described with special reference to the type of stricture and its treatment.

The association of peptic stricture with other lesions is commented upon.

Features of the differential diagnosis are illustrated with special reference to lower oesophageal rings.

A series of 18 patients with post-operative and post-recumbency strictures is detailed and a view as to their aetiology expressed. The relationship of post-vagotomy dysphagia and post-operative peptic stricture is discussed.

A section is devoted to high peptic stricture and lower oesophagus lined by columnar epithelium. Cases are described which have a bearing on the aetiology of the condition. Comment is made on a number of associated congenital anomalies.

The pathology of resected specimens is illustrated.

The natural history of peptic stricture is discussed in relation to treatment.

Various forms of treatment are discussed with emphasis on conservative management and hernial repair.

A novel feature is an exploration of the importance of the contraction of the oesophageal muscle, especially the longitudinal muscle.
Circumferential oesophageal myotomy is described. Its aims are to render the hiatal hernia more easily reducible in the presence of mild shortening of the oesophagus and to reduce the incidence of recurrence. A series of post-operative radiographs is included showing the characteristic and novel radiological appearances after this myotomy. It is considered that the rationale of circumferential myotomy is a new concept, the application of which may be of value.
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INTRODUCTION
The aim of this Thesis is to present a clinical picture of peptic stricture of the oesophagus as it is seen in a Thoracic Surgical Unit in a non-teaching hospital serving an industrial community in the north of England.

The term "peptic stricture" is used as we believe that the lesion is due to acid-pepsin digestion of a vulnerable oesophageal mucosa. Forty years ago Jackson mentioned "retrograde flow of gastric juice" as a cause of the perpetuation of an acute ulcer (Jackson 1929). As possible causes of this retrograde flow he listed malfunction of the stomach, hour-glass deformity, pyloric stenosis, hepatic disease, and duodenal and other intestinal disorders but did not recognize the significance of hiatal hernia. With the wider recognition of the importance of gastro-oesophageal reflux and what Barrett (1950) aptly called "reflux oesophagitis", the effect of gastric juice on the oesophageal mucosa has been generally accepted as the cause of peptic stricture and oesophagitis. There are those who do not agree. Palmer (1968) summarised his views as follows ..."this is not primarily a disease of the mucosal surface but a deeply intramural one. Briefly, there is subacute inflammatory reaction through the muscularis propria, the submucosa and the subepithelial layer, with variable extension up the rete pegs into the epithelium. This leads periodically to exfoliation of patches of superficial epithelium, forming multiple erosions and thus baring an extensive capillary bed for haemorrhage. The healing response within the oesophageal wall is marked by prominent fibrosis, and stricture is an occasional sequel". There are instances where the lesion has been attributed to alkaline digestion and the effect of bile salts on the oesophageal mucosa, particularly in the presence of pyloric incompetence (Cole, 1968) and after operations on the stomach.

As far as we are aware, none of the strictures in our series
can be attributed to what has been referred to as "stehosing nonpeptic oesophagitis (Reiss, Urea, and Nathan, 1963) where such causes as bacterial or viral infections, non-specific enteropathies, or vascular occlusion have been suggested.

The use of the term "peptic stricture" is not meant to imply that gastro-oesophageal reflux affords a complete explanation for the pathological changes in the oesophagus. Strictures having all the histological features of peptic strictures have been found in the presence of achlorhydria either naturally occurring or as a result of operations such as partial gastrectomy or vagotomy. While the view that peptic strictures are the result of reflux is supported by the fact that they are usually found in association with a hiatal hernia and that they may develop after operations which interfere with the valve mechanism at the cardia such as Heller's operation or oesophago-gastrostomy, this does not explain the variations in the response of the oesophageal mucosa to reflux. It remains a mystery why some patients with reflux are asymptomatic while others develop severe ulcerative oesophagitis and stricture. The occurrence of periodic variations in the severity of symptoms in any individual patient is also unexplained. The manner in which symptoms and indeed pathological changes are influenced by such factors as age and emotional states is "a mystery wrapped in an enigma". Do hormonal influences operate? We have described (Davidson, 1968) perhaps somewhat facetiously but with sincere bewilderment, some psychological aspects of hiatal hernia — "The oesophagus is a very emotional organ. The fat woman of 55 who has, as they say 'let herself go', who no longer feels needed by her grown-up family and her disinterested husband, may seek comfort from the priest, the psychiatrist, the beautician, or the surgeon. We claim cure of heartburn by repair of a hiatal hernia when all we may have done is to
provide circumstances in which a woman who feels neglected may again
become an object of sympathy. We accept blame for failures when the
symptoms are, in fact, related to business worries or domestic dis-
harmony. The dermatologist must be a psychologist and so must the
thoracic surgeon. It is very difficult to know when an oesophagus is
being digested by acid and when it is blushing for shame, pining for
love, quivering with anxiety, desolate with grief, or merely reflecting
downward trends on the stock market". Such observations are not help-
ful but serve to emphasize the obscure link between the emotions and
oesophageal symptoms and disorders.

It is disturbing to find how often armchair theories of the
aetiology receive little support from the laboratory bench. In a
thought provoking paper, Collis (1970) comments on various features
which, in theory, would seem to be inevitably related to the response
of the oesophagus to gastric reflux but whose relevance is difficult to
substantiate on more careful scrutiny. He found that while, in general,
the degree of response is proportional to the amount of reflux, there
is no correlation between the volume of reflux and the type of response.
As far as the level of gastric acidity is concerned, conflicting views
have been expressed. It has been claimed that patients with symptomatic
hiatal hernia have an increased gastric acid secretion (Casten et al.
1963, and Casten, 1964 and 1967). Others have found no close relation-
ship between gastric acidity and symptomatic hiatal hernia (Williams et
al. 1967; Squire et al. 1968). Collis found no definite association
between the presence of other lesions such as gallstones, duodenal and
gastric ulcer and symptomatic hiatal hernia and quotes the similar
findings of Palmer. Olsen and Schlegel (1965) considered that poor
peristalsis was associated with oesophagitis. Prolonged acid clearing
time from the distal oesophagus has been found in association with
symptomatic hiatal hernia (Booth et al. 1968). On the other hand, Collis is of the opinion that the clearance time of the lower oesophagus has no aetiological significance in the type of oesophageal response produced.

In the case of a lesion where the precise aetiology remains so much of a mystery, there is still a place for the accumulation and recording of clinical details which is what this Thesis sets out to do. Our cases range over a period of 15 years but the vast majority were seen in the last decade. d'Abreu (1963), referring to peptic stricture, said that "this condition which is being increasingly referred for treatment, exceeds in importance the management of carcinoma of the oesophagus". After earlier reports of sporadic cases and small series, a vast literature has grown up over the past two decades. In spite of this and the general experience of increasing referrals to hospital of patients with peptic stricture, there is, at the moment, no real evidence to prove that the incidence is increasing and the apparent increase is assumed to be due to improved diagnostic methods and increased awareness of the condition.

Included in this Thesis is a description of circumferential oesophageal myotomy which we believe to be a new surgical procedure. Whether or not this operation will prove to be of value in the treatment of peptic stricture remains to be seen.
MATERIAL
This account of peptic stricture of the oesophagus is based on 146 cases. Ten were children under the age of 12 years. Thirteen were seen between 1954 and 1959 and the remainder between 1960 and 1969.

Case reports of patients not suffering from peptic stricture are introduced where they illustrate points in differential diagnosis.

The section dealing with circumferential oesophageal myotomy is based on 24 patients on whom the operation has been performed. Of these, 13 are included in the series of 146 and 11 are additional patients who are not included in the general series.
DEFINITION
It is necessary to define what is meant by the term "stricture". In the presence of gastro-oesophageal reflux, the state of the oesophagus may range from one of normality to one of ulceration extending through all coats and into the peri-oesophageal tissues. In every patient included in this series all the following criteria had to be met.

1. The patient had to have dysphagia. This alone was not considered sufficient reason for inclusion as a sensation of difficulty in swallowing may be felt with only mild, superficial oesophagitis or in the presence of an uncomplicated hiatal hernia.

2. There had to be radiological evidence of stenosis. This had to be constant so as to exclude persistent spasm as far as possible. In the more recent patients it has been shown that the lesion did not respond to antispasmodics.

3. There had to be oesophagoscopy evidence of stricture. The oesophagoscope was, in all cases, held up at the stricture whose presence was further confirmed by the passage of bougies.
SECTION 1
AGE INCIDENCE

The age range was from 2 weeks to 89 years. The average age of adult patients was 63 years. The number of patients in each decade is shown in Table 1.

TABLE 1

<table>
<thead>
<tr>
<th>AGE</th>
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<tr>
<td>0 - 10</td>
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<td>11 - 20</td>
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The ages recorded are at the time of first attendance at hospital and are not necessarily an accurate reflection of the time of development of the stricture. In the patients in the second, third and fourth decades, there was sometimes a long history of mild dysphagia suggesting that some degree of oesophageal stenosis had been present since infancy. In the older age groups, the duration of dysphagia was variable, ranging from a few hours in patients presenting with food impaction to periods of as long as 15 years. The figures, therefore, reflect the time of life at which the dysphagia became severe enough to induce the patient to attend hospital rather than the time of onset of the stenosing process in the oesophagus.
In men under the age of 50, an important cause of peptic stricture was an abdominal operation, usually for duodenal ulcer. This will be dealt with in Section 11.

It is not possible to get accurate figures for the age incidence of hiatal hernia. Hernias exist in many asymptomatic persons. Many series of hiatal hernias are surgical ones and are, therefore, selected groups. Study of the literature suggests that the highest incidence of hiatal hernia is in the fifth decade. In our series, the highest incidence of peptic stricture was in the sixth decade. Not only is the peak incidence of peptic stricture 10 years later than that of hiatal hernia but the incidence of stricture remains high in the seventh decade. In most surgical series of hiatal hernia, the incidence has dropped to a low figure by the seventh decade, probably because patients of this age are less frequently referred for a surgical opinion. Burdette Nelson (1969) found that his patients with peptic stricture were, on average, fifteen years older than a comparable group of patients with symptomatic hiatal hernia and oesophagitis. In our series, 76 out of 134 adults were between 61 and 80 years of age. Not only was the incidence high in the older age groups but many of the more intractable strictures occurred in the older patients. Such observations suggest that advancing age has an influence on the response of the oesophagus to gastro-oesophageal reflux. Weber (1969) makes this point when he says that "acid peptic insult of the lower oesophagus often goes on for years without producing much gross change and then with age mucosal resistance weakens leading to oesophagitis and stricture."

According to Sandry (1962), the pathological type of chronic oesophagitis is also linked with the patient's age. Our pathological findings which are largely in agreement with those of Sandry will be
dealt with in Section 13.
**SEX INCIDENCE**

The sex incidence is shown in Table II.

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In children up to the age of ten years there was a preponderance of males. The numbers are small. However, Skinner and Belsey (1967) in a series of 119 children under the age of twelve years with hiatal hernia also found a male preponderance (68 per cent). As has been stated, some of the strictures found in patients under the age of forty may have originated in childhood. The sex incidence in these age groups is probably influenced by that found in infancy. Another factor influencing the sex incidence in this series was that all the patients who developed a peptic stricture after an operation for duodenal ulcer (six) were men.
In most series of symptomatic hiatal hernia there is a substantial preponderance of females. In a consecutive series of 136 sliding hiatal hernias operated on by us there were 95 females and 41 males.

In the patients with peptic stricture between the ages of 51 and 70, females outnumbered males in the proportion of 48 to 31. On the other hand, in the age group 71 to 80 the sex incidence is equal (17 men to 18 women). The numbers are small but it is tentatively suggested that in men over the age of 70 there is, for some unknown reason, a relative increase in the vulnerability of the oesophageal mucosa to gastro-oesophageal reflux.

In view of the over-all preponderance of woman, it may be of significance that, of the patients requiring resection of the stricture, in itself an indication of the severity and intractability of the lesion, there were more men than women (12 men to 9 women).
SECTION 3
SYMPTOMS

The essential symptom of peptic stricture of the oesophagus is dysphagia. This is often characteristic of the lesion and its particular features are of value in the differential diagnosis.

Previous history of heartburn or indigestion

A majority of patients who present with dysphagia due to a peptic stricture give a previous history of heartburn. In this series 84 adult patients (62 per cent) were recorded as having suffered from heartburn. In the remainder no specific mention was made of a previous history of dyspepsia. Previous indigestion, may, of course, not be attributable to oesophagitis in view of the frequency of associated gastric or duodenal ulcer or disease in the biliary tract.

The patient may find that heartburn ceases after the onset of dysphagia. Specific mention of this feature was made in 13 patients (9.5 per cent). In the majority, however, heartburn persists after the onset of dysphagia.

Some patients give a history of what they describe as "bilious attacks" in adolescence. Such attacks are often associated with prostration and with headache of migrainous type. They tend to disappear after adolescence, the patient remaining free from symptoms until heartburn develops in middle age.

Mode of onset of dysphagia

The commonest mode of onset is a gradually increasing difficulty in swallowing solid food. The dysphagia increases in severity for a time and may then remain much the same over long periods with only occasional exacerbations. Some patients experience a more rapid onset of dysphagia which is severe and persistent from the beginning. This type of onset is particularly common in the case of peptic strictures occurring after a sudden haematemesis, after an
abdominal operation, or after parturition where the pregnancy has been complicated by hyperemesis gravidarum (Abbey Smith, and Nelson, 1965).

Many patients experience a sudden onset of dysphagia which they relate to one particular meal. After the initial aphagia the bolus usually passes or is regurgitated and the subsequent dysphagia may be quite variable in degree and not necessarily severe.

In babies, regurgitation is the prominent feature and recognition of dysphagia is delayed until weaning is begun.

**Features of the dysphagia**

A common history, characteristic of peptic stricture, is of difficulty in swallowing the first mouthfuls of a meal, especially the first mouthfuls of food in the morning. After food, mucus, and saliva have been regurgitated, the dysphagia is relieved and the patient can go on and finish the meal. The patient may have a feeling of food passing through a raw, painful part of the gullet.

In 9 patients, episodes of severe dysphagia were associated with hiccups. In 5 patients, hiccups were initiated by the swallowing of food and, in 4, a bout of hiccups immediately preceded the onward passage of an impacted bolus.

Patients often volunteer the information that their dysphagia is worse if they are emotionally upset. They swallow best when they are at home where they can concentrate on the act of swallowing, eat at their own speed, and confine themselves to food to which they are accustomed.

**Duration of dysphagia**

The duration of dysphagia before the patient is first seen at hospital is very variable. Patients who, in spite of having dysphagia in infancy and childhood, survive into adult life give a
long history of dysphagia. In this series there were 4 such patients who had suffered from dysphagia for periods of 12, 17, 34, and 42 years respectively. On the other hand, the shortest history of dysphagia occurs in those patients in whom an episode of food impaction is the first indication of the presence of a stricture. Measurable in weeks or a few months at the most is the dysphagia due to an abdominal operation or a period of enforced recumbency. This leaves the majority of patients in whom, in this series, the average duration of dysphagia before they attended hospital was 2 years.

**Food Impaction**

As peptic stricture is compatible with life over long periods and as the stenosis is associated with additional factors such as spasm and exacerbations of oesophagitis, food impaction is a more prominent feature in the history of peptic than of malignant stricture. Food impaction occurred on one or more occasions in 14 per cent of patients. In 4 patients food impaction was the first indication of the presence of a stricture. In only 3 patients was oesophagoscopy necessary for the removal of the impacted bolus. The patient may find ways of dislodging the bolus. One man who had repeated bolus obstructions found that he could induce the impacted food to pass on by raising his arms and taking deep breaths. When the bolus is regurgitated, it is often accompanied by some bright red blood. Haematemesis of this type is of small amount and is transitory.

As has been said, a patient with peptic stricture swallows best when eating quietly at home. It is not surprising that food impaction is most likely to occur when he is eating out or on some festive occasion. The act of swallowing in the presence of a peptic stricture requires concentration. As one patient put it, her food got stuck in her gullet when she "was not thinking".
Unless disimpaction of the bolus occurs within a short time acute dehydration develops. Elderly patients may present in a state of peripheral circulatory failure. Removal of the bolus through the oesophagoscope becomes a matter of urgency but in an aged patient with cold, cyanotic extremities it is best to delay oesophagoscopy until rehydration by intravenous fluid has been achieved.

**PAIN**

Pain, as distinct from heartburn, was complained of by 27 patients (20 per cent). It may be of the type usually associated with oesophagitis, the patient experiencing much discomfort as the food passes the "narrow bit" in the oesophagus. Pain may be distressing during episodes of food impaction.

Radiation of the pain to the back is often an indication of the presence of a penetrating ulcer at the site of stricture. In 2 adult patients, pain in the back was of such severity as to require excision of the lesion. An ulcer adjacent to the hiatal margin gives rise to pain along one or both costal margins (Davidson 1958). In cases where peri-oesophagitis involves the adjacent pleura, pain may be felt on one or both sides of the chest and in the shoulders.

In babies and young children, pain is readily overlooked, being overshadowed by the regurgitation and vomiting. It results in fretfulness and loss of sleep. One child, aged 18 months, who was always miserable and who had kept his parents awake at night for many months, became a happy and placid child after resection of an ulcerated oesophageal stricture. Another child, aged 8 years, who had lost sleep for a long time and who had radiological evidence of an ulcerated stricture, became good tempered and began to sleep well after repair of his hiatal hernia.
Haemorrhage and anaemia

Frank haemorrhage, as distinct from the insidious development of anaemia, occurred in 28 patients (20 per cent). It is not always possible to be sure of the site of origin of the bleeding as a number of patients with peptic stricture have a duodenal or gastric ulcer. In at least 6 patients it seemed more likely that the bleeding had arisen from a duodenal or gastric ulcer than from the stricture. When there is a fairly large hiatal hernia, bleeding may come, not from the stricture but from a gastric ulcer on the lesser curvature of the stomach where it lies against the anterior margin of the hiatus. Such an ulcer is readily overlooked on radiography (Davidson, 1958.). In one instance haemorrhage occurred while the patient was under treatment with anticoagulants.

In comparison with haematemesis from gastric or duodenal ulcer, bleeding from a peptic stricture tends to be less severe. In this series, blood transfusion for haematemesis was required in only 3 patients.

In adult patients, haematemesis was episodic with long intervals between the bleeds. In infants with ulcerative oesophagitis, however, bleeding was more persistent, the baby continuing to bring back blood-stained fluid and developing severe chronic anaemia.

The series includes patients in whom, at operation, a penetrating ulcer was dissected off the aorta and one patient in whom an ulcer had penetrated the pericardium. No gross fatal haemorrhage was encountered.

In 5 patients, all of whom had had heartburn alone for some years, haematemesis was followed within a few weeks by dysphagia due to a hard, irregular stricture. It seems probable that they had developed an ulcer which gave rise to the bleeding and to the subsequent
fibrotic stricture. In one of them the stricture had to be resected and a deep ulcer was found. This sequence of events is described by Self (1965).

In 4 patients a small haematemesis followed the regurgitation of an impacted bolus.

Some bleeding following dilatation of a peptic stricture is common but it is usually small in amount and of short duration. In only 1 patient was there significant bleeding after dilatation. This happened twice in the same patient and, on both occasions blood transfusion was required.

Chronic anaemia without overt bleeding and with haemoglobin levels of less than 80 per cent was found in 18 per cent of patients. In such cases, the type of stricture found at oesophagoscopy was usually soft, inflamed one. The mucosa above it bled readily with only slight trauma. Patients who were proved at operation to have a penetrating ulcer were invariably anaemic.

Chronic anaemia is a well known feature of uncomplicated hiatal hernia. Windsor and Collis (1967) found that, in patients with hiatal hernia, there is an over-all incidence of anaemia in 15 per cent and that it was three times more common with para-oesophageal hernia (30 per cent) than with sliding hernia (11 per cent). Their figure for the incidence of anaemia in "short oesophagus having gross bleeding oesophagitis" was 18 per cent which is the same as in our series of peptic strictures. They conclude that there is little correlation between the state of the lower oesophagus and the incidence of anaemia and, furthermore, that bleeding from the lower oesophagus is unlikely to be the cause of anaemia. However, in our view, their figures of 18 per cent anaemia in short oesophagus as compared with 11 per cent with sliding hiatal hernia alone seem significant. Almost all our
patients with stricture had a sliding hiatal hernia and the incidence of 18 per cent anaemia is also higher than Windsor and Collis's for sliding hernia alone. As has been said, all our patients with a penetrating ulcer were anaemic. We find it difficult completely to accept the view that there is little or no relationship between the state of the lower oesophagus and the incidence of anaemia when the oesophageal changes are severe enough to cause ulceration and stricture.

Respiratory symptoms

There are probably two mechanisms causing respiratory complications secondary to peptic stricture. In the presence of a stricture sufficiently tight to limit free reflux, there is retention of food, mucus, and saliva in the oesophagus. This material may spill over and cause "dysphagia pneumonitis" with radiological evidence of segmental inflammatory changes in the lungs. More common is the situation where, in spite of the stricture, reflux persists. In such circumstances, the regurgitated and aspirated fluid is acid and causes chemical laryngitis, bronchitis, and sometimes severe bronchospasm often with little or no radiological changes in the lungs. There are cases where both mechanisms operate.

In the present series various respiratory complications were encountered.

Aspiration pneumonitis. Figure 1 shows radiological appearances typical of this type of pulmonary lesion. The patient had a very tight stricture which had to be resected as it could not be dilated. After the operation there was rapid clearing of the pneumonitis. In adults, we did not find this type of lung infection to be common (approximately 4 per cent of cases). On the other hand pulmonary infection and atelectasis are much more frequently found in babies. In 10 children under the age of 12 years there was radiological evidence
Aspiration pneumonitis which failed to respond to treatment with antibiotics but which cleared up rapidly after resection of the peptic stricture.
of lung infection in 6. If the oesophageal lesion is missed or remains untreated in children the lung infection may progress to bronchiectasis. Lung infection with radiological changes is an indication for surgical treatment in infants and children. Resection of the stricture is usually necessary. In a very limited experience of this type of lung infection in adults with peptic stricture, we have found it associated with an oesophageal lesion requiring resection and have no evidence to indicate the efficacy of hernial repair or dilatation alone. While the pulmonary infection is controlled to some extent by antibiotics, dramatic improvement follows operation on the stricture.

**Bronchitis.** In the presence of dysphagia and radiological signs of pneumonitis, the pulmonary condition can be attributed with confidence to aspiration. When the lung condition is one of bronchitis without radiological signs, its aetiology is much less definite. Of 136 adult patients 11 were recorded as having bronchitis (8.1 per cent). This figure is no doubt quite inaccurate both because of deficiencies in the records and because of the lack of precise definition of bronchitis. The diagnosis was based on the presence of cough, sputum, and wheeze with an absence of radiological signs. In an industrial community where the incidence of bronchitis is high, it is difficult to establish its relationship to aspiration. Five severely bronchitic patients were operated on. One man, who had bilateral basal bronchiectasis, was no better from the point of view of his chest condition after repair of his hiatal hernia. Two patients in whom the peptic stricture was resected and where a segment of jejunum was interposed between the oesophagus and the stomach continued to have bronchitis. Two patients were less bronchitic after relief of their dysphagia following hernial repair.
Asthma. Two patients suffered from symptoms of asthma. One of them, a woman aged 51, had an ulcerated stricture 30 cms. from the upper incisor teeth, with free reflux through it. During the induction of anaesthesia for an oesophagoscopy, aspiration occurred provoking severe bronchospasm and bringing the patient near to death. At bronchoscopy, there was no solid material in either bronchial tree. Following repair of her hernia and control of reflux, her asthmatic symptoms disappeared. The other patient, a woman of 49, had a history of infantile eczema and a family history of asthma. Her asthma was not improved by excision of the stricture and interposition of a segment of jejunum. The disparity in the results of control of reflux in the two patients is presumably due to the fact that the second patient, judging from her personal and family history, was suffering from true allergic asthma.

Laryngitis. As in uncomplicated hiatal hernia, reflux in the presence of a peptic stricture may cause chemical laryngitis. Eight patients suffered from recurrent hoarseness of the voice. In 4 of them who were operated on and the reflux controlled, there were no further episodes of hoarseness.

Nocturnal choking. Aspiration, especially the aspiration of acid, may result in a mistaken diagnosis of paroxysmal nocturnal dyspnoea. The patient is awakened suddenly during the night with choking. He is in great distress and gasping for breath. By the time the doctor arrives, the acute episode is past and a history resembling that of cardiac asthma due to left heart failure is obtained. Such a mistaken diagnosis is of importance if it is considered to be a contra-indication to surgical treatment of the stricture.

Emphasis has been rightly placed on the respiratory complications of gastro-oesophageal reflux due to hiatal hernia. Urschell and Paulson (1967) attributed respiratory tract infection to aspiration
in as many as 50 per cent of 636 patients, adults and children. Skinner and Belsey (1967) attributed bronchopulmonary infection to aspiration in 8 per cent of 829 adult patients with hiatus hernia. They found the incidence in children to be much higher (19.3 per cent). Lilly and Randolph (1965) record aspiration pneumonia in 30 per cent of children with hiatus hernia. Our figures for pulmonary infection in adults with peptic stricture are comparable with those of Skinner and Belsey for cases of uncomplicated hiatus hernia suggesting that, in adults, the development of a peptic stricture does not significantly increase the incidence of pulmonary complications. Although the numbers are very small, our high incidence of lung infection in babies with peptic stricture (60 per cent), suggests that, as compared with the situation in adults, the development of a stricture in a baby is much more likely to lead to serious pulmonary complications.

Nutrition of patients with peptic stricture

A striking feature in patients with dysphagia due to a peptic stricture is that many of them do not lose weight. It is common in this condition for a fat woman to present with dysphagia and yet for her to remain overweight for many years. This is in sharp contradiction to what happens when the lesion is a malignant stricture.

Specific mention of weight loss was made in 18 adult patients (13.2 per cent). It is of interest that 7 of them had had a previous abdominal operation (partial gastrectomy, 2; vagotomy and gastro-enterostomy, 3; closure of perforated duodenal ulcer, 1; laparotomy for diverticulitis, 1). Of the remaining 11 patients who had lost a significant amount of weight, 5 were over 70 years of age. 5 were recorded as having soft, inflamed strictures and 6 as having tight, firm strictures. In 1 patient the weight loss was probably attributable to respiratory deficiency due to pneumoconiosis as well as to the
oesophageal stricture.

Babies who have severe oesophagitis or an ulcerated stricture fail to thrive. This failure of development is often of itself an indication for surgical treatment.

It would appear, therefore, that in most patients in the fifth and sixth decades weight loss is not a prominent feature of peptic stricture but that nutritional problems arise in infancy, in patients who develop peptic stricture after an operation for gastric or duodenal ulcer, and in patients over the age of 70 years.
SECTION 4
Cineradiography has not been available to us except in two cases in Section 13. The opinions expressed in this section are based on x-ray screening and a study of films.

Peptic strictures fall into groups depending on their relationship to the oesophago-gastric junction and their situation in relation to the hiatus and the aortic arch. We recognise the following groups, namely strictures at or near the cardia, high strictures near the level of the aortic arch, and a more indeterminate group lying roughly midway between the diaphragm and the aortic arch.

**Group 1 : Low Strictures at the cardia**

This is the commonest type (60 per cent). At thoracotomy such strictures are often found to be slightly above the gastro-oesophageal junction. It is difficult to be certain of the exact situation of the cardia from the external appearances of the oesophagus.

This group is subdivided into those strictures in association with a hiatal hernia and those where no hernia is demonstrable. This distinction is difficult to make by radiological methods alone and the position may be clarified only at operation. (Figure 2)

**Subgroup a. Strictures at the cardia with a hernia.**

This group comprised 84 per cent of strictures at the cardia. The average distance of the stricture above the cardia in adults as seen on x-ray was 5.6 cms, depending on the degree of slide of the hernia. This type of stricture is illustrated by the radiographs of four patients in whom the situation of the stricture was confirmed at thoracotomy (Figures 3 - 6).

**Subgroup b. Strictures at the cardia without a hernia.**

In 9 per cent of strictures at the cardia there was no apparent hernia. Low strictures without a hernia were found especially
GROUP I STRICTURES 60%

Group Ia
Stricture at cardia with hernia

Group Ib
Stricture at cardia without hernia

Figure 2

Figure 3

Figure 4
GROUP II STRICTURES 21%

Group IIa
High stricture with hernia

Group IIb
High stricture without hernia

Figure 7

Figure 8

Figure 9
High Strictures where no hernia was found at thoracotomy.
in the group of post-operative strictures.

In the remaining 7 per cent of strictures at the cardia it was not possible to decide whether or not there was a hiatus hernia.

**Group 2 : High stricture**

There were 31 patients in this group (21 per cent of the whole series). Such strictures are in the vicinity of the aortic arch. Several of them were level with the upper margin of the arch. No stricture at a level higher than this was encountered. The average distance above the diaphragm of this group of strictures was 16.7 cms. in adults.

As in Group 1 strictures, those in Group 2 are subdivided into those with and those without a hiatus hernia. (Figure 7) 20 patients (69 per cent) had an associated hernia. In 6 patients (20 per cent), there was no apparent hernia. In the remaining 3 patients, the presence of a hernia remained uncertain. It seems from the findings in Groups 1 and 2 that the absence of a hernia is more common in the case of high strictures than with strictures at the cardia. High stricture with a hernia is illustrated by the radiographs of 2 patients (Figures 8 and 9). High stricture without a hiatus hernia is illustrated by 2 representative cases (Figures 10 and 11). The presence or absence of a hernia was confirmed at thoracotomy in the cases illustrated.

**Group 3 : Strictures midway between the hiatus and the aortic arch**

Strictures at this level are considered as a separate group because of the difficulty in determining their relationship to the oesophago-gastric junction, a relationship which we believe to be important in connection with their surgical treatment. A stricture at the level of the aortic arch is always associated with a
long segment of gastric lined oesophagus. The lower oesophagus and the region of the cardia are soft and not inflamed so that hernial repair is usually relatively easy. In the case of a low stricture near the hiatus reduction of the hernia is again likely to be possible. It is with midway strictures that there is the greatest difficulty in deciding pre-operatively whether or not the hernia is reducible (Figure 12). The average distance of such strictures from the diaphragm as seen radiologically was 7 cms.

This group is also subdivided into two.

Subgroup a. Strictures which are in close relationship to the cardia but which are at a relatively high level through being associated with a hernia which has slid upwards a long way. In order to show the relationship of the stricture to the cardia it is essential to achieve complete filling of the hernia with barium. Two strictures of this type where their relationship to the cardia was confirmed at operation are illustrated (Figures 13 and 14).

Subgroup b. Strictures midway between the hiatus and the aortic arch where, radiologically, there is a segment of the calibre of the oesophagus and 2 to 3 cms. in length between the cardia and the stricture. Such lesions are illustrated by radiographs of 4 patients (Figures 15 to 18). We do not have a case of this type where the lesion has had to be resected so that full histological evidence is lacking. At thoracotomy for repair of the hernia we have found the stricture to be 2 to 3 cms. above the oesophago-gastric junction. Biopsies at oesophagoscopy have shown gastric epithelium at the site of the stricture. It is presumed that the short segment of bowel of the calibre of oesophagus lying between the stricture and the cardia is lined by columnar epithelium. At operation the region of the cardia is found to be soft and not inflamed so that, if it can be brought
GROUP III STRICTURES 19%

Midway stricture with segment of gastric lined oesophagus

Midway stricture at cardia

Figure 12

Figure 13
Figure 14

Strictures at the cardia but lying midway between the hiatus and the aortic arch
down through the hiatus, there is a good prospect of reflux being prevented.

**The Radiological type of Stricture**

There are many pitfalls in trying to determine the nature of a stricture by radiology alone. Such factors as spasm or exacerbations of mucosal inflammation and oedema may give quite a wrong impression of the severity of the lesion. It has been suggested that many of the radiological appearances interpreted as being those of peptic stricture are, in fact, those of spasm. We believe that none of the stenoses illustrated in this section is due to spasm alone. In many, the presence of a stricture has been confirmed at operation. In the others the stricture has been seen and felt at oesophagoscopy. They have remained as permanent lesions from the radiological point of view. In more recent cases, they have been shown not to respond to antispasmodics.

It is often difficult to determine the length of a stricture. A trickle of barium passing through it gives the impression that it is a long one. Its length can only be determined if it allows the passage of sufficient barium for films to be taken with good filling in reflux from below. Such a situation is shown in Figure 19. The radiographs give no indication of the length of the stricture as no films in reflux are available. It may help to repeat barium swallow after dilatation of the stricture so that not only the length of the stricture but its relation to the cardia and the nature of the associated hernia may be demonstrated.

Peptic strictures have a great variety of radiological appearances. Several are illustrated. Figure 11 (Page 22) A short web-like stricture (confirmed at operation). Figure 13 (Page 25) A short, rigid-looking stricture. This lesion was resected. There
Figures 20 and 21 show smooth looking strictures where, histologically, there is total mucosal loss.
was a short, hard, thickened portion of oesophagus at and immediately above the cardia. Histological section showed chronic peptic ulceration. Figures 20 and 21 (Page 28) Long, rigid-looking strictures with smooth walls. Both patients were operated on and the stricture resected.

The oesophagus was thickened and firm over a long distance. Although the strictures look smooth radiologically, there was total mucosal loss over their whole length.

Figures 22 and 23 are radiographs showing short smooth strictures. In spite of this appearance, one was seen at oesophagoscopy to be intensely inflamed and the other to be ulcerated. Figure 24 A radiograph showing an irregular stricture. Figures 25 and 26 Radiographs showing an appearance of a tubular stricture which becomes kinked when seen with the hernia filled in reflux. We believe that this radiological sign indicates rigidity of the oesophageal wall. Both patients were operated on and a grossly thickened, hard segment of oesophagus found.

A high stricture at the level of the aortic arch may be missed by the radiologist. If such a stricture is tight it can hardly escape detection but many are less obvious and allow rapid transit of barium. The situation is analogous to that of upper oesophageal web where the rapid passage of barium may make discovery of the lesion difficult. Figures 27 - 29 are radiographs of two patients in whom a high stricture was initially missed.

A type of high stricture which is readily overlooked is one which is not sufficiently tight to cause persistent dysphagia but which gives rise to repeated episodes of food impaction. The lesion consists of a fibrous ring in the oesophageal wall which, though not producing a tight stricture, cannot distend like the oesophagus above and below it. As it allows the free passage of barium, its relative
Apparently smooth strictures.
The stricture in Figure 22 was intensely inflamed and, in Figure 23, it was ulcerated.
Figure 24
An irregular peptic stricture.

Figure 25
Two patients showing kinking of the stenosis when the hernia is filled in reflux.
Figure 27
The stricture lies just above the part of oesophagus shown.

Figure 28
In Figure 28 the stricture has been missed. Figure 29 an x-ray taken later shows the stricture level with the aortic arch.
indistensibility is not seen unless solid radio-opaque medium is used. Figure 30 is a radiograph of such a lesion. The patient had bouts of food impaction over a period of 7 years. The stricture, though not very obvious on barium swallow, was well seen at oesophagoscopy.

**Radiological evidence of ulceration at the site of stricture**

Small superficial erosions at and just above a stricture, though often prominent features at oesophagoscopy, are not seen radiologically. Ulcers at the site of stricture are of two types, namely penetrating ulcers and more superficial ulcers with total mucosal loss but not penetrating the muscle coats.

**Penetrating ulcer**: This type of ulcer should be demonstrable radiologically. It is important that it should be so detected as even a deep ulcer may not be visible at oesophagoscopy if it lies within the stenosed segment. As will be discussed later, the presence of a deep ulcer influences the surgical management.

The following are examples of typical penetrating ulcers at or near the oesophago-gastric junction.

Case 1. This patient, a woman of 56, began to have episodes of food impaction in 1962. Figure 31 is a radiograph taken in 1965 showing a hiatal hernia and stricture. In 1969 the patient began to suffer from severe and persistent pain in her back. Figure 32 shows that a penetrating ulcer had developed. This was confirmed when the stricture was resected.

Case 2. This patient, a man of 54, began to have dysphagia in 1967. Figure 33 shows the radiographic appearances. The lower oesophagus was resected and a deep ulcer found.

The features of penetrating ulcer in a gastric lined oesophagus
Figure 30
A type of high stricture readily missed radiologically. The stricture is not tight but consists of a relatively indistensible ring and tends to cause episodes of food impaction. It is easily seen at oesophagoscopy.

Figure 31 (1965)  Figure 32 (1969)
Radiographs showing the development of a penetrating ulcer. The lesion was subsequently resected.
A radiograph showing a penetrating ulcer the presence of which was confirmed at resection.

Figure 33

Radiograph showing a small collection of barium between a stricture and the cardia resembling an ulcer crater.

Figure 34
will be dealt with in Section 9.

In the case of strictures in Group 3b already described where there is a short segment of gut of the calibre of oesophagus between the stricture and the cardia, radiographs may give a false impression that a deep ulcer is present. If the region of the cardia happens to be contracted, some barium is momentarily trapped in the segment of oesophagus between it and the stricture. This small collection of barium may appear on films to be lying in an ulcer crater when, in fact, no deep ulcer is present. Figure 34 is a radiograph of the appearances described.

**Superficial ulceration.** Superficial mucosal ulceration without penetration deep to the muscularis mucosae produces a stricture which, radiologically, looks smooth. If the stricture is short we doubt if it is possible to make a radiological diagnosis of this type of ulceration, as for example in the smooth short stricture shown in Figure 23 where ulceration was demonstrated on oesophageal biopsy. The characteristic appearances are due not to the mucosal damage but to the secondary inflammatory changes in the deeper layers of the oesophageal wall. They result in a long, tubular, rigid-looking stricture with an absence of peristalsis in its wall. When such appearances are seen, the presence of a long stenosis with total mucosal loss may be assumed. Figure 35 is a radiograph of this type of stricture. The patient, a man of 71, had dysphagia for six months. At oesphagoscopy, there was a tight, inflamed stricture at 35 cms. A biopsy showed granulation tissue. The stricture was resected. Histological section showed an extensive area of total mucosal loss leaving an area of ulceration beneath which there was marked non-specific chronic inflammatory infiltrate and much fibrosis. Figures 20 and 21 are other examples of this type of lesion.
Figure 35

A long, smooth stricture in a man of 71.

The resected specimen showed an extensive area of total mucosal loss.
Radiological appearances of the oesophagus above a peptic stricture.

In the majority of cases, the oesophagus above a peptic stricture is radiologically of normal calibre even though the stricture is tight or has been present for a long time.

The lack of dilatation of the oesophagus is in contrast to what happens in achalasia and commonly in the presence of a slow growing annular neoplasm.

In some cases, however, the oesophagus above a peptic stricture may appear radiologically to be either dilated or contracted.

Three illustrative x-rays of dilatation are shown.

Figure 101 (Page 120). A man of 54 with severe dysphagia. The resected specimen consisted of an annular ulcerated area 1 cm. in length. The ulcer floor consisted of granulation tissue deep to which was marked fibrosis replacing much of the muscle.

Figure 36, a man of 21. At oesophagoscopy, there was a short ulcerated stricture.

Figure 37. This patient, a man of 71, had had dysphagia for three years. There was a tight, ulcerated stricture at oesophagoscopy. The degree of dilatation is unusual for peptic stricture.

An x-ray showing the oesophagus tapering down towards a peptic stricture was found, in this series, in patients in whom the stricture had followed soon after an abdominal operation usually for duodenal ulcer or where the onset of dysphagia was immediately preceded by a haematemesis.

Figures 38 and 39. This patient, a man of 70, had difficulty in swallowing soon after vagotomy and gastro-enterostomy for duodenal ulcer and pyloric stenosis. The stricture was tight and hard.

Histological section showed an area of ulceration bound proximally by
Figure 39 shows the typical tapering oesophagus of post-operative stricture in a man operated on for pyloric stenosis (Figure 38).
squamous epithelium and distally by gastric mucosa. Deep to the ulcer there was marked fibrosis replacing much of and extending through the muscle layers. Similar x-rays are shown in Section 11. Figure 40 shows the same radiographic features in a man of 70 in whom dysphagia followed soon after a large haematemesis.

We have been unable to correlate the x-ray appearances with the type or duration of the stricture. It is tempting to assume that dilatation above a stricture is an indication of absence of gross changes in the deeper layers of the oesophageal wall but we have found it above a stricture where there was ulceration and fibrosis involving all coats of the oesophagus. In patients showing the tapering oesophagus, it seems probable that the radiological appearances are due to inflammatory changes in the oesophageal wall. Figure 41 shows a very markedly tapering oesophagus above a stricture soon after closure of a perforated duodenal ulcer. Figure 42 shows the oesophagus returning to more normal state after a period of treatment by bouginage and the subsidence of the acute inflammatory changes.
The tapering oesophagus with a stricture following a haematemesis.

Figure 41

Tapering oesophagus with stricture after closure of a perforated duodenal ulcer.

Figure 42

The same patient after self-bougination.
SECTION 5
OESOPHAGOSCOPY

Oesophagoscopy is an essential investigation both for diagnosis and for the assessment of the type and situation of the stricture as they affect treatment. The problems of diagnosis will be considered in Section 10. We are concerned here with oesophagoscopy findings in relation to treatment.

This section is based on 490 oesophagoscopys on 139 patients.

It is customary to measure the distance between the stricture and the upper incisor teeth. This measurement is objective and repeatable. Unfortunately, it does not always give a good indication of the all-important relationship of the stricture to the hiatus. Distances measured at oesophagoscopy obviously vary with the amount of extension of the neck when the measurement is made and with the height of the patient. For instance, in one of our patients who was 6 ft. 7 ins. in height a 40 cm. oesophagoscope failed to reach the stricture. In adults, we found that high strictures were, on average, 27 cms. from the upper incisor teeth (16·7 cms. from the hiatus as measured on x-ray). Low strictures were at 36 cms. (5·6 cms. from the diaphragm) and strictures in Group 3 were at 33 cms. (7 cms. from the hiatus on x-ray measurement).

There is usually good correlation between the level of the stricture as seen radiologically and its distance from the upper incisor teeth as measured at oesophagoscopy. Occasionally there is a disparity between the two measurements. This is illustrated by two patients. In the first, the stricture as seen on radiographs appears to be relatively near the hiatus (3·5 cms.). Its distance from the upper incisor teeth at oesophagoscopy was, however, only 30 cms. (Figure 43). In the second patient, the x-rays suggest that the stricture is at some distance above the diaphragm (9 cms.) but, at oesophagoscopy, it
A mild stricture lying apparently a short distance above the hiatus but found, at oesophagoscopy, to be at only 30 cms. from the upper incisor teeth.

Figure 43

A stricture appearing radiologically to be at a fairly high level but found at oesophagoscopy to be 38 cms. from the upper incisor teeth.

Figure 44
was at 38 cms. (Figure 44). In neither patient were we able to explain the discrepancy in the measurements. Both patients were of average height. It is important to appreciate that such discrepancies may exist. Not only do the oesophagoscopy and x-ray measurements not agree in every case but both may be misleading. We have found repeatedly in the case of strictures at the cardia that it is only when, at thoracotomy, a large stomach tube is passed until it is arrested at the stricture that the exact relationship of the stricture to the hiatus is established. A number of hernias which appear radiologically and at oesophagoscopy to be irreducible are, in fact not so. In some instances, therefore, we hesitate to conclude that a hernia is irreducible on the radiological and oesophagoscopy findings.

In uncomplicated sliding hiatal hernia, the region of the cardia is mobile and moves downwards in front of the oesophagoscope. There is usually little, if any, mobility at the site of a peptic stricture. If a stricture does move downwards for a short distance in front of the oesophagoscope as was observed in 3 patients, this finding may influence the choice of treatment. While the detection of some mobility in a high stricture is irrelevant, downward movement of a stricture near the cardia suggests that the oesophageal wall at and above it is not unduly rigid and that the hernia is likely to be reducible. A lower oesophageal web which is considered by some to be a type of peptic stricture, may be extremely mobile and differs in this respect from the more usual type of peptic stricture.

**Character of the stricture**

An assessment of the nature of a peptic stricture as made at oesophagoscopy is imprecise, varying from one operator to another and being limited by the fact that, as only the mucosal aspect of the
entrance to the stenosis is visible, the pathological process throughout the length of the stricture and in the deeper layers of the oesophageal wall can only be guessed at. All the endoscopies in this series were performed by the author and, to this extent, there may be some correlation between the various endoscopic reports.

Peptic strictures may be grouped roughly into those which are tight and firm and those which are soft. An opinion as to the type of stricture is formed partly on its appearance and partly on how it responds to the passage of bougies. Both findings must be taken into account as a stricture which appears to have a very small lumen may admit the passage of bougies remarkably easily. In this series what is meant by a tight stricture is one which resists the passage of a bougie in the range 9 - 12 F. Soft strictures are those which, though felt to grip it, allow the initial passage of a middle size bougie (23 - 24 F).

Based on these criteria, tight strictures formed 36 per cent and soft strictures 54 per cent of the total series. Some of the lesions defied classification into this rough grouping.

There did not appear to be any significant difference between the incidence of tight and soft strictures at the various levels in the oesophagus. Of those at the cardia, 37 per cent were tight and firm and 52 per cent soft. 37 per cent of high strictures were tight and 62 per cent soft. Of intermediate strictures 36 per cent were tight and 60 per cent soft.

It is important to note the manner in which a stricture responds to dilatation. If a tight, hard stricture is difficult to dilate and grips each successive bougie up to the largest size, this suggests that there is considerable fibrosis over a significant length of the oesophagus. Sometimes a stricture will grip the smaller bougies
tightly but a stage is reached where they suddenly become easier to pass giving the sensation that a localised fibrotic ring has been split. A soft stricture, though offering some resistance to successive bougies allows their passage up to the largest size without increasing difficulty.

At oesophagoscopy, only the upper limit of the stricture is visible except in the case of some soft stenoses. The visible mucosa may look pale and smooth and yet there may be gross inflammation and ulceration concealed within the lumen of the stenosis.

The oesophagus above a stricture

It is often stated that a stricture prevents or limits reflux. It is certainly true that some patients cease to have heartburn once they develop dysphagia. We found, however, that there was often acute oesophagitis with superficial mucosal erosions above a stricture. This was found in as many as 27 per cent of cases. The percentage of cases showing oesophagitis above a stricture was almost identical for tight and soft stenoses. This is not surprising as a tight stricture is often like a rigid tube the lumen of which, though small, is permanently open and presents no obstacle to the reflux of gastric juice. On studying the oesophagoscopy reports of patients who were relieved of their heartburn after the onset of dysphagia, we found that the majority of them had soft and inflamed rather than tight, hard strictures and, as far as the naked eye appearances were concerned, they frequently seemed still to have acute oesophagitis above the stricture.

In summary, it may be said that, while x-ray and oesophagoscopy examinations are complementary, one or other is more informative in certain respects. The important distinction between soft and hard strictures is made at oesophagoscopy. A penetrating ulcer lying within a stenosis is better demonstrated radiologically. Changes in the
oesophageal wall above a stricture, for instance the presence of diverticula or the configuration of the oesophagus itself are better shown radiologically. Demonstration of the associated hiatal hernia, its size and type, is a radiological problem. As peptic stricture prevents the easy use of a gastroscope, one must rely on x-rays for the detection of associated gastric and duodenal ulcers.
ASSOCIATION OF PEPTIC STRICTURE WITH OTHER LESIONS

It is obvious that by far the commonest condition associated with peptic stricture of the oesophagus is a sliding hiatal hernia. In only 9 patients in this series was there no apparent hiatal hernia. All of them were operated on and the absence of a hernia confirmed. In 5 patients, 2 of whom were operated on, it was not possible to decide whether or not a hernia was present. Peptic stricture without a hiatal hernia occurred particularly in two types of patient. Thus 3 were in patients who developed a stricture after an operation for duodenal ulcer and 5 were children or adolescents in whom the lower oesophagus was lined by columnar epithelium.

Associated lesions fall into two groups, namely other lesions in the oesophagus or pharynx and conditions outside the oesophagus.

Associated oesophageal and pharyngeal lesions

1. Upper Oesophageal Web

Two patients had an upper oesophageal web in addition to a peptic stricture.

Case 1: A female of 54. There was a history of dysphagia over a period of 3 years. Koilonychia was present. The haemoglobin was 56 per cent. At oesophagoscopy, a post-cricoid web was seen. It was split with a bougie and a medium bore oesophagoscope passed beyond it. At 35 cms. from the upper incisor teeth a soft, inflamed stricture was reached. The patient was operated on. Just above the oesophago-gastric junction, the oesophagus felt thickened and firm. After repair of the hernia there has been no recurrence of dysphagia over the subsequent three years. The anaemia responded rapidly and has not recurred although treatment with iron was discontinued.
Case 2: A woman of 64. There was a history of heartburn over many years and dysphagia for four months. The haemoglobin was 61 per cent. At oesophagoscopy, there was a crescentic post-cricoid web and an inflamed stricture at 30 cms. At operation in 1964, a sliding hiatus hernia was repaired. At and immediately above the cardia, there was a firm, thickened segment of the oesophagus with enlarged inflammatory glands in the adjacent mediastinum. Following repair of the hernia there has been no recurrence of dysphagia or anaemia.

Comment

It has been suggested that there is an association between hiatus hernia and pharyngo-oesophageal obstruction (Smiley et al. 1963). They found that in 27 patients with pharyngo-oesophageal obstruction, 19 had radiological evidence of a hiatus hernia. They suggested that a hernia contributes to the onset or progress of sideropenic dysphagia either by causing stimulation of the crico-pharyngeal sphincter by acid reflux or by causing iron deficiency anaemia. They admit that people found to have a hiatus hernia from a general series of barium meal examinations do not have concomitant obstructive lesions in the pharyngo-oesophageal region and that a causal relationship between hiatus hernia and sideropenic dysphagia lacks proof.

Both of our patients had low levels of haemoglobin and serum iron and, in both, the dysphagia and the anaemia were cured by hiatus hernia repair. As only 2 such cases were found in 136 adult patients with peptic stricture, no conclusion as to a possible relationship between post-cricoid web and peptic stricture can be reached.

2. Pharyngeal Diverticulum

In 3 patients there was an associated pharyngeal diverticulum.

Case 1: A man aged 60. This patient who had a long history
of heartburn developed dysphagia for solids. The x-rays (Figures 45 and 46) show a small pharyngeal pouch and a peptic stricture. At oesophagoscopy, a small pouch was seen and, at 39 cms., there was a soft, inflamed stricture. At operation, there was a sliding hiatal hernia. For a distance of 2.5 cms. above the cardia the oesophagus was thick and hard. The patient has been free from symptoms since repair of the hernia.

In 2 patients, the diverticulum was unusual in that it was lateral in position.

**Case 2**: A man of 58 who had had episodes of food impaction for 7 years. A barium swallow (Figures 47 and 48) showed a small pharyngeal pouch lying laterally on the left side. At oesophagoscopy, there was an inflamed stricture at 38 cms.

**Case 3**: This patient, a man of 71, who had a partial gastrectomy 6 years before, had a tight stricture at 39 cms. Radiographs show a small lateral pouch (Figures 49 - 51).

**Comment**

As with post-cricoid web, it has been claimed that there is an association between hiatal hernia and pharyngeal pouch (Smiley et al. 1970). They maintain that gastro-oesophageal reflux leads to crico-pharyngeal dysfunction and to the development of the pouch. They found a hiatal hernia to be present in 32 out of 34 patients with pharyngeal pouch. The resting pressure in the crico-pharyngeal sphincter has been found to be high when there is gastro-oesophageal reflux and in most patients with a pharyngeal diverticulum (Hunt et al. 1970). These authors found a fall in the resting crico-pharyngeal pressure after hernial repair. The importance of dysfunction of the upper sphincter secondary to reflux has also been emphasized by Belsey (1966). Referring to spasm of the upper sphincter he states that it can be
Figure 45

X-rays of a patient with a hiatal hernia, peptic stricture, and small pharyngeal pouch.

Figure 47

A patient with a peptic stricture and a lateral pharyngeal pouch.
X-ray films of a second patient with a hiatal hernia, peptic stricture, and lateral pharyngeal diverticulum.
relieved by restoration of competence at the cardia.

In 3 of our patients operated on (2 with a peptic stricture and post-cricoid web and 1 with a stricture and a pharyngeal pouch), hernial repair alone relieved the symptoms. Two patients with a combination of peptic stricture and a small pharyngeal pouch have been managed successfully by dilatation of the stricture alone. We do not have a sufficient number of cases to be able to say in what proportion of patients with a peptic stricture and either a post-cricoid web or pharyngeal pouch, restoration of competence at the cardia is adequate treatment for both lesions.

In the case of both post-cricoid web and pharyngeal pouch it would appear that if one investigates patients with either lesion a high incidence of hiatal hernia is found. On the other hand, if one starts with a group of patients with reflux, the number of webs and pouches discovered is small. In our series of peptic stricture there were only 2 webs and 3 pouches. It is of interest that our patients with post-cricoid web in association with peptic stricture were all females while the patients with pharyngeal pouches were all males. This sex incidence is normal for both lesions and does not reflect the higher incidence of gastro-oesophageal reflux in women.

In patients presenting with a post-cricoid web or pharyngeal diverticulum it would seem to be important to look carefully for a hiatal hernia or peptic stricture.

**Pulsion diverticula above a peptic stricture**

Occasionally, small diverticula are seen radiologically above a peptic stricture. They are presumably pulsion diverticula.

**Case 1** A woman of 69 with dysphagia for 2 years and a tight, hard stricture at 31 cms. The x-ray (Figure 52) shows multiple small diverticula having the appearance of small mucosal pouches.
X-ray films of 3 patients showing varying degrees of diverticulum formation above a peptic stricture.

Figure 52

Figure 53

Figure 54
Case 2: A woman of 73 with dysphagia for 15 years and a hard, tight stricture at 33 cms. The x-ray (Figure 53) shows a small, irregular diverticulum hanging downwards as does a pharyngeal pouch.

Case 3: This patient, a woman of 74, had dysphagia for only 1 year. At oesophagoscopy, the stricture was soft and inflamed. The radiograph (Figure 54) shows a diverticulum considerably larger than those present in the previous two patients.

The diverticula were shown radiologically but were not seen at oesophagoscopy. The patients were all elderly but there was no apparent relationship between the formation of a diverticulum and the duration of the dysphagia or the type of stricture present. As none of the patients was operated on, histological findings are not available. The diverticula were not a cause of symptoms and it was possible to maintain the comfort and the nutrition of the patients by dilatation of the stricture.

Associated extra-oesophageal lesions

The commonest lesion of this type was duodenal ulcer which was noted in 23 patients (16.6 per cent). Six of the ulcers had previously perforated. Two had perforated twice. Pyloric stenosis was found in 4 patients. Gastric ulcer was much less common (4 patients).

Fifteen patients had been operated on in the past for duodenal or gastric ulcer. In spite of partial gastrectomy (5), and vagotomy and gastro-enterostomy (3), peptic stricture of the oesophagus developed at a later date.

The incidence of disease of the biliary tract and diverticulitis was much lower than that of duodenal ulcer (cholecystitis or stone - 6; diverticulitis - 5). Neither cholecystography nor barium enema were done as a routine which may explain their low incidence.

Three patients suffered from Parkinsonism and one child had repetitive purposeless movements due to phenylketonuria.
Two patients suffered from systemic sclerosis and one had the Zollinger-Ellison syndrome.

The relationship between duodenal ulcer and oesophagitis is by no means clear. Several series have been reported in which there was a frequent association between the two conditions. Winkelstein (1957) found that 16 out of 20 patients with severe oesophagitis had a duodenal ulcer. Benedict and Sweet (1948) found that of 60 patients with oesophageal stricture, 20 had a duodenal ulcer.

Burge et al. (1966), in a series of 44 patients with symptomatic hiatal hernia, estimated that 50 per cent had a duodenal ulcer, 77 per cent had pyloric channel disease, and 14 per cent a lesser curvature ulcer. On the other hand Collis (1970) points out that "if associated disease was a major factor in causing a response of a particular type or degree in the oesophagus it would have to be associated especially with symptomatic hiatus hernia. In fact, the findings seem the opposite". He quotes Palmer (1968) who found the association to be most common in his patients with asymptomatic hiatal hernia. At present, therefore, there is some uncertainty about the relationship between duodenal ulcer and oesophagitis.

If there is an association, the most likely mediating factor would seem to be gastric acidity. Again there are differing views. Burge (1966) mentions high fasting levels of acidity when there is a duodenal ulcer or pyloric channel disease. Weber (1969) states that "the degree of mischief caused by ordinary hiatus hernia is directly related to the levels of gastric acidity". The opposite view has been expressed, namely that there is no clear relationship between gastric acidity and the degree of oesophagitis or the type of response of the oesophageal mucosa to reflux. A rational plan of treatment must be profoundly influenced by which of the above conflicting views one accepts.
Casten (1967) says that singly, or in combination, the pathological entities, peptic oesophagitis, hiatal hernia, and duodenal ulcer, are manifestations of acid secretion disease with a common pathological mechanism. He therefore bases his surgical treatment on vagotomy and is of the opinion that "actual repair of the hiatus is of considerably less significance." Burge (1964) occasionally omits hernial repair from his operative procedure. Others, including ourselves, place the emphasis on control of reflux.

The extent to which the presence of a duodenal ulcer has influenced treatment in this series is indicated by the following facts.

1. It was possible to deal with a large number of patients by dilatation and medical treatment alone. The improved results of the medical treatment of peptic ulcer following the use of carbekoxolone sodium is of some relevance. In patients doing well on conservative management we have not attempted to influence the stricture by operative procedures designed to produce achlorhydria. We have not had occasion to discontinue treatment by dilatation because of complications arising from the presence of a duodenal ulcer.

2. Where surgery was undertaken, the favoured procedure was hernial repair where possible and the emphasis was placed on the control of reflux. In the presence of delayed emptying of the stomach or of chronic duodenal ulceration vagotomy and drainage operation have been added. It may be mentioned that, if there seems any likelihood that vagotomy might become necessary, it is probably better done at the time of hernial repair as it can be a difficult procedure, technically, at a later date.

3. Resection of a stricture and interposition of a jejunal or colonic segment involves vagotomy and a drainage procedure on the stomach. The relevance of duodenal ulcer here is that the state of the
pylorus and first part of the duodenum influences the choice of the type of drainage operation.

In short, we have placed much less emphasis on levels of gastric acidity or the presence of duodenal ulcer than some authors and, where surgical treatment has been undertaken, have concentrated on the control of reflux.
SECTION 7
Differential Diagnosis

Malignant stricture. The presence of malignancy must be excluded in every case though it is, of course, unlikely in infants and children. Even though a patient has a long history of heartburn and x-rays show a hiatal hernia with a narrowing at the cardia, it must not be assumed that the lesion is a peptic stricture. One negative biopsy is unreliable. Repeated biopsies may be necessary. This is particularly so in patients from whom a negative biopsy is obtained but in whom the stricture fails to respond to dilatation. If, after dilatation, dysphagia is as bad as ever within a day or two, there is a strong possibility that the lesion is a carcinoma.

Lower Oesophageal Web. The following case report illustrates certain features of this lesion and is of interest in that both upper and lower oesophageal webs were present.

The patient, a man of 56, had increasing dysphagia for solids for 4 years. An x-ray in 1966 (Figures 55 and 56) showed a post-cricoid web and, in addition, a lower oesophageal web. The haemoglobin was 69 per cent and the serum iron 51 μg/100 mls. At oesophagoscopy, the upper web was split. The instrument was held up at the cardia but the findings at this level were indefinite. At thoracotomy, there was a small sliding hiatal hernia but no visible or palpable lesion in the oesophagus. A large stomach tube was passed. It appeared to enter the stomach. This appearance was deceptive as, when the stomach was opened, the tube was found to be pushing a mobile web downwards in front of it. The web was excised by submucosal dissection (Figure 57). Histological section showed the mucosal junction at the rim of the orifice, submucous fibrosis with slight lymphocytic infiltration, and no ulceration. The muscularis mucosae appeared to be hypertrophied.

There has been controversy as to the nature and cause of this
Figures 55 and 56

X-ray films of a patient with both upper and lower oesophageal webs. Figure 56 has reproduced badly but the lower web is just visible.

Figure 57

A photograph of the lower web excised from the patient whose radiographs are shown above.
condition. Inglefinger and Kramer (1953) described it as a contractile oesophageal ring. Schatzki and Gary (1953) considered it to be a fixed, permanent ring. The lower oesophageal webs we have seen consisted of a fairly tough diaphragm and were not merely transient segmental spasm.

The aetiology is also in dispute. The lesion has been considered to be a congenital anomaly. If so, it is hard to explain why it causes symptoms only in later life (Bugden and Delmonico, 1956) and why it may cause progressive oesophageal narrowing in the elderly. The following case illustrates this point.

This woman was seen in 1963 at the age of 67. She gave a previous history of heartburn for many years and slight, intermittent dysphagia for 4 years. At oesophagoscopy, no lesion was seen in the lower oesophagus. Dysphagia became worse in 1969 and the patient could swallow liquids only (Figure 58). The oesophagoscope passed to 35 cms. At this point what looked like redundant mucosa was seen. The lumen could not be found. At thoracotomy, no lesion was visible or palpable in the oesophagus, but a stomach tube was held up within the associated hiatal hernia. When the stomach was opened, a tough diaphragm was found at the cardia. Its lumen was 3 mm. in diameter. There seems no doubt that, in this patient, progressive stenosis had occurred between the ages of 67 and 73.

Gerami and Cole (1970) describe 7 adult patients with oesophageal rings which they consider to be congenital in origin. In 3 of their patients the ring was situated at the oesophago-gastric junction, in 2 it was 2 inches above the junction, and, in 1 patient, 4 inches above the junction. They state, however, that none of their patients had gastric mucosa at the level of the ring. Mark and Goldenberg (1964) reported a child with two oesophageal webs. Apparently squamous epithelium was found above and below both of them. They considered
Radiograph of a patient of 73 with a lower oesophageal web. Dysphagia had increased steadily since she was 67 years of age.

A typical lower oesophageal web. The x-ray appearances are distinctive and unlike those of the more common peptic stricture.
the condition to be a congenital one. We describe on page 86 a baby with a web in the middle of the oesophagus covered on both sides by squamous epithelium.

Barrett (1962) maintains that the lesion is a form of peptic stricture and Paulson (1967) describes it as an annular peptic stricture. Just as there are some features which make it difficult to accept a congenital aetiology, so there are others which are hard to explain if the lesion is to be considered as a peptic stricture. The radiological appearances are distinctive and unlike those of the more common types of peptic stricture (Figure 59). If the condition is due to reflux, it should be found higher up the oesophagus when the lower oesophagus is lined by columnar epithelium. Although we have seen many high strictures, none of them has shown the characteristic features of a web. Lower oesophageal web also differs from the usual peptic stricture in that it may be very mobile and may be difficult or impossible to see at oesophagoscopy. Also, it is not ulcerated. This is the classical lower oesophageal web.

Those who believe that lower oesophageal web is a form of peptic stricture assume that it is secondary to the hernia associated with it. In our cases and in those illustrated in the literature, the hernia is relatively small. The calibre of the oesophagus immediately above the web is similar to that of the hernia. Why the hernia should be almost invariably of this type is not easy to explain. We would suggest as a hypothesis that the web may not be secondary to the hernia but that the converse may be true. With obstruction to the passage of food over a period of many years and the consequent necessity for powerful contractions of the oesophageal muscle including the muscularis mucosae to overcome it, a small hernia may eventually be produced by upward traction especially by the longitudinal muscle. Some webs in
older people look somewhat irregular and their mucosa is thickened. This may be a mucosal response to reflux in the older age groups. These two factors might explain why the dysphagia tends to get worse in later life. The concept of the importance of the longitudinal muscle in elevating the oesophago-gastric junction is elaborated in Section 13.

In our experience, there are two types of web-like stricture. One is a constriction at the oesophago-gastric junction in association with a hiatal hernia and lined on its upper surface by squamous and on its lower aspect by gastric mucosa, of which an example is described on page 59. The second type lies above the oesophago-gastric junction and is covered on both sides by squamous epithelium. The latter type is presumably congenital. It is in the former type that the question of aetiology remains a problem.

While it is admitted that some lower oesophageal webs may be a form of peptic stricture, the condition is included here in the differential diagnosis as it is essential to consider this type of lesion as a distinct entity from the point of view of treatment. A web, if it is causing significant dysphagia and does not respond to the passage of bougies, may be dealt with by submucous resection and repair of the hernia. Resection of a segment of the oesophagus is never necessary. We have no information as to whether or not this lesion will respond to hernial repair alone as will many peptic strictures.

Neuromuscular disorders of the oesophagus.

When cardiospasm is found together with a hiatal hernia it is important from the point of view of treatment not to mistake it for a peptic stricture. The following is an illustrative case.

The patient, a man of 62, had a history of dysphagia and
regurgitation of food and mucus for 4 years. He had lost weight. The x-ray appearances are shown in Figure 60. At oesophagoscopy, the cardia was at normal level. There was a smooth narrowing without inflammation. The narrow segment felt soft but gripped each successive bougie. At thoracotomy, the findings were typical of cardiospasm except that there was a hiatal hernia and the cardia was 3.5 cms. above the hiatus. A Heller's operation was done and the hernia repaired. There was no sign of submucosal inflammation. The patient did well and, within 6 months, had gained 1½ stones in weight.

While cardiospasm and hiatal hernia are not commonly associated, diffuse muscle spasm of the oesophagus is often found together with a hiatal hernia. Creamer (1962) stated that a sliding hiatal hernia is almost invariably present in cases of diffuse spasm but Craddock et al. (1966) in a series of 12 cases of diffuse spasm were unable to demonstrate such a high incidence of hernia. The differential diagnosis between diffuse spasm and peptic stricture is rarely a problem but confusion may arise if the oesophagus above a stricture is hyperperistaltic.

Figure 61 is the x-ray of a patient with true diffuse spasm and a hernia. This patient, a man of 59, complained of intermittent discomfort deep to the xiphoid. He had dysphagia of variable severity both for liquids and solids. He often regurgitated food and mucus. An x-ray in 1967 showed appearances thought to be due to cardiospasm or to more diffuse spasm together with a hiatal hernia. In 1970, the hernia was more easily demonstrable and the condition of the oesophagus was more definitely that of diffuse spasm. The lesion responded to oesophageal myotomy and hernial repair.

**Oesophageal Spasm.** This is essentially a transient condition. It may increase the dysphagia due to an existing stricture or occur in association with acute oesophagitis alone. It may respond to anti-
Radiograph of a man aged 62 who had cardiospasm and also a hiatal hernia.

A male patient, aged 59, with a hiatal hernia and diffuse muscle spasm of the oesophagus.
spasmodics. A typical example of this condition was a man of 72 who, after prostatectomy, had to have the foot of his bed raised because of haemorrhage. He became unable to swallow even liquids. A barium swallow showed the barium forming a tapering column down to a complete obstruction. After 12 hours during which the patient was kept sitting up and was given alkalis, he again swallowed normally. Subsequent x-rays showed gastro-oesophageal reflux but no stricture.

**Corrosive Stricture.** Mare and Wooler (1969) have described peptic stricture following corrosive stricture of the oesophagus 20 to 50 years before. They believe that the hiatal hernia was produced by contraction of the fibrotic wall of the oesophagus.

**Systemic Sclerosis.** It is debatable if the oesophageal stricture found in systemic sclerosis should be considered in the differential diagnosis as it is probably a form of peptic stricture due to reflux secondary to the pathological condition and disordered function of the oesophagus. Figure 62 shows an oesophagus and stricture in systemic sclerosis.

**Extrinsic Pressure.** Conditions causing extrinsic pressure on the oesophagus do not usually mimic peptic stricture. We have seen only 1 patient where extrinsic pressure created a problem in differential diagnosis. The patient, a woman of 60, had had an operation for repair of a hiatal hernia 5 years before. She returned with dysphagia. The barium swallow is shown in Figure 63. In view of her history there seemed a possibility that the condition was a peptic stricture. It was, in fact, due to malignant tissue in the mediastinum secondary to a breast carcinoma which had been removed 18 years before.
An oesophageal stricture in diffuse systemic sclerosis.

Extrinsic compression of the lower oesophagus due to metastases from a breast cancer in a woman who had been operated on for hiatal hernia 5 years before.
POST-OPERATIVE AND POST-RECUMBENCY

STRUCTURES OF THE OESOPHAGUS

In this series there were 18 patients who developed an oesophageal stricture following either an operation or a period of recumbency. In addition, two patients with slight dysphagia had more difficulty in swallowing after an operation. One was a woman of 56 who had a leg amputated and the other, a man of 70 who had vagotomy and gastro-enterostomy for a duodenal ulcer.

The Table on Page 70 shows the details of the 18 patients.

It will be seen that the most common previous condition was peptic ulcer (duodenal ulcer - 6; gastric ulcer - 1). In 3 patients there was pyloric stenosis. 2 patients in whom the dysphagia followed prostatectomy also had a duodenal ulcer as had 1 patient in whom the dysphagia followed cholecystectomy.

All the patients in whom the stricture followed an operation for duodenal ulcer were men. This sex incidence is quite different from that of peptic stricture in general. Post-operative peptic stricture accounts for a high proportion of those occurring in men between the ages of 40 and 50.

In this type of lesion there is a higher proportion of tight, hard strictures than in the rest of the series (14 tight and hard, and 4 soft).

Weight loss is more common in this group. Several of the patients had operations after which some weight loss is to be expected, but, in addition, dysphagia tends to be severe with this type of lesion.

One radiological appearance, though not present in every case, is common with this stricture. The oesophagus tapers down to the stricture in a rat tail manner (Figure 64). This appearance has already been illustrated (Figures 39 and 41). A similar radiological appearance
Table shows the details of the 18 patients

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age</th>
<th>Previous condition</th>
<th>Operation</th>
<th>Time of onset of dysphagia</th>
</tr>
</thead>
<tbody>
<tr>
<td>M</td>
<td>79</td>
<td>Pyloric stenosis</td>
<td>Gastroenterostomy</td>
<td>3 weeks</td>
</tr>
<tr>
<td>M</td>
<td>71</td>
<td>Pyloric stenosis</td>
<td>Gastroenterostomy</td>
<td>4 weeks</td>
</tr>
<tr>
<td>M</td>
<td>45</td>
<td>Perforated duodenal ulcer</td>
<td>Closure of perforation</td>
<td>3 weeks</td>
</tr>
<tr>
<td>F</td>
<td>66</td>
<td>Gastric ulcer</td>
<td>Partial gastrectomy and vagotomy...</td>
<td>4 weeks</td>
</tr>
<tr>
<td>M</td>
<td>67</td>
<td>Chronic duodenal ulcer</td>
<td>Vagotomy and gastroenterostomy followed by Billroth I...</td>
<td>3 weeks</td>
</tr>
<tr>
<td>M</td>
<td>45</td>
<td>Perforated duodenal ulcer and pyloric stenosis</td>
<td>Gastroenterostomy</td>
<td>8 months</td>
</tr>
<tr>
<td>M</td>
<td>80</td>
<td>Perforated duodenal ulcer</td>
<td>Closure of perforation</td>
<td>6 months</td>
</tr>
<tr>
<td>F</td>
<td>86</td>
<td>Gall stones and chronic duodenal ulcer</td>
<td>Cholecystectomy</td>
<td>4 weeks</td>
</tr>
<tr>
<td>M</td>
<td>73</td>
<td>Cholecystitis</td>
<td>Cholecystectomy</td>
<td>3 months</td>
</tr>
<tr>
<td>M</td>
<td>68</td>
<td>Prostatic enlargement and chronic duodenal ulcer</td>
<td>Prostatectomy</td>
<td>4 weeks</td>
</tr>
<tr>
<td>M</td>
<td>76</td>
<td>Prostatic enlargement and chronic duodenal ulcer</td>
<td>Prostatectomy</td>
<td>1 week</td>
</tr>
<tr>
<td>M</td>
<td>61</td>
<td>Suspected carcinoma of stomach</td>
<td>Prostatectomy</td>
<td>4 weeks</td>
</tr>
<tr>
<td>F</td>
<td>68</td>
<td>Fibroids</td>
<td>Laparotomy</td>
<td>2 weeks</td>
</tr>
<tr>
<td>M</td>
<td>60</td>
<td>Diverticulitis</td>
<td>Hysterectomy</td>
<td>4 weeks</td>
</tr>
<tr>
<td>F</td>
<td>82</td>
<td>Fracture of femur</td>
<td>Resection of bowel</td>
<td>6 weeks</td>
</tr>
<tr>
<td>F</td>
<td>69</td>
<td>Fracture of spine</td>
<td>Pin and plate</td>
<td>5 weeks</td>
</tr>
<tr>
<td>F</td>
<td>67</td>
<td>Fracture of tibia</td>
<td>Reduction</td>
<td>3 weeks</td>
</tr>
<tr>
<td>M</td>
<td>66</td>
<td>Coronary thrombosis</td>
<td>Reduction</td>
<td>2 weeks</td>
</tr>
</tbody>
</table>

Note: "..." indicates operations not specified in the table.
The oesophagus tapering down towards a stricture in a man of 70 who developed dysphagia after vagotomy and gastroenterostomy for duodenal ulcer.

Figure 64

An x-ray film of a patient who had dysphagia following vagotomy and partial gastrectomy. The appearances are suggestive of a phase of cardiospasm but there was, in fact, a firm, fibrous collar round the cardia.

Figure 65
is seen in some post-recumbency strictures. This is shown in Figure 5 (Page 20) which is the barium swallow of a patient who developed severe dysphagia after a period of enforced recumbency following a coronary infarction.

As the stricture develops in a patient where attention has previously been concentrated on a lesion elsewhere, there are often no adequate films to show if there was a pre-existing hiatal hernia. Of 8 patients operated on for the stricture, 5 were found to have a hiatal hernia. Three out of 5 patients not operated on were thought to have a hernia on the evidence of films taken at the time of their original illness. In 5 patients it was not possible to say whether there was a hernia or not. It would appear, therefore, that there was a pre-existing hernia in about half of the patients.

Post-operative vomiting which has been incriminated as a cause was recorded in only 2 patients. As far as could be determined, a naso-gastric tube had been used in about half of the patients.

In 5 patients, the stricture was resected. In all of them, histological section showed a deep ulceration with dense underlying fibrosis extending through and replacing much of the muscle layers.

It is of interest that the radiological appearances in patients who develop dysphagia soon after a haematemesis and where there is presumably a deep ulcer present are similar to those of post-operative strictures (Figure 40).

Treatment.

As would be expected from the pathological findings this type of stricture is often intractable. Resection was necessary in 5. Repair of the hernia was successful in curing the stricture in 3 patients and ineffective in 1. Management by dilatations via the oesophagoscope was satisfactory in 6 patients but failed in 4 who were taught self-
bouginage and subsequently did well.

**Aetiology.**

We have previously reported 7 patients with post-operative peptic stricture (Davidson, 1967). As in the larger number of patients described here, it was found that it occurs most often in male patients who have had an operation for duodenal ulcer. Benedict (1960) reported 12 oesophageal stenoses following surgery for duodenal ulcer (sub-total gastrectomy - 10; repair of perforation - 2). The total number of patients described in papers by Bingham (1958), Cox (1961), Douglas (1956), Hurst (1961), and McKean (1958), was 16 of whom 13 were men. 14 of their patients had been operated on for peptic ulcer which was, almost without exception, a duodenal ulcer. There was also a high incidence of perforation and pyloric stenosis. In their patients, the dysphagia began about 4 weeks post-operatively, an interval similar to that found by us.

We believe that the stricture is the result of a localised burn of the oesophagus by gastric juice and that the lesion has many of the features of a corrosive stricture. The period during which the actual burn occurs is probably quite short. This conclusion is based on the following clinical observations.

1) The stricture occurs most often in patients known to be susceptible to peptic ulceration.

2) The time of onset of dysphagia in relation to the operation is remarkably constant which suggests that the damage to the mucosa is done at the same time in each case.

We believe that it occurs at the time of operation and that this is the only way of accounting for the frequency with which dysphagia develops 3 to 5 weeks afterwards.

3) The strictures tend to be of the same pathological type.
with deep localized ulceration and fibrosis extending through the muscle coats.

4) A stricture may develop about 4 weeks after an adequate partial gastrectomy or vagotomy where achlorhydria is achieved. If it is the case that the lesion is due to the effect of acid on the oesophageal mucosa, it is logical to conclude that the mucosal damage is done during or immediately after the operation while acid is still present. It has been assumed that, when a stricture follows an operation which produces achlorhydria it is the result of alkaline oesophagitis (Cole, 1968). However, the time of onset of the dysphagia is the same whether it follows an operation producing achlorhydria or one such as simple closure of a perforation where gastric acid remains. Also, the histological findings are the same regardless of the nature of the preceding operation. If some of the strictures are due to acid and some to alkaline reflux, one would expect more variation in the time of onset and in the pathological features.

We suggest, for these reasons, that the lesion is due to an acute burn at the time of operation when the patient is recumbent and anaesthetized. The situation is different from that of a person asleep when the discomfort of acid in the oesophagus causes waking and the lower oesophagus is emptied or its contents diluted or neutralized.

Post-operative stricture and post vagotomy dysphagia

In 2 of the above group of patients, vagotomy had been carried out in addition to partial gastrectomy.

The radiological appearances in patients described in the literature as suffering from post-vagotomy dysphagia are very similar to those found in a high proportion of our cases of post-operative strictures. The barium swallow shows a slight dilatation and tapering
down to the stenosis. From the radiological point of view, therefore, there is a problem in differential diagnosis. Clinically, post-vagotomy dysphagia is of earlier onset being noticed at the first attempt to swallow solid food (on average 7 to 14 days post-operatively). Post-vagotomy dysphagia is usually transient, disappearing spontaneously or requiring perhaps one dilatation. The strictures in our series were permanent and, though sometimes manageable by dilatation, this was required repeatedly over long periods of time. In a few cases of post-vagotomy dysphagia, repeated dilatations have been necessary and, on occasion, operative treatment has been required. It is this type of lesion which may be difficult to distinguish from post-operative peptic stricture. Guillory and Clagett (1967) describe two patients with post-vagotomy dysphagia in whom thoracotomy was done. In one patient, a woman of 58 who had a cholecystectomy, vagotomy, and pyloroplasty, they found a very tight ring of scar tissue surrounding the oesophago-gastric junction. The mucosa was apparently normal. The dysphagia was attributed to traumatic peri-oesophageal fibrosis. In the other patient, a woman of 56 who had a vagotomy, pyloroplasty, and repair of hiatal hernia, they again found scarring round the cardia but there was also oesophagitis. The dysphagia was considered to be due to a combination of peri-oesophageal fibrosis and oesophagitis. They state, however, that vagal denervation of the distal oesophagus accounts for most cases of post-vagotomy dysphagia.

As a cause of post-vagotomy dysphagia varying degrees of emphasis have been placed on denervation of the lower oesophagus (Moses, 1947), trauma to the region of the oesophago-gastric junction (Edwards, 1970), and reflux oesophagitis.

We shall consider these various hypotheses in relation to the findings in our patients with post-operative strictures.
"Neurogenic" dysphagia.

While Bagradi et al. (1962) are of the opinion that the commonest cause of post-vagotomy dysphagia is a cardiospasm due to denervation of the lower oesophagus and injury to the myenteric plexus, Williams and Woodward (1967) found that abdominal vagotomy, far from causing cardiospasm, results in a fall in the lower oesophageal sphincter pressure. Figure 65 (Page 71) shows the oesophagus of a woman with a stricture following vagotomy and partial gastrectomy. The appearances are a little suggestive of a stage of cardiospasm but the lesion was in fact a firm fibrous collar. We have not met a case of cardiospasm following vagotomy.

Oesophagitis.

In 1 of our cases where vagotomy had been done, the resected specimen showed an ulcerated area bounded proximally by squamous and distally by columnar epithelium. Deep to the ulcer there was fibrosis and chronic inflammatory infiltrate extending into the muscle coats. The histological findings seem more suggestive of a lesion originating in the mucosa than in trauma to the external coats of the oesophagus. The lesion was identical with those occurring post-operatively where no vagotomy had been done. Dysphagia began 3 weeks after operation. It is doubtful if the vagotomy need be implicated in any way. Clarke et al. (1965), however, consider that post-vagotomy stricture is due to incompetence at the cardia resulting from its increased mobility following the operative procedure.

Trauma.

In 1 patient in our series, the stricture could have been due to trauma. This was a woman of 66 who had dysphagia soon after partial gastrectomy and abdominal vagotomy. At oesophagoscopy two years later, there was a smooth, firm stricture at the cardia. The mucosa seemed normal. At thoracotomy, the oesophagus narrowed just above the hiatus.
The muscle coats seemed to have disappeared and to have been replaced by a fibrous collar round the oesophagus.

There is no doubt that, at abdominal vagotomy, considerable trauma may be inflicted on the lower oesophagus. We have seen 3 patients where the effects of such trauma were obvious.

**Case 1**: A man of 49 had a vagotomy and gastroenterostomy for chronic duodenal ulcer. He was known to have a hiatal hernia and some shortening of the oesophagus. When he was seen by us 6 days after operation, he had general peritonitis and died on the same day. At autopsy, there was a traumatic perforation at the cardia communicating both with the mediastinum and the peritoneal cavity.

**Case 2**: This patient, a man of 31, was seen 6 days after vagotomy and gastroenterostomy for duodenal ulcer. He had a left pyo-pneumothorax. He died 1 month later. There was a fistula between the lower oesophagus, the left lower lobe, and the left pleural cavity.

**Case 3**: A man of 62 was operated on for a chronic duodenal ulcer. At operation, there was said to be difficulty in performing vagotomy. A hole was made in the oesophagus. Two weeks later an x-ray showed a tight stricture and rat-tail oesophagus. It was apparent on studying the previous films that this patient had a small hiatal hernia and changes in the lower oesophagus before the vagotomy was done (Figures 66 and 67).

Such cases illustrate the degree of trauma which may be inflicted at abdominal vagotomy. Dragstedt (1945) and Dragstedt and Williams (1951) believed post-vagotomy dysphagia to be due to trauma. Dagradi (1962) found it to be more common in the practice of surgeons who probed deeply into the oesophageal wall or maintained prolonged traction or torsion on the oesophagus. Bruce and Small (1959) have attributed it to temporary spasm due to oedema and haemorrhage.
A post-vagotomy stricture where there had been trauma to the oesophagus at the time of operation (trans-abdominal vagotomy). The oesophagus shows the same configuration as is found in post-operative peptic stricture.

Figure 66

A previous x-ray of the above patient showing a hiatus hernia and possible changes in the lower oesophagus not appreciated at the time of abdominal vagotomy.

Figure 67
Edwards (1970) considers it to be due to tissue damage outside the muscle wall. A haematoma becomes organized producing a peri-oesophageal cuff with a high resistance to stretch. This is usually absorbed but may persist as a fibrotic collar.

In our cases of post-operative stricture, with the possible exception of the 1 patient described where there was a fibrous collar round the oesophagus, no stricture seemed definitely attributable to vagotomy.

Of the 3 patients (not in the peptic stricture series) described as having severe trauma at vagotomy, 2 at least had previous changes in the lower oesophagus. These changes had been ignored. We suggest that tearing of the lower oesophageal mucosa or of the muscle layers is more likely to occur during the downward traction on the cardia if there is already some fixation of the mucosa and shortening of the oesophagus due to reflux oesophagitis.

Cases of post-vagotomy dysphagia referred to a Thoracic Surgical Department are those where the dysphagia is persistent. The findings in the patients described above suggest that persistent dysphagia following trans-abdominal vagotomy is most likely to be due either to trauma or to the development of a post-operative peptic stricture.

Where vagotomy has been done, its possible effect on the lower oesophagus must be taken into account in the differential diagnosis of post-operative peptic stricture.
HIGH PEPTIC STRICTURE AND
LOWER OESOPHAGUS LINED BY COLUMNAR EPITHELIUM

In the past there have been many accounts of the findings of abnormal epithelium in the oesophagus. What is meant here by "gastric lined oesophagus" is not the presence of islets of columnar epithelium in the oesophagus but columnar epithelium, usually of gastric type, extending upwards from the oesophago-gastric junction in an unbroken sheet.

Controversy has persisted as to whether the lower oesophagus lined by columnar epithelium is a congenital or an acquired condition. Allison and Johnstone (1953) discussed both possibilities but favoured the congenital theory. In considering the view that the condition is acquired, they mentioned the possibility that healing of oesophageal ulceration is by gastric mucosa at the expense of squamous mucosa. This view was accepted without reservation by Hayward (1961). Allison is now more certain that the congenital theory is the correct one and states categorically that "as part of a developmental error, a variable length of the lower oesophagus may be lined not by squamous epithelium but by columnar epithelium identical with that at the cardiac end of the stomach" (Allison, 1970). Sandry (1962) and d'Abreu (1963) support this view.

A second problem is that of the secretory activity of the columnar epithelium in the oesophagus. Cases have been described where oxyntic cells were absent while, in others, oxyntic cells have been seen in large numbers. In the patient described in detail by Abrams and Heath (1965), there were many oxyntic cells and it was considered that, from the histological point of view, the epithelium was capable of secreting hydrochloric acid. On the other hand Barrett (1957) suggests that the abnormal epithelium probably secretes little digestive juice.
Belsey (1966) has apparently accepted that the columnar epithelium may sometimes secrete active digestive juice.

We are concerned here mainly with the clinical aspects of the condition and the light they shed on the aetiology and secretory activity of the abnormal epithelium. This section is based on 31 patients. All of them had high peptic strictures. In 13, the presence of gastric lined oesophagus was proved histologically. In 18, the presence of columnar epithelium in the lower oesophagus seemed certain although histological proof was not obtained. High stricture and gastric lined oesophagus are, in our experience, commoner than is sometimes suggested.

**Diagnosis.**

The criteria for diagnosis are as follows:

1. Radiographic and endoscopic evidence of a high benign stricture.

2. Radiological and endoscopic evidence that the stricture is undoubtedly at a considerable distance above the cardia.

3. Histological evidence of the presence of gastric mucosa at the stricture when it is possible to be certain that the biopsy has not been taken from within a herniated portion of stomach.

4. The diagnosis may be made from the examination of a resected specimen.

The following case histories are illustrative of the patients in whom the diagnosis was established.

1. **Diagnosis by x-rays and histological examination of an oesophageal biopsy.**

   The patient, a woman of 65, had heartburn for many years and dysphagia for 1 year. The radiographs (Figures 68 and 69) show an irregular stricture at the level of the upper border of the aortic arch and a hernia with reflux. At oesophagoscopy, there was a tight,
A high peptic stricture in which gastric mucosa was found in an oesophageal biopsy at 22 cms.

A peptic stricture level with the upper border of the aortic arch. A biopsy contained granulation tissue only. The presence of gastric lined oesophagus is assumed from the radiological appearances.
smooth stricture at 22 cms. A biopsy from within the stenosis showed the presence of gastric type epithelium. A similar case is illustrated in Figure 70.

2. Diagnosis by x-rays, oesophageal biopsy, and findings at thoracotomy for hernial repair.

The patient, whose barium swallow is illustrated in Figure 9 (Page 21) was a man of 60 who had a long history of heartburn which had become less severe following the onset of dysphagia two years previously. The radiographs show a stricture at the level of the upper margin of the aortic arch and a hiatal hernia with reflux. At oesophagoscopy, there was an ulcerated stricture at 23 cms. and gastric mucosa was found in a biopsy from it. At thoracotomy for repair of the hiatal hernia, the oesophagus was normal from the oesophago-gastric junction to the aortic arch. The hernia was relatively small and it was obvious that the biopsy could not have been taken from within it.

3. Diagnosis by x-ray, oesophagoscopy and examination of the resected specimen.

Two patients in whom the diagnosis was made in this way are illustrated in Figures 20 and 21 (Page 28).

The patient, whose barium swallow is shown in Figure 20, was a mentally defective youth of 17, with a long history of dysphagia which had recently become more severe and was producing emaciation. The x-rays show a high stricture. The presence of a hernia was uncertain radiologically. At oesophagoscopy, there was a hard, tight, inflamed stricture at 28 cms. At thoracotomy there was no hernia to be seen. The oesophagus was normal in appearance from the level of the hiatus to just above the level of the inferior pulmonary vein. From this point up to the aortic arch it was much thickened. Histological section of the resected specimen showed the lower oesophagus from the
cardia to the stricture to be lined by gastric epithelium.

In some cases it is not possible to obtain histological proof of the presence of gastric lining in the oesophagus. A biopsy may show, as it did in a number of our cases, only ulceration with no recognisable epithelium. Unless the stricture is resected, therefore, histological proof is lacking. We believe that the presence of columnar epithelium may be assumed in the following circumstances:

1) X-ray and endoscopic evidence of a high stricture at a considerable distance above the cardia.

2) Such findings with, in addition, evidence at thoracotomy where, though the lesion was not resected, it was found to be at a considerable distance above the oesophago-gastric junction.

The following are two illustrative cases.

The first patient, a woman of 65, had a history of heartburn for 30 years and of dysphagia for 2 years. X-rays (Figure 70, page 82) show a stricture level with the upper margin of the aortic arch and a relatively small sliding hiatal hernia. At oesophagoscopy, there was a tight, inflamed stricture at 22 cms. A biopsy showed vascular granulation tissue only.

The second patient, a woman of 54, had dysphagia for 4 years. X-rays (Figure 8, page 21) show a stricture level with the upper margin of the arch and a hiatal hernia with free reflux. At oesophagoscopy, there was a tight, inflated stricture at 20 cms. A biopsy from it showed ulceration and hyperplastic squamous mucosa. At thoracotomy for repair of the hiatal hernia, the oesophagus from the cardia to the aortic arch looked and felt normal.

In such circumstances we believe that the presence of columnar epithelium in the lower part of the oesophagus may be assumed even though histological proof is lacking.
As has been stated in the section on Radiology, there is a group of strictures intermediate in position between the high and the low ones, where it may be difficult to determine their relationship to the cardia and, consequently, difficult to know if a segment of gastric lined oesophagus is present unless resection of the lesion provides the necessary additional information. At this level, gastric epithelium obtained at biopsy from the site of stricture may have come either from within a hiatal hernia or gastric lined oesophagus below the stricture.

The following is an illustrative case:

A woman of 49 with heartburn for 13 years and dysphagia for 1 year. The x-rays (Figure 21, page 28) show a long rigid-locking stricture. The presence of a hernia is uncertain on radiological examination. At oesophagoscopy, there was a soft, very inflamed stricture at 30 cms. Biopsies showed part of an ulcer base with no epithelium. It was not possible on the available evidence, to establish the relationship of the stricture to the cardia. The patient was operated on. There was no apparent hernia. The oesophagus, for some distance above the hiatus, looked and felt normal but became grossly thickened higher up. The oesophagus was resected from just below the aortic arch to just below the cardia. At the lower end of the specimen there was normal gastric mucosa. Above this, there was thin glandular mucosa of gastric type. Then there was a very short segment of squamous epithelium with gastric glands below it. Above this, there was ulceration with fibrosis in the deeper layers.

**Differential diagnosis.**

The most important differential diagnosis is from carcinoma of the oesophagus. When gastric mucosa is found in a biopsy from a high stricture, the lesion is unlikely to be a carcinoma. Upper oesophageal webs occur in the post-cricoid region and, as has been stated,
we have not encountered a peptic stricture at a level higher than the upper margin of the aortic arch. A high stricture may be difficult to distinguish, radiologically, from a congenital web. Such webs in the middle third of the oesophagus are rare but the following case record illustrates the similarity of such lesions to high peptic stricture.

The patient, a boy of 2 1/2 years, had difficulty in swallowing since weaning. He had episodes of food impaction. The radiographs (Figures 71 and 72) show a web-like stricture at the level of the tracheal bifurcation. At oesophagoscopy, a smooth web was seen. It opened and closed with the phases of respiration. A biopsy showed squamous epithelium without inflammation. At thoracotomy, the oesophagus at the site of the web was soft. The web was excised. Squamous mucosa was present both on its upper and lower aspects, indicating that it was not a high peptic stricture. Similar lesions have been reported by Mark and Goldenberg (1964) and by Gerami and Cole (1970).

**Age incidence.**

In adults, the average age was 62 years. In two instances, although the patients were first seen at the age of 17 and 23, there was a previous history of dysphagia since childhood. The age incidence in adults was the same as for low strictures.

There were 2 babies aged 6 weeks and 11 months, and 3 children under the age of 12 years. Almost half of the peptic strictures in infants and children in the series were at a high level.

**Sex incidence.**

Female patients with high stricture out-numbered males in the proportion of 22 to 9.

**Symptoms.**

The common symptoms were dysphagia and heartburn. We were unable
Radiographs of a boy of 2½ years with a web-like stricture at the level of the tracheal bifurcation.

Strictures level with the upper margin of the aortic arch in two women aged 71 and 73.
to detect any difference between the symptoms associated with high and low strictures. All the adult patients had dysphagia. 18 had heartburn. 3 ceased to have heartburn after the onset of dysphagia. If it is considered that heartburn is the result of mucosal inflammation due to reflux, it is surprising that it should occur so commonly in patients where the lower oesophagus, often from the hiatus to the aortic arch, is lined by columnar epithelium which is thought to be resistant to acid-peptic digestion. Many patients with high stricture had oesophagoscopic evidence of superficial erosions in the squamous mucosa above the stricture but the situation of the heartburn was similar for high and low strictures. Barrett (1957) says that the discomfort associated with a high stricture is felt higher up than with a low one. In general we were unable to make this distinction.

Just as a low stricture may occur after a period of recumbency or an operation so may a high stricture. One patient, a man of 73, began to complain of dysphagia 2 months after cholecystectomy, and was found to have a stricture at aortic arch level. Another patient, a woman of 67, who had heartburn for 9 years, began to have dysphagia due to a high stricture 3 weeks after a period of enforced recumbency as a result of a broken leg.

There was an associated duodenal ulcer in 12 per cent of patients.

High peptic strictures immediately above, level with, and just below the aortic arch are illustrated in Figures 73 to 78.

Radiology

There was invariably gastro-oesophageal reflux and, in the majority of patients, there was a hiatal hernia. In 2 adult patients no hernia was found either on x-ray or at thoracotomy. The situation is different in infants and children where, more often than not, no
Two strictures level with the aortic arch.

Two strictures just below the level of the aortic arch.
hernia was found at thoracotomy. This was the case also in young adults with a history of dysphagia since childhood.

Figure 79 shows an example of the slightly dilated appearance of a gastric lined oesophagus described by Allison and Johnstone (1953). We found this appearance in a minority of cases only.

In 6 adult patients (5 women and 1 man) the stricture was level with the upper margin of the aortic arch but none were found above this level.

**Gastric lined oesophagus above a peptic stricture**

Commonly a high stricture forms at the upper limit of columnar epithelium in the oesophagus. On occasion, a stricture may be found with gastric mucosa above as well as below it. The following are 3 illustrative cases:

This patient, a man of 81, was first seen in 1964. He gave no previous history of heartburn but had had dysphagia for a year. An x-ray in 1963 had shown what appeared to be an ulcer at the cardia. At oesophagoscopy in 1964, a mucosal junction was seen at 30 cms. At 35 cms. there was a tight stricture which was difficult to dilate. A biopsy from 3 cms. above the stricture showed gastric epithelium. The patient was kept going by repeated dilatations. In 1970, biopsies taken from the stricture and from 3 cms. above it both showed gastric epithelium. The presence of a penetrating ulcer within a gastric lined oesophagus and causing severe stenosis was confirmed at subsequent resection of the lesion. (Figures 80 and 97)

The second patient was an obese woman of 64 who was first seen in 1964 and who had complained of dysphagia for 2 months after many years of heartburn. An x-ray in 1962 had shown a hiatal hernia with an ulcer apparently at the cardia. At oesophagoscopy, there was an abrupt transition from pale to red velvety mucosa at 25 cms. The
A high stricture level with the aortic arch with the dilated and rather invert looking oesophagus below it as is sometimes seen in gastric lined oesophagus.

Figure 79

A penetrating ulcer with stricture at 35 cms. A biopsy from 3 cms. above the stricture contained gastric mucosa. The resected specimen is shown in Figure 97.

Figure 80
Cardia was at 34 cms. and there was a stricture at this level. A biopsy taken from immediately below the mucosal junction at 25 cms. showed gastric epithelium. An attempt was made to repair the hernia. For 1.5 cms. above the oesophago-gastric junction, the oesophagus felt normal but above this point it was much thickened. It was mobilized with difficulty. In 1966 the patient was no better. (Figure 81) The stricture was resected with oesophago-jejunostomy. She died 8 days later from a pulmonary embolism. An ulcer causing narrowing of the oesophagus and lying in gastric lined oesophagus was found at thoracotomy. Figure 82 shows the junction of squamous and columnar epithelium at some distance above the oesophago-jejunal anastomosis. As in the previous case, there was no ulceration or stricture formation at the mucosal junction.

A similar ulcer but without significant stricture was found in the third patient. He was a man of 54 complaining of only slight dysphagia but of severe pain radiating through to the back. He suffered from hypertension due to polycystic disease of the kidneys, and, after a haematemesis, became temporarily uraemic. Figure 83 shows a radiograph taken in 1962 showing a small ulcer in the oesophagus and free reflux. At oesophagoscopy the junction of squamous and columnar epithelium was shown by biopsies at various levels to be at 25 cms. There was a deep ulcer at 36 cms. In spite of attempted hernial repair, vagotomy, and gastroenterostomy and the production of achlorhydria, the ulcer progressed relentlessly. Figure 84 shows the progress of the ulcer. The lower oesophagus was resected. The patient died in uraemia. Histological sections showed a chronic ulcer with gastric mucosa both above and below it.

It is considered that these are examples of penetrating ulcer in gastric lined oesophagus. It is of interest that in 2 of the patients
Figure 81 shows a stricture and penetrating ulcer at 34 cms. A biopsy from 25 cms. showed gastric mucosa.

Figure 82 is a photograph of the post mortem specimen showing clearly the squamo-columnar mucosal junction at some distance above the oesophago-jejunal anastomosis.
Radiographs showing the progression of a penetrating ulcer between 1962 and 1964. The ulcer was at 36 cms. Gastric mucosa was found up to 25 cms.

A malignant stricture of the oesophagus with columnar epithelium extending for 5 cms. above it.
95.

this ulcer had given rise to a tight stricture.

Gastric mucosa lining the lower oesophagus may be found not only above a benign ulcer but above a malignant one as is shown in the following case.

The patient was a woman of 60 who had had heartburn for 4 years and dysphagia for 6 months. (Figure 85) At oesophagoscopy, there was a tight stricture at 32 cms. The mucosa above it was like red velvet. Biopsies taken from 32 and 27 cms. showed mucosa of gastric type. At operation, there was a hiatal hernia and a penetrating ulcer at the cardia. The lesion was resected. Section showed it to be a well-differentiated adenocarcinoma. Columnar epithelium extended for a distance of 5 cms, above the malignant ulcer. There was no ulceration or inflammation at the mucosal junction itself.

Gastric lined oesophagus without ulceration or stricture

The findings of gastric lined oesophagus without either ulceration or stricture formation at the mucosal junction may be of importance in relation to the aetiology of the abnormal oesophageal lining. This condition is illustrated by 3 patients.

1) This patient, a woman of 68, was seen in 1967. There was a previous history of perforated duodenal ulcer in 1966 and a long history of dyspepsia. The patient complained of cramp-like pains round her chest coming on particularly during the night and being relieved by alkalis. There was no dysphagia. Figure 86 shows a relatively small hiatal hernia and free reflux. At oesophagoscopy, the mucosal junction was at 30 cms. The level of the cardia was difficult to determine but it was thought to be at 38 cms. There was no evidence of ulceration or oesophagitis. A biopsy taken at 31 cms. contained gastric epithelium.

2) A woman of 63 was seen in 1966 complaining of epigastric pain radiating upwards behind the sternum into her jaws. She, like the
The radiographs shown below are of two female patients in whom columnar epithelium of gastric type was found in biopsies from 31 cms. and 22 cms. respectively.

In both cases there was gastro-oesophageal reflux but in neither was there evidence of oesophagitis or ulceration.
previous patient, complained of cramp-like pains in her chest. An x-ray (Figure 87) shows a small hiatal hernia and reflux. At oesophagoscopy, the mucosal junction was at 22 cms. Biopsies taken from this level and immediately below contained gastric epithelium.

3) This baby aged 4 months had persistent "vomiting" and failed to thrive. An x-ray (Figure 88) showed gastro-oesophageal reflux but no hernia. There was some doubt about the emptying of the stomach. At oesophagoscopy, the mucosa below the level of the aortic arch seemed unusually red. The baby was operated on. There was no hiatal hernia. An attempt was made to render the cardia competent. At the same time the stomach was opened and a catheter passed round the duodenum. It was felt to reach the duodeno-jejunal flexure. It was concluded that there was no significant obstruction in the duodenum. At the same time a biopsy was taken from the oesophagus through the oesophagoscope from just below the aortic arch. This showed gastric epithelium. The baby improved a little but remained underweight and had frequent vomiting. He sometimes vomited food he had eaten a day or two days before. The vomit was sometimes offensive. At this time a year after the original operation a further x-ray (Figure 89) showed a diaphragm across the second part of the duodenum. At laparotomy the second part of the duodenum was exposed. Its upper two-thirds were distended. There was no palpable lesion but when it was opened a web with a very small orifice was found. The web was mobile and it was excised. The histology was as follows:-

"Sections show a web consisting of a thin core of loose fibrovascular tissue covered on both sides by mucosa of small intestinal type which includes a muscularis mucosae." The child has since done well. It is concluded that previously when a large catheter was passed round the duodenum it had pushed the web ahead of it so that the catheter tip
Two radiographs of a baby aged 4 months.

Figure 88 shows gastro-oesophageal reflux. Gastric mucosa was found in a biopsy taken from the level of the aortic arch. At thoracotomy, there was no apparent hernia.

Figure 89 shows the presence of a diaphragm across the second part of the duodenum.
could be palpated at the duodeno-jejunal flexure giving rise to the erroneous impression that there was no duodenal obstruction. As has been described on Page 59 one can be deceived in much the same way in the case of lower oesophageal web.

Does columnar epithelium ascend the oesophagus?

Although it has been confidently stated (Hayward 1961) that the oesophagus may become lined by columnar epithelium by a process of upward migration of this type of mucosa, we are not aware of any instances where its ascent has been actually observed. In our patients with gastric lined oesophagus but no stricture we have not noted any change in the level of the mucosal junction over periods of 3 and 4 years respectively. In patients with peptic stricture followed up over many years we have not observed ascent of the stricture except possibly in the following 2 patients.

Case 1 : A woman of 59 was seen in 1962 with a previous history of duodenal ulcer and heartburn. She had complained of dysphagia for 9 months. In 1962 there was a stricture at 29 cms. In 1966 and again in 1969 it was found to be at 26 cms.

Case 2 : This patient, a woman of 71, was seen in 1967 with a long history of heartburn and dysphagia for 2 years. In 1967 there was an irregular, ulcerated stricture at 30 cms. In 1968, it was at 29 cms. and, in 1969, at 28 cms.

These two patients were the only ones where there seemed to be some ascent of the stricture. Unfortunately, there is no radiological confirmation and the accuracy of the endoscopic measurements may be open to question.

Gastric lined oesophagus and mental defect

A feature of this series is the high incidence of congenital abnormalities, especially mental defect, in children and young adults
with peptic stricture.

The following are cases showing such defects:-

**Case 1**: (Figures 90 and 99) A girl of 12 years who was mentally retarded and displayed repetitive purposeless movements as a result of phenylketonuria. Her peptic stricture was resected.

There was no hiatal hernia but the oesophagus up to the level of the stricture which was midway between the hiatus and the aortic arch was found to be lined by gastric epithelium.

**Case 2**: (Figures 123 - 125) This boy, aged 8, who had an ulcer in a gastric lined oesophagus is also a deaf mute.

**Case 3**: (Figure 20) This 17 year old youth is mentally defective. At operation, no hernia was found. The stricture was resected. Histological section showed gastric mucosa lining the oesophagus below an ulcerated stricture.

**Case 4**: (Figure 36) This mentally deficient patient, aged 21, had a high peptic stricture a considerable distance above the cardia.

A biopsy from the stricture showed inflamed granulation tissue only but it is assumed that the oesophagus below the stricture is lined by columnar epithelium.

**Case 5**: (Figure 122) A physically and mentally handicapped child of 8 who was proved at operation for resection of her stricture to have no hiatal hernia but a lower oesophagus lined by columnar epithelium.

Of 13 patients with high stricture in the age group 0 to 30, six were found to have other defects, most commonly mental defect. We are unable to say whether this incidence of mental defect is a significant finding or whether it merely reflects the interest taken by the local mental health officers in oesophageal lesions.
A high peptic stricture and lower oesophagus lined by columnar epithelium in a girl aged 12 suffering from phenylketonuria.  

The resected specimen is shown in Figure 99.

An abnormal oesophagus in a grossly deformed girl of 18, suffering from microcephaly and spastic paraplegia.  

An oesophageal biopsy has not been obtained.
Clinical findings in relation to the aetiology of gastric lined oesophagus

What light do the clinical findings throw on the lower oesophagus lined by columnar epithelium? Positive evidence from this series that the lesion is an acquired one is slender. It depends on the 2 patients already described where a stricture appeared to ascend as seen at oesophagoscopies repeated at intervals over a period of years. As has been said, we have reservations about the accuracy of the findings in both cases. In none of the remaining 144 patients was ascent of a stricture observed. If the presence of columnar epithelium in the oesophagus is due to the fact that healing of ulceration is achieved by columnar rather than squamous epithelium, it is surprising that ascent of an ulcerated stricture is not more often seen. Also, in 2 of our patients where healing of an ulcer occurred after hernial repair and where smooth fibrous strictures remained and were subsequently resected, healing was by squamous epithelium.

The findings in a patient described later (Page 106) where mucosa of jejunal type was found above an oesophago-jejunal anastomosis may support the theory of upward ascent of the abnormal mucosa.

One piece of evidence in favour of the theory that the abnormality is an acquired one is of a negative nature. Although we have described the case of a baby of 4 months with a gastric lined oesophagus and no ulceration or stricture, we have not had the opportunity to ascertain that the condition is found at autopsy in still-born babies or neonates.

The following clinical findings appear to support the congenital theory. The condition is found in babies. We found it in a baby aged 3 months who had been regurgitating altered blood since the age of 6 weeks. (Figures 104 and 105) At oesophagoscopy in 1968, a
tight stricture was found. It bled readily when touched. The child did not thrive. He suffered from chronic anaemia. The stricture was resected early in 1970. There was no hiatal hernia. The stricture lay midway between the hiatus and the aortic arch. On histological examination, the oesophagus below the stricture was found to be lined by columnar epithelium. It seems unlikely that a complete and regular gastric lining could have taken over from squamous epithelium in the short time between birth and the finding of the stricture at the age of 3 months. Also no ascent of the columnar epithelium was observed over a period of 2 years between the discovery of the stricture and its resection.

Probably of more significance is the case of the baby aged 4 months who, as described on page 97 (Figures 88 and 89) had a gastric lined oesophagus without evidence of ulceration or stricture formation, who had no hiatal hernia, and who had a congenital web in the second part of the duodenum. In the absence of oesophageal ulceration there is no apparent reason for the ascent of columnar epithelium and it is difficult to escape the conclusion that it was a congenital anomaly especially as it was associated with a congenital duodenal diaphragm.

Two adult patients have been described in whom there was columnar epithelium up to the level of the aortic arch without any evidence of ulceration. Again it is suggested that, in the absence of ulceration, there would seem to be no cause for columnar epithelium to ascend the oesophagus.

Another group of patients in whom it is difficult to explain the presence of the abnormal epithelium as an acquired condition is that in which it is found not only below an ulcer but above it. Such patients have been described on pages 90 - 92 together with 1 patient in whom gastric
lining was found above a malignant ulcer. (page 95)

A striking feature at thoracotomy is that the oesophagus below the stricture looks and feels normal. There is no mural thickening or peri-oesophagitis. There are no signs of residual changes such as one would expect if there had been recurrent ulceration in the portion of oesophagus now lined by columnar epithelium.

The final proof of the congenital nature of the anomaly must lie in its discovery at autopsy in a proportion of new-born babies. To our knowledge, this information is not available. However, Rector and Connerley (1941) found 78 cases of heterotopic gastric mucosa in the oesophagus in 1000 consecutive necropsies in children. The mucosa was of cardiac type with no oxyntic cells in 63 of the 78 cases.

If gastric lined oesophagus is a congenital anomaly, one would expect to find it occasionally associated with other congenital defects. An association between hiatal hernia and inborn metabolic error and mental defect was suggested by Moncrieff and Wilkinson (1954). Abrahams and Burkitt (1970) reported an abnormal barium swallow in 29 of 77 children suffering from cerebral palsy. 13 had a hiatal hernia and 16 had gastro-oesophageal reflux without a demonstrable hernia. Figure 91 (page 101) is a radiograph of an abnormal oesophagus in a mentally defective patient with kyphosis, scoliosis, and inco-ordination of swallowing who is all the time recumbent because of her severe physical deformities. She suffers from grand mal epilepsy, microcephaly, and spastic paraplegia.

It is of interest that in the early paper by Brown Kelly (1936) in the section entitled "Progressive Narrowing and Shortening of the Oesophagus with Hiatal Hernia", one of the two children described was a mentally defective girl aged 2 1/2 years. Incidentally it is pleasant
to observe his use of the word "snippet" instead of the less
descriptive term "biopsy" now in universal use.

We have described several children and young adults with
peptic stricture and with other defects namely inborn metabolic error,
and duodenal diaphragm, and mental defect. The numbers are small.
We are unable to say whether there is an association between lower
oesophagus lined by columnar epithelium and certain genetically
determined abnormalities or whether oesophageal abnormalities in the
type of patients mentioned are the result of factors such as extensor
spasm, inco-ordination of deglutition accompanied by aerophagy, kypho-
scoliosis, or prolonged recumbency as suggested by Abrahams and Burkitt.
It is of interest that Shine and Allison (1966) reported a family with
tylosis, which is an expression of a single autosomal gene in hetero-
ygous state, in which both the father and a son, had gastric lined
oesophagus.

The secretory activity of columnar lined oesophagus

The problem of the secretory activity of the columnar
epithelium in the oesophagus remains unsolved. As all patients with
a stricture at the upper limit of a gastric lined oesophagus have
reflux either with or without a hernia, it is difficult to examine the
oesophagus in isolation from the secretory point of view. The problem
is of practical importance. If the columnar epithelium is secreting
digestive juices in significant amount, it is unreasonable to treat
the stricture by control of reflux alone. In practice, however, repair
of hernia with the prevention of reflux is often an effective treatment
of high strictures in adults. In children we have found it much less
successful but there is no evidence that failure was due to the secretory
activity of the columnar epithelium. In adults, we believe that the
secretory activity of the abnormal oesophageal mucosa can be ignored as
long as competence at the cardia is achieved.

If the abnormal epithelium is actively secretory, it would seem logical to remove it completely. This involves an extensive procedure if the stricture is at the level of the aortic arch. In our series, we had no patient with a stricture at this level where resection was necessary. However, the problem presented itself in cases of ulcer and stricture within a gastric lined oesophagus. We have divided the oesophagus at a convenient distance above the lesion without making efforts to reach squamous epithelium. This seems satisfactory in practice. Cases are reported of stricture formation at oesophago-jejunal and oesophago-colic anastomoses. The question arises as to whether the anastomotic stricture is the result of technical error with partial break-down at the suture line or to the fact that gastric type mucosa remained adjacent to it. It seems likely that the former explanation is the correct one and that total removal of the abnormally lined portion of oesophagus is unnecessary.

Some years ago, we published an account of a case which might have shed light both on the origin of columnar epithelium in the oesophagus and its secretory activity had more information been obtainable (Davidson, 1964). This was a case of high peptic stricture developing after total gastrectomy in a patient suffering from the Zollinger-Ellison syndrome. The patient was a man of 54 who had recurrent gastric ulceration and a tumour of the pancreas. In 1951 a perforation of a duodenal ulcer was closed. In 1952 a partial gastrectomy was done followed by a higher partial gastrectomy in 1953. In 1956 a gastro-jejuno-colic fistula was closed. A total gastrectomy was done for a very large gastric ulcer in 1957. A jejunal loop was brought up in the manner of Roux. Unfortunately, an oesophageal biopsy was not obtained at this stage. Four-and-a-half years after
total gastrectomy, the patient developed dysphagia and a barium swallow showed a peptic stricture level with the aortic arch (Figure 94). The patient died 6 years after the total gastrectomy, the pancreatic tumour being a malignant one. Before death examination of oesophageal secretion had not shown the presence of hydrochloric acid. At autopsy, the patient was found to have an ulcerated stricture at the upper limit of a gastric lined oesophagus (Figures 92 and 93). Histological examination showed that the columnar epithelium resembled small intestinal mucosa rather than gastric mucosa. There were many goblet cells and Paneth cells but no oxyntic cells (Figure 95).

From the point of view of the secretory activity of columnar epithelium in the oesophagus it is interesting that a high stricture developed in the absence of gastric acid. It is tempting to postulate that the columnar epithelium was producing digestive juice under the influence of the ulcerogenic tumour of the pancreas. On the other hand no hydrochloric acid was found in the oesophagus. As to the origin of the columnar epithelium no conclusions can be drawn as a biopsy from the oesophagus was not obtained initially. It is of interest from the point of view of possible upward migration of columnar epithelium that the lining of the lower oesophagus resembled small intestinal mucosa and was found above an oesophago-jejunal anastomosis.

Although, judging from this series, the evidence seems to be weighted in favour of the view that lower oesophagus lined by columnar epithelium is a congenital anomaly, it would probably be unwise at this stage, to discount the theory that it may be acquired.
Radiological and macroscopic appearances of a high stricture of the oesophagus which occurred 4\(\frac{1}{2}\) years after total gastrectomy. The diagram of the specimen shows, from above downward, squamous epithelium, an ulcerated stricture, oesophagus lined by columnar epithelium, and the jejunal loop.
Histological section of the oesophagus between the stricture and the oesophago-jejunal anastomosis. The epithelium resembles small intestinal mucosa rather than gastric mucosa.

Figure 95
Complete histological examination was possibly only in the more severe and especially the grossly ulcerated lesions where the stricture was resected. In most cases histological examination was limited to study of oesophageal biopsies which give an incomplete picture of the lesion.

A review of the biopsy reports indicates the type of information given by oesophageal biopsy.

1) They helped to exclude the presence of carcinoma. Too much reliance should not be placed on one negative biopsy.

2) A number of biopsies from the site of stricture showed part of the mucosal junction. Such a biopsy may be obtained at any level from that of the hiatus to the upper margin of the aortic arch. Its importance is that it establishes the fact that the stricture lies at the mucosal junction and that it is, therefore, likely to be due to reflux and to respond to surgical control of reflux. The findings of squamous mucosa in the biopsy from the stricture has the same significance.

3) If gastric mucosa is obtained from the stricture but not from the oesophagus above it, its significance depends on other considerations. If obtained from a low stricture or one midway between the hiatus and the aortic arch, it may merely indicate that it has been obtained from within a herniated portion of stomach. If it is obtained from a stricture in the region of the aortic arch and well above the sliding hernia, it indicates the presence of lower oesophagus lined by columnar epithelium.

4) Gastric mucosa may be present in a biopsy taken some distance above a stricture. This suggests the presence of an ulcer causing stenosis within a gastric lined oesophagus. In general it is good policy to take a biopsy not only from the stricture but from above
it in order to distinguish between lesions occurring at the squamo-columnar junction and those developing in a gastric lined oesophagus. It is not possible from the naked eye endoscopic appearances alone to determine the upper limit of columnar epithelium in some cases.

5) Frequently granulation tissue or material resembling an ulcer base was found at biopsy. Though establishing the presence of ulceration, the biopsy gives no indication of its extent or depth.

A great deal has been written about the pathology of peptic stricture and different interpretations have been put on the findings. We cannot hope to add anything which previous detailed studies have failed to note. The following illustrated account will, however, clarify the types of lesion with which we have been dealing.

Lower oesophageal webs have not been included in the series because of the doubt about their aetiology. Those we have seen have all shown the same histological appearances. A slightly thickened layer of squamous epithelium is in continuity with mucosa of upper gastric type at the rim of the orifice. The underlying connective tissue shows some chronic inflammatory infiltration. The muscularis mucosae sometimes appears to be hypertrophied. The lesion forms a tough diaphragm. It is usually not palpable when the oesophagus is exposed and there is no external evidence to indicate its presence.

Cases of peptic stricture treated by resection or coming to autopsy fell into the following groups in adults.

1) Localized penetrating ulcer at the mucosal junction.

Figures 96 and 32 show the macroscopic and radiological appearances of this lesion. At thoracotomy, there was a sliding hemia. Just above the oesophago-gastric junction the oesophagus was thickened and felt stony hard. There was an external depression indicating the site of the ulcer. The oesophagus was thickened for
The macroscopic appearances of a localized penetrating ulcer at the squamo-columnar mucosal junction.

The corresponding radiograph is shown in Figure 32 (page 34).
some distance above but was relatively soft. There was extensive chronic mediastinitis with enlarged glands adjacent to the ulcer.

The resected specimen showed an extensive ulcerated area at the oesophago-gastric junction with, in the middle of it, a deeper ulcer 1 cm. in diameter. There was thickening of the adjacent wall. As shown in Figure 96, the mural thickening is limited to the region of the ulcer.

**Histology.** Longitudinal section showed an extensive area of ulceration varying in depth and bordered above by stratified squamous epithelium and below by gastric type epithelium in which a few glands were present. The ulcer floor consisted of debris and acutely inflamed granulation tissue beneath which there was marked fibrosis. At the site of the deep ulcer, the whole wall was replaced by fibrous tissue.

**Comment** This type of ulcer looks like a gastric ulcer. As its lower margin is formed by gastric mucosa and its upper margin by squamous epithelium, it is difficult to know in which type of mucosa it originates.

We have seen one similar ulcer where vestiges of squamous epithelium were found together with gastric mucosa at its lower margin. Sandry (1962) has described healing of such an ulcer by squamous epithelium. In 2 patients to be described later (Page 154) healing of a penetrating ulcer following hernial repair was by squamous epithelium. Such findings suggest that this type of ulcer though looking like a gastric ulcer may have originated in squamous mucosa adjacent to the mucosal junction.

2) **Penetrating ulcer in a gastric lined oesophagus**

Figures 97 and 80 show the macroscopic and radiological appearances.

At oesophagoscopy, the ulcer was seen to be causing a tight,
Figure 97

A penetrating ulcer within a gastric lined oesophagus. Columnar epithelium extends for some distance proximal to the upper margin of the ulcer. The corresponding radiograph is shown in Figure 80.
hard stricture at 35 cms. Biopsies taken at 35 and 33 cms. both showed gastric mucosa. At operation, there was a sliding hiatal hernia. Some distance above the oesophago-gastric junction there was a hard mass 3.5 cms. in length in the oesophagus. There was dense peri-oesophageal fibrosis.

Histology. Longitudinal section showed columnar epithelium extending from the proximal edge of the resected specimen to the ulcer and also distal to it. Proximal to the ulcer the glandular epithelium formed simple mucosal glands lined by columnar and goblet cells. Distal to the ulcer, similar epithelium was seen. It contained cardiac glands. Typical gastric glands were found lower down. The ulcer floor consisted of debris and acutely inflamed granulation tissue deep to which fibrosis had occurred extending through the whole wall.

Comment This type of ulcer occurring in lower oesophagus lined by gastric epithelium is identical with a gastric ulcer. It has been said to cause stenosis of the oesophagus only rarely but, in our experience, it may cause a hard, irregular, intractable stricture. A localized penetrating ulcer is associated with palpable thickening of the oesophagus for some distance above and below it just as a deep lesser curve ulcer causes thickening of the curvature distal and proximal to it. However, the length of the oesophagus involved in mural thickening is less than with ascending superficial ulceration. Typically a penetrating ulcer gives rise to a localized, stony hard area in the oesophagus which is not felt in the case of superficial ulceration.

3) Superficial ulceration.

(Figures 98 and 21) At oesophagoscopy, there was a tight, rather firm stricture at 29 cms. A biopsy showed a distorted fragment of granulation tissue with mixed inflammatory infiltrate.
Resected specimen showing superficial ulceration. The ulcerated area is 5 cms. in length.

The corresponding radiograph is shown in Figure 21 (page 28).
The external appearances of the lesion were those of a firm, swollen segment of oesophagus extending from the level of the inferior pulmonary vein to the aortic arch. There was peri-oesophagitis. No hiatal hernia was found.

The resected specimen consisted of a shallow ulcer 3 cms. in length. Longitudinal sections of the whole specimen were taken.

Histology. Sections showed the following changes from below upwards. There was some normal gastric mucosa with parietal cells. Above this there was a rather thin glandular mucosa of modified gastric type. Then there is squamous epithelium with gastric glands below it. Further up there is patchy ulceration with fibrosis of the deeper tissues and some abscesses. Scattered below the ulcerated area were dilated glands with mucus salivary type epithelium and some squamous metaplasia which were situated below the muscularis mucosae. Following along the ulcerated surface there were several traces of thin layers of squamous epithelium occurring from 3 to 6 times in the various sections. The deep muscle layers appeared normal and ganglion cells were readily found.

Comment The lesion appears typical of ascending superficial ulceration occurring in squamous epithelium. Although the deeper muscle layers are reported as histologically normal, they are, as shown in the photograph, considerably thickened. Again, the mural thickening is limited to the area of mucosal loss.

4) Long superficial ulceration with deeper penetrating ulcer
(Figures 99 and 90) At oesophagoscopy, there was a tight, hard stricture at 28 cms. At thoracotomy, the oesophagus felt hard and thickened from the oesophago-gastric junction to the level of the aortic arch. There was no apparent hernia.

The resected specimen which was much shrunken consisted of 5.5 cms.
Macroscopic appearance of a long superficial ulcer with deeper extensions of ulceration in various places.

The corresponding radiograph is shown in Figure 90 (Page 101).
of oesophagus and 4 cms. of stomach. The wall of the oesophagus was much thickened and the mucosal surface was ulcerated for a distance of 4 cms. proximal to the gastric epithelium.

A longitudinal histological section showed the lower oesophagus ulcerated with squamous epithelium above and gastric epithelium below the ulcer. Superficially there was inflamed crusted and deep to this there was considerable fibrosis involving all coats and replacing much of the circular and longitudinal muscle. Chronic inflammatory cells and lymphocytes were scattered diffusely in the fibrous tissue with occasional lymphocytic nodes.

Comment
This lesion is a long ulcer with total mucosal loss but in addition, unlike the previous case, there is considerable involvement of the deeper layers.

The photograph of the specimen shows that the thickening of the oesophageal wall is confined to the area deep to the ulcer.

5) A stricture typical of those found occurring soon after an operation for duodenal ulcer.

Figure 100 shows the radiological appearances.

Histological section showed an ulcerated area bounded distally by gastric and proximally by squamous epithelium. Deep to the ulcer there is considerable fibrosis with chronic inflammatory infiltrate extending into and replacing much of the muscle layers.

Comment
In patients with post-operative peptic stricture where the lesion was resected we have found that, in every case, there was evidence of deep ulceration with fibrous replacement of the muscle layers.

6) Penetrating ulcer leaving a stricture after healing.

Figure 33 (page 35) is an x-ray showing the initial ulcer.
A penetrating ulcer (Figure 33, page 35) had healed after attempted hernial repair leaving a tight, fibrous stricture. Healing was affected by the squamous mucosa.
At oesophagoscopy when the patient was first seen there was a stricture at 37 cms. A biopsy showed a portion of ulcer base.

The hernia was repaired with difficulty. There was a hard mass just above the oesophago-gastric junction.

Figure 101 shows a further x-ray after attempted hernial repair. There is now a smooth, tight stricture. This lesion was resected. The specimen consisted of a much thickened portion of oesophagus. Histological examination showed the presence of a healed ulcer at the site of stricture with dense fibrosis involving all coats of the oesophagus and replacing the circular and longitudinal muscle layers. The ulcer had healed by a covering of squamous epithelium.

Comment

A penetrating ulcer at the mucosal junction may heal after hernial repair but, as happened in 2 of our patients, a tight, fibrous stricture may be left. In both cases healing was by squamous epithelium.

Pathological types of Peptic Stricture in Children

1) Penetrating ulcer in a baby aged 2 months

Figure 102 shows the macroscopic appearances. This ulcer perforated. Its upper margin was formed by squamous and its lower margin by columnar epithelium.

2) Figure 103 shows a superficial ulcer in a child of 2 years with no hiatal hernia. The lesion was resected.

The opened specimen showed a lining of white squamous epithelium at the upper end and gastric mucosa at the lower end. The middle section had an irregular, shallow, serpiginous ulcer with small, white islets of squamous epithelium in the ulcerated area.

The specimen was divided into 7 longitudinal strips for section. The sections all showed areas of shallow ulceration sometimes recurring in 3 foci in a longitudinal strip. Each area of ulceration
A large penetrating ulcer in a baby 2 months old. This ulcer perforated.
Resected specimen from a mongol boy 2 years old showing a shallow, serpiginous ulcer with islets of squamous epithelium in the ulcerated area. The Radiographs of this patient are shown in Figures 118, 119, 120. Page 157.
was flanked above and below by squamous epithelium. At the lower end, the squamous epithelium merged with a modified glandular mucous membrane which led below into normal gastric (cardiac) mucosa. The ulcers had a floor of granulation tissue and varying amounts of chronic inflammatory cell infiltrate forming a few lymphoid follicles and interrupting the muscularis mucosae. Deeper, there was fibrous thickening of the sub-mucosa and below the central area of ulceration some fibrosis broke up the fibres of the circular muscle. Otherwise the muscle layers were normal. The myenteric plexus, with nerve fibres and ganglion cells was prominent.

3) **Superficial ulceration with deeper extensions.**

Figures 104 and 105 show the radiological and macroscopic appearances of such a lesion in a boy of 2 years.

At oesophagoscopy there was a stricture. At thoracotomy, the oesophagus felt thickened from the level of the inferior pulmonary vein to the aortic arch. There was gross chronic peri-oesophagitis.

Longitudinal sections showed a patchily ulcerated area commencing at the squamo-columnar junction and extending proximally. The areas of patchy ulceration were bounded by regenerating squamous epithelium and the ulcer floors were composed of inflamed granulation tissue deep to which there was fibrosis extending into the muscle layers.

**Summary of the Pathological Findings**

Our findings are similar to those of Sandry (1962) who has given an excellent description of the pathology of chronic oesophagitis. As in his series, our patients showed the following types of lesion, namely, localized penetrating ulceration with varying degrees of replacement and destruction of the muscle coats, long superficial ulcers with total mucosal loss, and a combination of these two.

In adult patients in whom resection was necessary, the numbers
Illustrations of an ulcerated stricture in a boy 2 years old.

Histologically, the lesion was mainly one of superficial ulceration but in places, deeper extensions of ulceration were seen.

The radiological appearances had remained unaltered since the child was three months old.
of penetrating and superficial ulcers was almost equal. In children,
superficial ulcers appeared to be more common than penetrating ulcers.

Although the concept of "ascending fibrosis" as described by
Brown Kelly (1939) is generally accepted, the resected specimens from
our series show the mural thickening to be localised to the area of
oesophageal wall underlying the ulcer. Thus in the case of a penetrating
ulcer the thickened oesophageal segment is usually relatively short
whereas it is much longer when there is a long superficial ulcer.
Information regarding the natural history of peptic stricture has been gained, in this series, from consideration of the previous histories of the patients and also from their subsequent follow-up.

When a baby develops a stricture, the lesion tends to be a severe one and surgical treatment is often necessary within a short time. In most instances, there is no opportunity to study the natural history of such lesions over a prolonged period. In this respect the behaviour of strictures in babies differs from that commonly seen in adults.

Judging from the small group of patients who presented with increasing dysphagia in early adult life but who had a previous history of some difficulty in swallowing since childhood, it would seem that if an infant or young child has a stricture which is not bad enough to require surgery more severe symptoms may be anticipated after an interval of 20 to 30 years. Apart from his patients who developed a stricture following parturition where there had been previous hyperemesis gravidarum, Abbey Smith (1965) found that, in all his cases where dysphagia was due to a peptic stricture between the ages of 20 and 35, the condition was a relapse of one established in infancy. We would add, however, that post-operative peptic stricture may occur in young adults, usually in men who have had an operation for duodenal ulcer.

The majority of patients presented in the sixth decade. It must be accepted that advancing age has a profound influence on the development of the lesion although the manner in which it does so remains obscure. The fact that the patient eventually comes to hospital because of dysphagia does not mean that this symptom is going to be progressive. Indeed, a majority of patients come to hospital because of a temporary exacerbation of the inflammatory element in the stricture.
and may subsequently be kept going quite well with only occasional
dilatations of the stricture and without any oesophagoscoptic evidence
of its progression.

Judging from a study of patients with a previous history of
dysphagia of 5 years or more, most strictures seem to retain the same
caracter over long periods of time with only intermittent variations
in the severity of the dysphagia. In only a minority of cases do the
strictures become more difficult to manage. In some instances there is
an apparent reason for the change, for example an abdominal operation
or a period of recumbency. Other exceptions to the general rule that
strictures remain true to type are to be found especially in the seventh
decade. It is not uncommon to find strictures in this decade developing
deeper ulceration, beginning to give rise to persistent pain, and
becoming difficult to manage by conservative measures.

As has been said, some strictures where there is a short
history of dysphagia are difficult to manage from the outset, in
particular post-operative strictures and those following soon after a
haematemeses.

Information regarding the natural history was gained by the
follow-up of 56 patients observed over a minimum period of 4 years, in
whom the management was by intermittent dilatation. Again, most of
these strictures remained true to type. A soft, inflamed stricture is
likely to remain so and one which is initially firm and irregular will
continue as such. As has been observed in relation to hiatal hernia
and reflux, each patient seems to exhibit an individual response. If
this response involves stricture formation the type of stenosis which
forms tends to remain characteristic for that patient. Indeed, it may
be said that, if one has been carrying out repeated oesophagoscopies
on a patient, one comes to recognise his stricture almost as one
recognises his face.

There are, of course, exceptions to this general rule. During follow-up, the stricture appeared to become progressively harder in 5 patients. In 2 patients it was found to change from a hard to a soft stricture. In 3 patients followed up over periods of 8, 9, and 10 years respectively the stricture seemed to vary in character between soft and hard at different times. Once a penetrating ulcer has formed, either at the squamo-columnar junction or within a gastric lined oesophagus, the lesion tends to be progressive. Such exceptions do not alter the fact that in general, a peptic stricture tends to retain its initial characteristics.

An important feature of the natural history from the point of view of treatment is that most patients present with an established stricture. In this series, a stricture developed during the period of follow-up in only 3 patients.

**Case 1**: The patient, a woman of 60, was first seen in 1960 with a history of heartburn and acid regurgitation. An x-ray in 1960 showed a hiatal hernia with reflux. At oesophagoscopy, the appearances suggested the presence of a gastric lined oesophagus but no biopsy was taken. There was no stricture. The patient broke her leg in 1967 and was confined to bed for 3 weeks. Soon afterwards she complained of dysphagia. When x-rays were taken in 1970 (Figures 28 and 29 - page 32) there was an acutely inflamed stricture at 20 cms. This stricture was presumably the result of recumbency and its development could not have been anticipated.

**Case 2**: The patient, a woman of 53, was found to have a hiatal hernia in 1955. She was treated medically. She began to complain of dysphagia in 1961. In 1962 there was an acute oesophagitis and a slight stenosis at 36 cms. In 1963 there was a tight stricture
which was difficult to dilate. (Figure 106)

Case 3: This patient, a summary of whose case record is given on Page 106 developed a high peptic stricture during follow-up 4½ years after a total gastrectomy for Zollinger-Ellison syndrome.

An interpretation of the natural history of oesophagitis is essential for the formulation of a logical plan of treatment. Much depends on which of the following views one accepts. Collis (1969) says that "generally patients present with the type of response which will always be characteristic of their case". "Operation is not indicated to prevent the development of stricture". Again in 1970, Collis states that "occasionally a patient with diffuse oesophagitis progresses to stricture formation but generally a stricture patient presents with his stricture". On the other hand, many authorities take the view that oesophagitis is in so many instances a progressive lesion that surgical treatment is called for at an early stage. This view gives rise to the concept of "the neglected hiatal hernia" described by, among others, Burdette Nelson (1969). He notes the incidence of strictures in older patients and feels that they should be prevented by operation on all symptomatic hernias in whom significant reflux is demonstrated. Harnan et al. (1953) consider that the proper treatment of chronic oesophagitis and its sequelae is prevention — in short reduction and repair of the hernia before irreparable damage has occurred.

In the face of such irreconcilable points of view, it is obviously difficult to arrive at a confident opinion as to the correct treatment of oesophagitis. The difficulty is increased by the fact that oesophagitis is not always easy to diagnose and that its natural history can be appreciated only by following up patients over long periods of time.
A radiograph taken in 1963.

The patient was found to
have a hiatal hernia in 1955.
Dysphagia began in 1961.
From our series we may make the following observations.

1) Infants, if they are going to form a stricture, do so at an early stage. Those with reflux only when they are first seen rarely develop a stricture and may be successfully treated by medical measures. There is, in our view, little opportunity in practice for the prevention of stricture in infants by surgery. Only a small number of patients are encountered in the second and third decades where there is a history suggesting that the stricture was established in infancy. Their number is too small to constitute an indication for operation in infancy to prevent reflux.

2) Post-operative and post-partum strictures are of rapid onset and their occurrence cannot be anticipated.

3) Most of our patients with stricture presented as such. There was no opportunity for prevention of stricture by earlier surgery. The very small number of patients in whom progression from oesophagitis to stenosis was observed have been described.

4) The main difficulty is the natural history of the lesion in the older age groups. As has been pointed out, strictures are relatively common in the aged and may be difficult to treat. In theory, it would seem that they should be preventable. In an effort to find out what happens in practice, we have consulted the case records of 63 patients of 65 years of age or more. The following facts emerge.

In 10 patients, the stricture followed an operation or period of recumbency and could not have been anticipated.

23 patients gave a previous history of heartburn but they presented with dysphagia and had not previously been investigated.

16 patients who had no previous history of indigestion or heartburn presented with dysphagia.

Some patients who presented with dysphagia were known to have
a duodenal ulcer (5) or disease of the biliary tract (4) but had not previously been shown to have a hiatal hernia.

1 patient presented with food impaction.

In 3 patients the dysphagia followed soon after a haematemesis there having been no previous investigations.

1 patient who had always had "a narrow swallow", came to hospital because of some increase in his dysphagia.

It must be concluded that while, in theory, prevention of stricture should be possible, the opportunity to do so rarely occurs.

The question remains not so much how to prevent stricture as how to avoid the situation where, although it may remain easily manageable by conservative measures for many years, it becomes a serious problem when the patient is old and possibly unfit for major surgery. This is a difficult problem which we have not solved.

Many factors have to be taken into account. Initially, although a stricture is manageable by dilatation, it might require resection if surgery were to be undertaken. This involves some mortality and morbidity and only a few strictures become worse in old age. The patient may die from some other cause in the meantime. In the present state of our knowledge, there are many imponderables when we attempt to look into the future of a peptic stricture. Just as we have not made efforts to anticipate a stricture surgically, so we have not so far undertaken surgery for one which was being managed successfully by conservative measures on the assumption that it would get worse in old age.

**Malignant change**

It is of obvious importance in the natural history of peptic stricture to try to assess the incidence of malignant change. As with gastric ulcer, this is difficult to do and divergent views have been
expressed.

The following 3 cases illustrate the problem.

**Case 1**: This man was seen in 1957 at the age of 61. He had a long history of heartburn and indigestion. Dysphagia began 1 year before. An x-ray showed a hiatal hernia and a stricture at the cardia. The stricture was confirmed at oesophagoscopy. It was at 32 cms. A biopsy showed no evidence of neoplasm. The lesion was considered to be a peptic stricture and was treated by dilatations which were required in 1957, 1959, and 1960. At the next dilatation in 1963 the stricture had become harder and tighter. Dilatation was needed 3 times in 1964, once in 1965, 3 times in 1966, and once in 1967. In 1967 the patient suffered perforation of a duodenal ulcer. In 1968, a biopsy from the stricture showed for the first time the presence of adenocarcinoma. Death occurred 13 years after the onset of dysphagia.

**Case 2**: This patient complained of dysphagia in 1954 when he was 71 years old. An x-ray was reported as showing a hiatal hernia and a stricture level with the aortic arch. It was found to lie at 21 cms. Two biopsies showed hyperplastic squamous epithelium and granulation tissue. Dilatations were carried out in 1955, 1956, and 1957. All biopsies were negative. The patient then swallowed quite well until 1964 when he returned with a hard, irregular stricture at 22 cms. A biopsy showed a poorly differentiated adenocarcinoma. The patient died 10 years after the onset of dysphagia.

**Case 3**: A woman of 40 was first seen in 1964 (Figure 107, page 131). She gave a history of dysphagia since childhood. Repeated dilatations were done until she was 18 years of age. In 1964 the difficulty in swallowing became worse. At oesophagoscopy, an adenocarcinoma was found. At operation there was a hiatal hernia and a growth extending upwards from the cardia.
It would be surprising if a lesion such as peptic stricture which is often associated with ulceration were not, on occasion, to undergo malignant change. There are, however, difficulties in establishing proof of such change in any individual patient. A carcinoma of the oesophagus in an elderly man may be a very slow growing tumour. This is especially so in the type of neoplasm giving rise to annular stricture resembling a peptic stricture both radiologically and at oesophagoscopy. A negative oesophageal biopsy may merely mean that it has been taken from the wrong site. Even with periods of survival of a number of years after the onset of dysphagia it is difficult to be sure that the lesion was not a malignant one from the beginning. What interval has to be allowed between the first difficulty in swallowing and histological proof of malignancy for a definite diagnosis or malignant change in a peptic stricture to be made seems quite arbitrary. Certainly an interval of 2 or 3 years is inadequate.

In all our patients where there was a question of malignant change, the eventual histology was that of adenocarcinoma. We believe that oesophagitis due to reflux occurs primarily in squamous mucosa. In theory, the subsequent neoplasm should be a squamous cell carcinoma as occurs following corrosive stricture. In many patients with oesophageal ulceration, the lower margin of the ulcer is formed by gastric mucosa and the upper margin by squamous mucosa. Presumably both are subject to chronic irritation. The explanation of why all the lesions are adenocarcinomas is not apparent. The fact that this was so adds to the uncertainty regarding malignant change.

In cases 1 and 2, even though there were periods of 13 and 10 years between the onset of dysphagia and death from adenocarcinoma and the findings were very suggestive of malignant change, it is by no means certain that absolute proof of such change has been established.
In case 3, where malignant change appeared to have occurred in a stricture of 40 years duration, proof is again lacking as no previous films were available to establish the fact that the carcinoma had arisen at the site of the previous stricture.

We have described 1 patient with a malignant ulcer in a gastric lined oesophagus. There were no previous x-rays to show if there had been a benign ulcer previously.

The important point to be decided is whether or not the possibility of malignant change should influence one to treat peptic stricture by resection. The problem is somewhat similar to that which exists in the case of gastric ulcer with the same difficulty in the initial diagnosis and the same divergent views as to the incidence of malignant change. Brain (1967) referring to a series of 106 patients with peptic stricture said "we must be aware of the risk of secondary carcinomatous change appearing in chronic strictures especially in those associated with reflux oesophagitis. 8 patients not appearing for obvious reasons in this series provide an experience that profoundly affects one's thinking on oesophagitis and its management by conservative methods". We have taken a different view taking into account the uncertainty already discussed regarding malignant change in such lesions. It is essential in all cases to make every effort at the time of the initial diagnosis to exclude neoplasm but, when this has been done as far as possible, subsequent treatment should be as for a benign lesion.
TREATMENT

The methods of treatment used in this series were as follows:

1) Dilatation (either via the oesophagoscope or by self-bouginage) - 86 patients.

2) Repair of hiatal hernia - 36 patients.

3) Excision of the stricture - 28 patients. Some patients had resection following failed hernial repair.

Over the years, our views on treatment have not remained the same. In spite of this, the number of patients receiving the various forms of treatment still reflect our methods fairly accurately apart from the fact that, in the past 3 years, with alterations in technique (see Section 13), we have had more success with hernial repair and have tended to carry out this operation more often in the treatment of stricture.

Treatment by dilatation

Indications

We have not used dilatation in the treatment of children. Bill et al. (1963) claim success in children using the method of passing a bougie over a previously swallowed string or by oesophagoscopy and the passage of a filiform guided bougie. The type of lesion we have encountered in children has not seemed to us suitable for dilatation.

1) Age. The average age of adult patients treated by dilatation was 66 years. 37 patients were aged 70 or over. The average age of patients treated surgically was approximately 10 years less. In practice it appears that advanced age has been accepted as an indication for treatment by dilatation where this was effective.

2) Other diseases. A number of patients were considered unsuitable for surgical treatment because of concomitant disease, namely, mental deficiency (1), myocardial ischaemia (6), atheromatous
occlusion of the abdominal aorta (1), systemic hypertension (2), chronic bronchitis and asthma (9), pneumokoniosis (1), systemic sclerosis (2).

3) Good response to dilatation. In some patients, an excellent response to dilatation was accepted as an indication for the continuation of this form of treatment, even though the patient was not elderly (under 65 years of age) or unfit. Continued good progress is often not so much the result of dilatations as a reflection of the type of stricture present.

**Indications for abandonment of dilatation in favour of surgery**

1) Failure of dilatation to relieve dysphagia for an adequate length of time. An acceptable interval between dilatations is difficult to give in precise terms as it varies with circumstances. In an old or unfit patient, it may be best to persevere with dilatations over the years even though they are required at short intervals such as every 4 to 6 months. In a younger, fit patient such a frequency of dilatations is unacceptable and is an indication either for surgery or for self-bouginage.

2) In fit patients under the age of 65, it may be considered that their expectation of life is too long for repeated dilatation to be a suitable form of treatment unless, as is not at all uncommon, it is found that dilatation is required only at long intervals.

3) Certain types of stricture are unsuitable for dilatation. This applies to those associated with a penetrating ulcer and to those which appear radiologically as a long, rigid stenosis and where it is likely that there is total mucosal loss over a long segment of the oesophagus. Apart from such lesions, it is usually impossible to tell, except by trial and error, whether or not a stricture will be manageable by dilatation. Some which looked tight and firm and which, initially,
seemed unlikely to respond, did well after the first few dilatations.

The decision to continue dilatation or not is influenced by
the magnitude of the operation which it is anticipated would be
necessary if surgical treatment were to be undertaken. In some patients
it is obvious that nothing less than excision of the stricture will be
satisfactory. This is a major procedure which, in our view, is to be
avoided if possible in the elderly or unfit. Where it is considered
that a less exacting procedure such as hernial repair will be effec-
tive, a greater number of relatively old or unfit patients may be
accepted for surgery.

Results of Treatment by Dilatation

It is difficult to set out the results in exact figures as
the success or otherwise of the treatment must be judged on the opinion
of the patient. A degree of dysphagia acceptable to an elderly, bird-
like lady with a small appetite may be an insupportable hardship for
the gourmet or the glutton. The fact that dilatation was persevered
with in so many patients in this series may be merely a reflection of
the stoicism of north country folk or of the patience of the surgeon,
but we believe that it is an indication of the important place of
dilatation in management.

Except in patients where age or infirmity made surgical
treatment unduly hazardous, we have required dilatation to achieve
certain objectives before being accepted as the best form of treatment.
Firstly, and most important, the patient must feel satisfied with the
treatment. This usually implies that the dysphagia is adequately
relieved for a reasonable period of time. The patient must be free
from distressing pain or heartburn or such symptoms must be controllable
by medical means. Anaemia, if present, must respond to medical treat-
ment. There should be no complications such as the development of
penetrating ulcer, haemorrhage, or pneumonitis.

The longest period over which a patient was treated by dilatation was 16 years. The average duration of treatment up to the time of analysis was 5 years. The intervals between dilatations varied from patient to patient but tended to be fairly constant for any one patient. The figure for the average interval between dilatations was \( \frac{1}{2} \) years. This figure is misleading. In a majority of patients, dilatation was required every 6 to 9 months but there was a surprising number of patients in whom only 1 dilatation was required or where it was necessary only at long intervals, sometimes extending to several years. In 1 patient, for instance, only two dilatations were done with an interval of 10 years between them. Two patients who were admitted with food impaction and in whom a stricture was confirmed at oesophagoscopy and at subsequent x-ray required no further dilatations. They were warned to avoid swallowing large lumps of food. Certain patients who required an initial dilatation managed quite well afterwards with weight reduction and medical treatment.

In patients where dilatation is required frequently and where surgery is contra-indicated, self-bouginage is useful. It is best to admit the patient to hospital for instruction in how to pass the bougie. So that the bougie will pass through the stricture easily when the patient first swallows it, it is helpful to dilate the stricture through the oesophagoscope and to begin self-bouginage on the following day. The initial difficulties are more easily overcome if the patient is taught by someone he knows and in whom he has confidence. If the surgeon himself spends a short time each day instructing and encouraging the patient, there will be few instances of failure of this method due to lack of co-operation on the part of the patient. Careful follow-up is necessary as it may happen from time to time that, though the patient
is apparently passing his bougie satisfactorily, he is not in fact negotiating the stricture. Oesophagoscopy is then required after which self-bouginage can proceed.

Complications of Dilatation

In this series complications were few.

Perforation of the oesophagus. One patient died from instrumental perforation. He was a man of 34 who had some difficulty in swallowing since childhood and who had had many dilatations of his stricture over the years. On the last occasion, oesophagoscopy was followed after 2 hours by pain in the chest. A gastrografin swallow showed no escape of the medium from the oesophagus. A week later the patient developed an empyema on the left side. This was drained. Satisfactory progress was made until the patient died suddenly from a massive pulmonary embolism 3 weeks later.

This was the only instance of perforation in almost 500 dilatations. Dilatation is a safer procedure in peptic than in malignant stricture. The route through the stricture is more direct than is the case with an irregular, fungating malignant stricture and a false passage is less likely to be made. Also, in the case of minor mucosal tears some protection is afforded in many cases of peptic stricture by the intramural and peri-oesophageal fibrosis.

Haemorrhage. One patient bled on two occasions after dilatation and required blood transfusion.

Respiratory complications. One patient developed a dangerous degree of bronchospasm due to aspiration of acid during the induction of anaesthesia.

A note on the Management by Dilatation

Considerable symptomatic improvement may be achieved by medical measures such as weight reduction, advice as to appropriate
food, and treatment of anaemia. In addition to alkalis some of the newer preparations such as carbenebolone sodium may be used and have enhanced the efficacy of medical treatment.

The successful management of patients by intermittent dilatations through the oesophagoscope depends to a considerable extent on the arrangements under which the treatment is carried out. Preferably the dilatation in any one patient should be done by the same surgeon. The operator then knows the type of stricture he is dealing with, what difficulties are likely to be met with, and how they may be best overcome. Such knowledge adds to the safety and effectiveness of the procedure. Delegation of this work to inexperienced and continually changing junior staff produces inferior results. Rapport between the patient and the surgeon is of great importance if this form of management is to succeed and it cannot be built up if the patient finds himself being dealt with by a succession of doctors whom he does not know. It is equally important that the surgeon should if possible work with the same anaesthetist using a standard technique. A local arrangement must be made whereby the patient can readily get in touch with the hospital as soon as his dysphagia gets worse (Davidson, 1968).

Dilatation must then be carried out within one or two days. The treatment will not succeed if the patient, having had one dilatation, has to go through the "usual channels" in order to have the procedure repeated. This aspect of the management is emphasized as, in our view, some part of the dubious reputation which treatment by dilatation has acquired is due to neglect of such simple matters.

An objection to the treatment is that hospital beds are not available. We have carried out oesophagoscopy and dilatation over the past 20 years as an out-patient procedure. There is an obvious danger that the symptoms and signs of perforation may be delayed until the
patient returns home. This happened in the one patient who died 3 weeks after dilatation. Although, ideally, it might be best to admit the patients to hospital, this is impracticable and indeed most of those who have to attend regularly prefer to be dealt with as out-patients. It is considered that the procedure can be carried out with safety on an out-patient basis if certain precautions are taken.

1) The patient must be made thoroughly conversant with what is taking place and must know what untoward symptoms to look for.

2) The family doctor is informed that the procedure is being carried out.

3) It is ascertained beforehand that there will be someone at home when the patient returns and that he will not be alone on the first night. A patient who lives alone is kept in hospital overnight unless a relative or friend has agreed to spend the night with him.

4) The following types of patient are admitted to hospital overnight:

   a) Elderly patients (70 and over)
   b) Patients with a history of angina or coronary thrombosis.
   c) Patients with severe bronchitis or asthma.
   d) Diabetics
   e) Patients on steroid therapy
   f) Patients in poor general condition

5) No patient is allowed to leave the hospital without having had a drink of tea and without having been examined by a doctor. A doctor's signature is required by the nursing staff before the patient is allowed to leave.

6) Patients are taken home by ambulance.

**Comments on Treatment by Dilatation**

The treatment of a disease is determined to a large extent
by its natural history. As has been shown, peptic stricture is not necessarily a progressive lesion. In some patients the stricture is intractable from the outset as far as dilatation is concerned and surgery is required at an early stage. In most cases, however, the approach should be more deliberate. As the lesions tends to remain true to type in any one patient; a period of observations is often necessary before it is possible to decide what kind of stricture is present. As the natural history is very often that of a lesion causing dysphagia of varying degrees of severity over long periods of time, it is our view that no precipitate decisions regarding management should be taken. It is necessary to observe the response to a number of dilatations before it is possible to decide if a stricture is likely to be manageable over a period of years by minor measures or if surgery is indicated.

There are divergent views regarding the place of dilatation in the treatment. Palmer (1966) has used bouginage as the basis of his treatment and has commented on its value in elderly patients (Palmer, 1961). Benedict (1960) also believes that bouginage and medical treatment should be tried before surgery is considered. Hoover (1952), in a series of 72 patients with disabling oesophagitis, found that most patients responded well to dilatation. Olsen and Harrington (1948) had similar experience. The fact that 60 per cent of our patients were treated by dilatation indicates that our views are the same as those of the authors just mentioned.

Dilatation has been condemned on various counts. It is said by certain authors to be either ineffective, impracticable, or illogical. This has not been our experience. Barrett (1962) says that not only is dilatation of a stricture a destruction of nature's attempt to limit
upward spread of oesophagitis, but that it is illogical as the cause of mechanical obstruction is not simply scar tissue but is a combination of scar, circular muscle mass, congestion, and oedema. He found muscle hypertrophy at and not above the stricture in 17 resected specimens. He admits that the appearance of muscle hypertrophy may be an artifact. Paulson (1967) states that "although temporary relief may be obtained by dilatation, repeated endoscopic dilatation without correction of reflux removes the defensive barrier and increases the extent of oesophagitis".

In connection with the three symptoms, dysphagia, heartburn, and pain, our findings were as follows. Dysphagia is often satisfactorily relieved for adequate periods of time if one excludes the types of lesion already mentioned as being unsuitable for dilatation. The precise cause of heartburn and pain may be obscure. The view that they are merely a reflection of the amount of acid reflux is probably an over-simplification. Other factors such as spasm have to be taken into account. We found that increase in heartburn followed dilatation in only a small number of patients. A remarkable finding which is difficult to explain is that a number of patients complaining of pain were actually relieved of their pain after dilatation.

In attempting to answer the argument that dilatation destroys Nature's efforts to limit reflux, we are perhaps unfortunate in not having been initiated into Nature's precise intentions, nor do we fully understand what is meant by "Nature". This view is of necessity conjectural and we would put forward two equally conjectural theories as to why, in our experience, neither heartburn nor pain are necessarily made worse by dilatation. Sometimes a stricture is seen at endoscopy as a rigid orifice through which reflux is occurring. Because of the rigidity of the walls, the lumen remains permanently open. When the
striction is dilated and the rigid ring split, the walls become more mobile and can come into apposition. This would seem to explain, in theory, why reflux is not necessarily increased by dilatation in many patients. The manner in which pain may sometimes be relieved by vigorous dilatation suggests the analogy with the effect of stretching the anal sphincter in a case of fissure-in-ano. It is appreciated that there is no sphincter at the cardia comparable with the anal sphincter. However, Barrett (1962) suggests that when a peptic stricture is present there is an aggregation of muscle fibres around it. Coligher (1967) says that when an anal fissure is relatively superficial the sphincter usually undergoes tight spasm but when the fissure deepens and bares the sphincter fibres, this becomes even more pronounced. Eventually after several months the muscle may become fibrosed in its spastic condition so that a rather fibrotic, tightly contracted internal sphincter may result. Spasm of the anal sphincter has been freely indicted as a factor responsible for chronicity of the fissure. In our view a similar process occurs in the oesophagus and just as vigorous stretching of the anal sphincter will relieve pain and lead to healing of the fissure so dilatation of an ulcerated peptic stricture will often abolish pain for a time. Unfortunately, in the oesophagus stretching of a stricture cannot be carried out to the same extent as at the anus and its effects are temporary.

In summary, we believe that dilatation, if carried out correctly and in properly selected cases, is a satisfactory treatment in more than half of patients with peptic stricture.
One in every 4 patients in this series was treated by hernial repair.

**Indications.** If a peptic stricture is to be successfully treated by hernial repair the two absolute requirements are that it must be possible to reduce the hernia in such a way that it will not recur and that changes in the oesophageal wall are reversible.

As some elderly or unfit patients may be considered fit for hernial repair but not for the more extensive procedure of resection of the stricture, it is often important to know pre-operatively if the hernia is reducible. In the case of a high stricture with a gastric lined oesophagus, the associated hernia is, in our experience, reducible. In the case of a stricture midway between the arch of the aorta and the hiatus but with radiological appearances suggesting a segment of gastric lined oesophagus below it, the hernia is likely to be reducible. It is particularly in relation to strictures at the cardia with a fairly large sliding hernia that the radiological appearances and the oesophagoscopy measurements are of most importance in deciding whether or not the hernia will be reducible. As has been said before, it is sometimes impossible to tell if a hernia is reducible until the time of thoracotomy.

We have been pleasantly surprised on many occasions to find a reducible hernia when the radiological and endoscopic findings suggested otherwise. In earlier years, the hernia which was difficult to reduce tended, in our hands, to recur. As will be described in Section 13, changes in technique have reduced the incidence of recurrence and extended the indications for hernial repair.

Are the pathological changes reversible? This is always difficult to tell but we believe that, if the hernia can be satisfactorily
reduced, the changes in the oesophageal wall are reversible in most cases. The most promising situation is when, at oesophagoscopy, the stricture is found to be soft and inflamed and, at thoracotomy, the hernia is found to be easily reducible. However, some strictures which felt hard and irregular at oesophagoscopy responded well to reduction of the hernia. The following is an example.

The patient, a man of 60, had dysphagia for 9 months. In July 1967, there was an oedematous, inflamed stricture at 38 cms. In August 1967 the stricture had become hard and appeared to be ulcerated. In November 1967, it was very difficult to pass a bougie through it. The patient was losing weight. The hernia was repaired in February 1968. Over the past 3 years the patient has gained 2 stones in weight. At oesophagoscopy, there is now only a slight stenosis which looks smooth and feels soft.

Lesions which we think should be considered to be irreversible will be detailed later in this Section.

**Surgical Procedures Combined With Hernia Repair**

In a third of both adult patients and children, hernial repair was combined with vagotomy and either pyloromyotomy, pyloroplasty, or gastro-enterostomy. Such procedures were done when there was evidence of duodenal ulceration either past or present or when there was any indication of abnormality of the pyloric antrum. Sometimes the vagi were divided not so much in order to produce achlorhydria as to facilitate reduction of the hernia. The vagi, lying on an inflamed oesophagus, become thickened and rigid and form taut cords which hinder reduction of the hernia. We have insufficient evidence from this series to show if vagotomy is a useful adjunct to hernial repair in the treatment of peptic stricture, but tend to agree with Edwards et al. (1964) who are of the opinion that in the absence of
a duodenal ulcer there is little evidence to suggest that patients with reflux oesophagitis have greater than normal acid secretion and that there is no convincing proof that vagotomy with gastric drainage is necessary in addition to hernial repair.

Results of Hernial Repair in Adults.

The follow-up period ranged from 1 to 8 years. Successful symptomatic results with relief of dysphagia were achieved in 70 per cent of patients, 12 per cent were improved, and 18 per cent no better.

High strictures.

In 6 patients with strictures level with the aortic arch, hernial repair gave a good result in 5. On post-operative x-rays of the successful cases, there was disappearance or marked improvement in the stricture in 4 and reflux had been controlled in 3. In 1 patient no post-operative x-rays were available. One patient showed no clinical or radiological improvement. Post-operative x-rays showed persisting reflux.

Two patients are illustrated where a high stricture responded to hernial repair.

1. Figure 8 (page 21) shows the pre-operative x-rays of a woman of 54 with a tight, inflamed stricture at 20 cm. Figure 108 shows the post-operative film. At oesophagoscopy there is now only a smooth, soft narrowing. The patient has had no dysphagia for 3 years.

2. Figure 9 (page 21) shows the pre-operative stricture in a man of 50. The patient has had no dysphagia over the past 10 years. Figure 109 shows the post-operative barium swallow.

Strictures midway between the hiatus and the aortic arch.

There were 3 patients with strictures in this situation where it was considered that there was a segment of gastric lined oesophagus below the stenosis. All of them were eventually relieved of their dysphagia.
A stricture midway between the hiatus and the aortic arch. There was a long thickened segment in the oesophagus. After hernial repair only a web-like stenosis remained (Figure 111). This responded to 1 dilatation.
One was not immediately cured and his post-operative course is of interest.

This was a man of 45 who had dysphagia for a year. There was an inflamed stricture at 34 cms. X-rays showed a moderately high stricture (Figure 110). The hernia was repaired in 1963. There was a long, thickened segment in the oesophagus. The patient was considerably improved but had an episode of food impaction 1 year later. Radiologically, a web-like stricture was found (Figure 111). At oesophagoscopy, it was smooth and not inflamed. It split on dilatation. The patient has had no dysphagia in the past 6 years.

**Strictures at the cardia.**

24 patients with strictures at this site were treated by hernial repair. A good symptomatic result with relief of dysphagia was obtained in 14, 5 were improved, and 5 were no better. In spite of symptomatic improvement as far as dysphagia was concerned, post-operative x-rays showed persistence of a small hiatal hernia with reflux in 5 patients. In 1 patient where hernial repair was unsuccessful, a second repair 1 year later resulted in relief of the dysphagia for a period of 6 years until the patient’s death from uraemia.

The following patient is typical of successful cases in this group.

The patient was a man of 55 with increasing dysphagia for 3 years. There was a soft, inflamed stricture at 38 cms. (Figure 112). Following hernial repair, the patient had no further dysphagia. X-rays (Figure 113) show the absence of reflux and disappearance of the stricture. No abnormality is now seen at oesophagoscopy.

In 5 patients who were improved but still had slight dysphagia and heartburn, the post-operative x-rays showed persistence of reflux and some residual stenosis in all cases. In such patients, though some
Radiographs showing resolution of stricture after hernial repair. There is now no reflux. The patient no longer has dysphagia.

An ulcerated stricture causing severe pain.

The post-operative film shows (extreme top of picture) a smooth, residual narrowing.
symptoms persist, it is considered that the end result is as good as or possibly better than would have been achieved by resection. The following patient is typical of such cases.

The patient was a woman of 51 with severe dysphagia, asthma, anaemia, and an ulcerated stricture at 30 cms. (Figure 114). The lesion did not respond to dilatation. The hernia was repaired in 1967. The post-operative film (Figure 115) shows a residual smooth stricture of mild degree. At oesophagoscopy, there is now a smooth, non-inflamed narrowing at 33 cms. There is slight reflux. The patient has been much improved and has had only slight, occasional dysphagia in the past 4 years. Her asthma is no longer troublesome and she is no longer anaemic.

In 5 patients with a stricture at the cardia, repair of the hernia produced no improvement. As they illustrate errors in the selection of patients for hernial repair they are described individually.

**Case 1** : This patient, a man of 71, developed dysphagia soon after partial gastrectomy in 1962. In 1965 he had a firm, irregular stricture at the cardia. It responded poorly to dilatation and thoracotomy was carried out. There was no apparent hernia. The oesophagus felt hard and thickened immediately above the cardia. An attempt was made to make the cardia competent. This failed and the patient has required 14 dilatations in the subsequent 5 years.

This type of stricture has been described in Section 11. As its origin is, in our view, an acute burn of the oesophagus involving all coats, the pathological changes are probably irreversible in most cases.

**Cases 2 and 3** : Two patients already referred to on Page 90 and illustrated in Figures 81 - 84 (pages 93 and 94) had stenosis in association with an ulcer in a gastric lined oesophagus. The precise pathology was
not fully appreciated at the time. We would not now attempt to treat such lesions by hernial repair. Whereas an ulcer arising in squamous mucosa at a squamo-columnar mucosal junction, whether at the cardia or higher up in the oesophagus, may heal if reflux is prevented, an ulcer occurring within a gastric lined oesophagus does not appear to be influenced by the control of reflux.

Case 4: The patient was a man of 54 with a history of acid regurgitation for 8 years and increasing dysphagia for 3 years. There was a previous history of duodenal ulcer. At oesophagoscopy, there was a stricture at 35 cms. It felt firm. An ulcer was not visible at oesophagoscopy but was well shown radiologically (Figure 33, page 35). At operation, there was a small hiatal hernia. The oesophagus was difficult to mobilize because of peri-oesophagitis. It felt hard a short distance above the cardia. The hernia was repaired. An x-ray 2 weeks later suggested improvement with healing of the ulcer. 4 months later the patient had severe dysphagia and films showed a tight, smooth stricture (Figure 101, page 120). At subsequent resection of the lesion, there was a shallow, healing ulceration at the mucosal junction with fibrosis replacing much of the muscle coats. The histological appearances suggested that a deep ulcer had partially healed but had done so with fibrous contracture.

Case 5: The patient, a man of 70, had dysphagia for 4½ months. There was an inflamed stricture at 35 cms. No ulcer was visible at endoscopy but a biopsy showed granulation tissue resembling an ulcer base. A deep ulcer was shown on the barium swallow (Figure 116). At operation, there was a sliding hernia with a fairly long segment of firm, thickened oesophagus above the cardia. Repair of the hernia was difficult. Dysphagia persisted. Post-operative films showed progress resembling that seen in the previous case with disappearance of the ulcer.
Radiographs showing healing of a deep ulcer at the oesophago-gastric junction after attempted hernial repair leaving a tight, smooth stricture. Healing by squamous mucosa was confirmed at subsequent resection.
but the development of a tight, smooth stricture (Figure 117). At subsequent resection, there was no residual ulceration but dense fibrosis extending through all coats of the oesophageal wall.

It is worthy of note that in both cases where healing or partial healing of an oesophageal ulcer followed hernial repair, the healing process was by squamous and not by columnar epithelium.

In view of the results in such cases, we believe that if a confident diagnosis of a penetrating ulcer can be made, the stricture should be treated by resection and not by hernial repair.

**Results of hernial repair in infants and children.**

6 children were treated by hernial repair, or an operation to make the cardia competent. The results were bad. One patient died, 3 were no better, and only 2 were improved. In view of the bad results, the patients are described individually.

**Case 1:** This baby boy already referred to on Page 121 whose oesophageal ulcer is illustrated in Figure 102 (Page 122) was seen in 1953 at the age of 2 months. He was in poor condition, dehydrated, anaemic, and regurgitating blood-stained fluid. A gastrostomy was done. His condition continued to deteriorate. An attempt was then made to repair the hernia. The baby died 3 days later. At autopsy there was a large ulcer at the oesophago-gastric junction with a perforation at its centre. The hernia appeared to have been satisfactorily repaired.

**Case 2:** This mongol baby began to bring back blood-stained fluid at the age of 5 weeks. His haemoglobin was 53 per cent. Figures 118 - 120 show serial barium swallows. At oesophagoscopy there was free reflux. The baby failed to thrive. He remained anaemic and haematemesis persisted. Thoracotomy was done when he was 2 years old. There was no apparent hiatal hernia. The lower part of the oesophagus
Radiographs showing the progression of changes in the lower oesophagus in a mongol boy. There was no hiatal hernia. Progression of the lesion was not arrested by an attempt to make the cardia competent. The lesion was later resected and is shown in Figure 103 (page ).
was thickened and bulky. There were enlarged glands in the mediastinum. An attempt was made to render the cardia competent. The child improved only temporarily and had to be re-admitted after 2 months. The lower oesophagus was then replaced by a segment of colon. It was the site of multiple ulcers. There was squamous mucosa above and gastric mucosa below the ulcerated area (Figure 103, page 123).

**Case 3**: A child aged 3 years who had suffered from vomiting since he was 6 weeks old. From the age of 1 year, he had been unable to swallow solids. Figure 121 is a film in 1962 showing a long, rigid-looking stenosis and a hiatal hernia. There was frequent regurgitation of blood-stained fluid. The haemoglobin never rose above 58 per cent. At oesophagoscopy, there was an inflamed stricture which became tighter between 1961 and 1963. At thoracotomy in 1963, there was a sliding hiatal hernia. The oesophagus was greatly thickened and there was peri-oesophagitis with masses of inflammatory glands in the mediastinum. Hernial repair produced no improvement. The lesion was resected in 1966. At the site of stricture there was total mucosal loss and chronic inflammatory changes extending through all coats. There was a segment of gastric lined oesophagus between the stricture and the cardia. The upper margin of the ulcer was formed by squamous epithelium.

**Case 4**: Exactly the same features were found in a mentally retarded girl aged 8 years who had haematemesis when 16 days old and dysphagia thereafter. The only difference was that there was no apparent hernia. In her case, resection of the stricture became necessary 3 years after failure of an operation designed to render the cardia competent. (Figure 122)

**Case 5**: This boy, a deaf mute aged 8 years, had dysphagia since infancy. At the age of 8 he began to complain of pain in addition to dysphagia. Figures 123 and 124 show the progress of the lesion from
Film showing a long, rigid-locking stricture in a boy of 3. It did not respond to hernial repair and had to be resected.

Histologically, there was total mucosal loss and chronic inflammatory changes involving all coats.

Figure 121

Barium swallow in a mentally retarded girl of 8 years. At thoracotomy, there was no apparent hernia. An attempt to render the cardia competent was unsuccessful. The stricture was later resected.

Figure 122
Films showing the development of a large oesophageal ulcer between 1959 and 1964.

A film taken a year after hernial repair. There was both symptomatic and radiological improvement.
1959 to 1964. On the film taken in 1964 there is evidence of a large penetrating ulcer which was presumably causing the pain. At oesophagoscopy, there was an inflamed, ulcerated stricture. At thoracotomy, there was a hiatal hernia. For a short distance above the cardia, the oesophagus felt normal but from the level of the inferior pulmonary vein to the aortic arch it was much thickened and there was peri-oesophagitis. The hernia was repaired and vagotomy and pyloroplasty carried out. There was considerable improvement. Nine months later x-rays showed the oesophagus looking much more normal (Figure 125) although there was still some reflux. Six years after the operation, dysphagia is minimal. The boy has developed normally.

Although this case turned out well, we would not now attempt to treat a similar lesion by hernial repair.

Case 6: This child had a rather tight, smooth stricture a short distance above the cardia. A hiatal hernia was repaired when she was 2 years old. Three years later a tiddleywink stuck in the oesophagus. At oesophagoscopy for its removal, a slight stenosis was seen. The child eats normal food and has developed well.

**TREATMENT OF PEPTIC STRICURE BY RESECTION**

The stricture was resected in 28 patients (19 per cent). 5 of the patients were children, 1 a mentally retarded youth of 17, and the remainder were adults with an average age of 59 years.

**Indications.**

The indications for resection have evolved with experience. In earlier years, strictures were sometimes treated by resection without an adequate trial of lesser measures. The tendency in later years has been to reserve resection for strictures not responding to or unsuitable for dilatation or hernial repair. Resection has been
carried out in the following circumstances:

1. Where treatment by dilatation has failed.
2. Where, at thoracotomy, the hernia has been found to be irreducible.
3. Where hernial repair has failed to influence the stricture.
4. Where it is considered that the type of stricture present will not respond either to dilatation or hernial repair as, for instance, some post-operative strictures, those associated with a penetrating ulcer, and those where there is extensive mucosal loss and gross thickening of the oesophagus.

Type of operation and Results.

Until 1957, patients who did not respond to dilatation were treated by resection of the stricture. The operation commonly performed at that time was resection of the lower oesophagus, total gastrectomy, and oesophago-jejunostomy in the manner of Roux. Seven operations of this type were done; 5 prior to 1957. Two patients died. The others, although relieved of their dysphagia, were not altogether satisfactory results. One man of 36, of no fixed abode, was lost sight of after 2 years. One man, aged 70, died 3 years after operation of a stroke. A man of 65, though having no further dysphagia, remained underweight and suffered from post-thoracotomy wound pain. He died 10 years later of a bronchial carcinoma. A woman of 56 is alive 14 years after surgery but suffers from anaemia and osteoporosis. In recent years only 1 patient has had this operation. He was a man of 87 with a penetrating ulcer at the stricture. During construction of the jejunal loop it was observed that there was extensive atheroma in the mesenteric arteries and it was considered safer not to divide the loop for anastomosis to the stomach. The patient has been followed up for 1 year and is doing well.

Since 1957, the operation usually performed has been resection of the oesophagus from above the stricture and interposition of jejunum
between the oesophagus and stomach. There were 11 such operations with no deaths. The results have been satisfactory from the point of view of the patients' well being, nutrition, and haematological state in all but 1 case. This patient, a psychotic woman of 49, who, before operation had made several attempts at suicide and who suffers from chronic bronchitis, has lost weight since the operation.

In children, a colonic rather than a jejunal transplant has been used. Five children have been dealt with in this way. One mentally deficient girl of 12 died.

Oesophago-gastrostomy was done in 5 patients. Two patients died. Although there is no doubt that some patients do well after this procedure it is not a logical one and 1 patient developed a post-operative stricture due to reflux. At the time of his death 10 years after operation he was found to have a stricture and a penetrating ulcer at the site of the oesophago-gastric anastomosis. The other 2 patients have done well and have complained of very little heartburn over periods of 3 and 6 years. Where a lesion was such that its resection was essential but the patient was a poor risk, we have been tempted to use oesophago-gastrostomy rather than jejunal or colonic replacement. This accounts for the high death rate with this procedure. The wisdom of such a choice is doubtful.

Collis's gastoplasty has not been used in this series nor has reliance been placed on operations such as vagotomy and gastric drainage.

The usual objection to treatment of peptic stricture by resection and, in particular, to the use of jejunal or colonic transplants is that the mortality is too high for the treatment of a non-malignant condition. The mortality from all resections was 17.8 per cent which is certainly very high but it has to be taken into account
that the fatal cases were all poor surgical risks.

The following is an analysis of the patients who died.

1. Oesophago-jejunostomy and total gastrectomy (2 deaths in 7 patients).

One fatality was a woman of 64 who had oesophageal stenosis due to an ulcer within a gastric lined oesophagus. After hernial repair, she had a pulmonary embolism. The lesion was later resected and the patient died from a massive pulmonary embolism.

The second patient was a man of 54 with a penetrating ulcer and stricture also within a gastric lined oesophagus. He suffered from hypertension due to polycystic disease of the kidneys. He had previously been in uremia following a haematemesis. Death occurred in uremic coma after resection of the lower part of the oesophagus.

2. Interposition of a colonic transplant (1 death in 5 patients).

The fatal case was a girl of 12, mentally handicapped due to phenylketonuria. There was a long oesophageal stenosis with total mucosal loss. Death occurred due to gangrene of the colonic transplant.

3. Oesophago-gastrostomy (2 deaths in 5 patients).

One patient was a man of 76 who suffered from severe chronic bronchitis. He had an ulcerated stricture causing a great deal of pain. The patient died of heart failure post-operatively.

The other patient, a man of 70, had an ulcerated stricture following vagotomy and gastro-enterostomy for duodenal ulcer. His condition was poor. At the time of oesophago-gastrostomy he was found to have a subphrenic abscess from the previous operation. He died as a result of an anastomotic leak.

As has been described, it is our policy to reserve resection for patients in whom lesser measures have failed or appeared to be unsuitable.
We accept that, in the seventh decade, there is a tendency for progression of the lesion demanding radical surgery. The result is that the group of patients where resection has been done comprises the worst cases both from the point of view of the local lesion and the general condition of the patient. In 2 patients, death was directly attributable to breakdown of the anastomosis but, in both, there were obvious difficulties accounting to some extent for the surgical failure. The other 3 patients were poor risks because of previous pulmonary embolism, previous uraemia, and severe chronic bronchitis in old age respectively.

In view of the type of patient in whom resection was performed, it is not considered that the mortality rate should be interpreted as an argument against treatment by resection.
SECTION 13
CIRCUMFERENTIAL OESOPHAGEAL MYOTOMY

An account of circumferential myotomy is included in the thesis as it involves a little of description of surgical technique, and its interest lies in the physiological and anatomical features of the oesophageal muscle.

Many properly selected peptic strictures can be successfully treated by hernial repair which is a relatively safe procedure and is associated with fewer complications than those in which the gut is opened either for gastroplasty, oesophago-gastrostomy, or jejunal or colon transplant. Unfortunately, shortening of the oesophagus may render the hernia irreducible or give rise to recurrence. d'Abreu (1963) makes the following statement with which we entirely agree:

"Unfortunately, and I think this is becoming more apparent, adequate hernia operation is often impossible once stricture formation has developed. At thoracotomy in some patients the oesophagus can be returned to its normal site with its true length; if, however, the shortening of the oesophagus is material, an ominous sign for the surgeon is the cone-like appearance of the diaphragm round the elevated cardio-oesophageal junction. I now believe that if the oesophagus has to be dragged down below the diaphragm under tension the results will be disappointing". The operation to be described is aimed at allowing easier reduction of the hernia in a greater number of patients and at diminishing the recurrence rate.

The procedure is technically simple and involves division of the muscle layers of the oesophagus in a circumferential manner above the level of the stricture.

In the years following 1957, as a result of work on local resection of small carcinomas of the oesophagus with end-to-end oesophageal anastomosis (Davidson, 1967), we became interested in the
action of the longitudinal muscle of the oesophagus. It was found possible to excise up to 5 cms. of the oesophagus and still carry out end-to-end anastomosis. One difficulty met with was the distraction of the cut ends of oesophagus as a result of contraction of the longitudinal muscle. Indeed, even if the oesophagus is merely transected without removal of any part of it, the cut ends are drawn apart and, if left in this position for a short time, may be quite difficult to bring together again. This distraction of the cut ends was largely overcome by infiltrating the muscle coats along most of the oesophagus with local anaesthetic in order to throw the longitudinal muscle out of action before the oesophagus was divided. It was also observed that, if the muscle coats alone are divided circumferentially, their cut ends move apart for a distance of about 1.5 cms. leaving a tube of bare mucosa between them.

The manner in which the longitudinal muscle can powerfully distract the cut ends of the oesophagus and the way in which the muscle coats retract when divided leaving a mucosal tube between them suggested that their division might render some hiatal hernias reducible which would not otherwise be so. Initially, therefore, this procedure was used in an effort to gain a little extra length in the oesophagus sufficient to allow reduction of a hiatal hernia in the presence of a stricture with some shortening. As will be described, the initial idea has been developed along additional lines.

Anatomy

The longitudinal muscle of the oesophagus is highly developed and, unlike the condition usually found in the digestive tract, it is as stout as, or in places stouter than, the circular layer. Figure 126 are photographs taken at the same magnification showing that the oesophageal muscle is much better developed than that of the stomach,
Sections at the same magnification of - 1, oesophagus; 2, stomach; 3, jejunum; 4, ileum; and 5, colon. They show that the oesophageal muscle layers are much better developed than at the other sites.
jejum, ileum, or colon. It is assumed that the power of the muscle is related to its bulk and is, therefore, correspondingly greater in the oesophagus.

That the muscle of the pharynx and oesophagus may undergo violent contraction to the extent of spontaneous rupture is suggested by two of the patients. The first was a woman of 50 who after swallowing a mouthful of pudding was seized with intense pain on the left side of her neck. At operation she was found to have a clean, longitudinal split in the left postero-lateral wall of the pharynx. The lesion had all the features of a spontaneous rupture of the pharynx (Davidson, 1964). The second patient was a woman of 65 who, without previous dysphagia, got a piece of soft meat impacted in her oesophagus. She made violent swallowing efforts and developed sudden pain in her chest. A gastrografin swallow showed rupture of the oesophagus with the bolus still impacted (Figure 127). It was apparent that the oesophageal rupture above the impacted bolus could not have been due to the force of a vomit. At oesophagoscopy the bolus was removed. It was composed entirely of soft material. The right pleural cavity was drained and the patient recovered. Subsequent barium swallows were normal.

These cases are described in order to emphasize the force which may be generated from contraction of pharyngeal or oesophageal muscle.

The oesophageal mucosa is relatively inextensile. It is only loosely connected with the circular muscle coat by the submucosa. It is thrown into longitudinal folds when the oesophagus is empty and contracted. As it is without transverse folds it can be stretched only a short distance in a longitudinal direction. However, in the presence of a hiatal hernia where the cardia has moved upwards, there may be some slack in the oesophageal mucosa so that the cardia can be
Spontaneous rupture of the oesophagus above an impacted bolus of soft meat.
moved downwards for a greater distance than is possible in normal circumstances. It has been suggested (Stiennon, 1968) that transverse oesophageal mucosal folds may appear when the factors maintaining oesophageal length in spite of longitudinal muscle contractions are negated. Such redundancy of the oesophagus is present with hiatal hernia. It is also said to occur in kyphosis.

In mobilizing the oesophagus, one gains the impression that its upper half is fixed to a greater extent than its lower half. The upper part of the oesophagus is applied to the posterior wall of the trachea and there is some fixation in the region of the aortic arch and to the left main bronchus where the broncho-oesophageal muscle is described. There is a greater freedom of movement of the oesophagus in the region of the hiatus. Upward movement occurs at this level on swallowing, eructation, and vomiting (Johnson, 1966). In normal circumstances, the anatomy is such that although the oesophago-gastric junction may move upwards, it subsequently resumes its normal position. In the presence of a sliding hiatal hernia, the mechanism causing the cardia to revert to its normal position no longer operates. It is assumed that, with this loss of fixation of the lower end of the oesophagus, the longitudinal muscle may act in a partially unopposed manner and exert an upward pull tending to accentuate the upward slide of the oesophago-gastric junction.

**Shortening of the Oesophagus**

While it is appreciated that some of the changes in the oesophageal wall are in the submucosa, there is no doubt that inflammatory changes occur in the muscle layers which may become thickened and rigid as a result of inflammation, oedema, and sometimes fibrosis. Such thickened muscle when it is in a situation of shortening is one factor in causing difficulty in reduction of a hernia.
A second type of shortening of the oesophagus receives little attention but we believe that it exists. When a large sliding hernia has been present for a long time, even though there is only superficial oesophagitis, the oesophageal muscle may become adapted to its shortened state much as a skeletal muscle will do when a neighbouring joint has been fixed for a long time in a flexed position. Thus, on occasion, it may be difficult to bring the oesophageal-gastric junction back to its correct anatomical situation when it has been lying well above the hiatus for a number of years.

Factors causing difficulty in reduction of a hernia and leading to its recurrence

1) If a hernia can be reduced only by exerting considerable downward traction on the oesophagus there is, from the outset, an upward pull which may render the repair ineffective. If this upward pull results in coning of the diaphragm at the hiatus, the cardia will continue to be incompetent.

2) In the absence of normal fixation of the lower end of the oesophagus, the longitudinal muscle may, by exerting a predominantly upward pull, contribute towards hernial recurrence. It is of interest that hiatal hernia occurs in some cases of diffuse muscle spasm in the oesophagus. Cremer (1962), and Craddock et al. (1966).

3) If there is inflammation and early fibrosis in the muscle layers, contraction of this fibrous tissue may continue post-operatively causing upward traction on the oesophageal-gastric junction just as may happen as a result of the mural fibrosis following corrosive burns. Imre and Wooer (1969).

Rationale of Circumferential Myotomy

Circumferential myotomy is an attempt to counteract the three forces just mentioned.

1) The immediate upward pull which comes into play when a
hernia is reduced with difficulty.

This force would be prevented if the oesophagus could be elongated. Such elongation was the initial aim when we first performed myotomy. The amount of lengthening of the oesophagus which can be achieved is limited. As has been pointed out, the main obstacle is the relatively inextensible mucosa and the absence of transverse mucosal folds. In the presence of a hernia, however, some elongation may be possible. In an attempt to measure it we have taken the distance of the oesophago-gastric junction under mild downward traction and the lower margin of the aortic arch before and after myotomy. Elongation of the oesophagus of about 2 - 2.5 cms. may be achieved. This seems a small distance but may, in border-line cases, make all the difference between success and failure of hernial repair and of the production of competence at the cardia.

2) The upward pull of the longitudinal muscle.

Code et al. (1962) in a paper on manometric studies in the oesophagus describe movements of the oesophagus at the hiatus on deglutition. They suggest that two actions are involved: first the raising of the oesophagus by elevation of the cricoid cartilage associated with the voluntary part of the act of swallowing and secondly, shortening of the oesophagus by contraction of its longitudinal muscle fibres. Nauta (1955) says that the peristaltic wave forces food through the oesophagus is characterised by an important shortening and slight narrowing of the outer wall of the oesophagus. It is with this upward tug of the longitudinal muscle that we are concerned here.

After the longitudinal muscle has been divided, it is assumed that its ability to exert an upward pull on the oesophago-gastric junction is impaired and that muscle contraction above and below the myotomy will lead, at least to some extent, to distraction of the cut
ends of muscle rather than a direct upward pull on the region of the cardia. (Figure 128)

3) Similarly it is postulated that, if further contraction of fibrous tissue occurs within the muscle layers, shortening of the muscle can occur in an upward and downward direction away from the line of section without causing diminution in the length of the oesophagus.

For any or all of the aims of myotomy to be achieved it is essential that, at the time of hernial repair, the gastro-oesophageal junction is firmly fixed below the level of the hiatus. While Allison's method of hernial repair (1951) is an admirable approximation to the restoration of the normal anatomy, it is possibly inadequate in the presence of shortening of the oesophagus. This repair depends to some extent on suture of the phreno-oesophageal ligament to the under surface of the diaphragm. It is doubtful if the tissues of the diaphragm in the vicinity of the hiatus, stretched and weakened as they are in the presence of a hernia, can afford the fixation of the oesophago-gastric junction which is necessary if there is some shortening of the oesophagus. For this reason the method of hernial repair used with myotomy has been that described by Hill (1967), where the technique consists of anchoring the gastro-oesophageal junction to the median arcuate ligament and pre-aortic fascia. The median arcuate ligament, though not always easy to display and, on occasion, less well developed than usual, is an excellent point of fixation in most cases.

**Technique**

The operation is performed through a left thoracotomy or thoraco-abdominal incision.

Dissection is carried out to determine as far as possible the nature and extent of the pathological changes in the oesophagus. A large stomach tube is passed. The point at which it is held up shows
Upward pull of longitudinal muscle

The effect of contraction of the longitudinal muscle after Myotomy

Figure 145
the site of the stricture. The oesophagus is usually mobilized as far up as the aortic arch. A decision is then made as to whether or not reduction of the hernia is going to be possible. In some cases it is obvious that reduction of the hernia is impossible. In others the discovery of a penetrating ulcer may contra-indicate repair. This leaves a number of borderline cases where reduction seems possible but only by pulling the oesophago-gastric junction downwards. It is in such patients that myotomy may be useful.

If it is decided that reduction of the hernia may be possible, attention if first directed to the vagi. If they are embedded in a swollen oesophageal wall and are themselves thickened and rigid, they are divided. If they are relatively normal, they are dissected off the oesophageal wall and held away from it by threads passed round them.

Myotomy is then carried out preserving the vagi. The myotomy is done a short distance above the stricture. This site has been chosen from the point of view of safety. Division of the muscle at or near the stricture might result in a breach of the mucosa through the base of an unsuspected ulcer. A second reason for doing the myotomy some way above the stricture is that there is less inflammatory change in the oesophageal wall at this point and more movement between the mucosa and the muscle layers as possible. Above the stricture the myotomy is easy and safe.

To begin with, an attempt was made to confine the myotomy to the longitudinal muscle. It was feared that division of both the longitudinal and circular muscle layers might lead to a dangerous weakness of the oesophageal wall. It is, however, not easy to limit the myotomy to division of the longitudinal muscle and, with further experience, it has been found best to divide both muscle layers.

On two occasions myotomy has been done at two levels, one
just below the aortic arch and one just above the stricture. Normally only one myotomy above the stricture is done.

Myotomy has also been carried out below a high stricture with the lower oesophagus lined by columnar epithelium. In this situation, the myotomy is through normal muscle and is designed not so much to achieve elongation of the oesophagus as to impair the action of the longitudinal muscle.

After the myotomy has been completed, the oesophago-gastric junction is again brought down to see if it will reach a satisfactory level without undue tension on the oesophagus. If it does so, the hernia is repaired by Hill's method.

To help towards the identification of landmarks on post-operative x-rays, markers (Gushing clips) have been placed in a number of patients operated on in the past 18 months. A clip has been placed on each cut edge of muscle, on the margin of the hiatus, and on the oesophago-gastric junction.

Figure 129 illustrates the operative procedure.
Figure 129

Cut margins of muscle

Thread holding off Vagus

Tube of mucosa

Site of stricture

Diaphragm

CIRCUMFERENTIAL MYOTOMY
Operative Results

Circumferential oesophageal myotomy has been done in 25 patients during a period of 3½ years. The longest follow-up is 3½ years and the shortest 6 months.

The first patient was operated on in October, 1967. She was a woman of 55 who had discomfort opposite the xiphoid on swallowing. X-rays showed what appeared to be an ulcerated narrowing of the oesophagus above a hiatal hernia. At oesophagoscopy, there was an inflamed, oedematous stricture at 32 cms. It dilated easily. At thoracotomy, there was a thickened segment of oesophagus some distance above the oesophago-gastric junction. There were enlarged glands in the adjacent mediastinum. A circumferential myotomy confined to the longitudinal muscle was done above the thickened area and the hernia was repaired by Allison's method. The patient was followed up for 2 years during which time she was well apart from occasional belching.

In the earlier patients, post-operative investigation and follow-up was somewhat inadequate. Post-operative oesophagoscopy was sometimes omitted. In patients operated on in the past 18 months careful and repeated post-operative x-rays have been taken, markers have been placed at the time of operation in a number of patients, and post-operative oesophagoscopy has been done. During the past 2½ years, Hill's method of repair has been used in preference to that of Allison.

The types of lesion dealt with were strictures at or near the cardia (13), high strictures (3), strictures midway between the hiatus and the aortic arch (1), penetrating ulcer (3), post-operative stricture (1), large sliding hernia without stricture where reduction was difficult (3), and one patient without stricture but with severe aerophagy.

1. Strictures at or near the cardia.

In all the patients in this group there was a firm, thickened
area to be felt in the oesophagus, commonly a short distance above the
oesophago-gastric junction. There was evidence of peri-oesophagitis
with enlarged mediastinal glands. There was relief of dysphagia and
a good symptomatic result in all patients. Competence of the cardia
was judged by the usual radiological methods and also by distending the
stomach with gas. In 2 of the 13 patients some reflux was produced.
Radiologically, it was not possible using liquid barium to detect a
residual stricture. We have been impressed by the efficiency of
Hill's method of repair in producing competence at the cardia.

At post-operative oesophagoscopy, there was either no evidence
of a residual stricture or there was a smooth, non-inflamed narrowing
insufficient to cause dysphagia. No post-operative dilatations of
stricture were required.

This group is illustrated by the following patients:

a) The patient was a woman of 57 who had a previous history
of heartburn and acid regurgitation and of dysphagia for 14 months.
Figure 130 shows the barium swallow. At oesophagoscopy, there was a
very inflamed stricture at 34 cms. The mucosa above it was inflamed
and bled readily when touched. A biopsy showed mucosal ulceration.
At operation there was a thickened segment of oesophagus 2.5 cms. above
the oesophago-gastric junction. Figures 131 and 132 show the post-
operative appearances. The cardia is competent. There is no residual
stricture to be seen radiologically or on oesophagoscopy. The patient
has been free from symptoms for 15 months.

b) This patient, a woman of 54, had dysphagia for 3 years.
She has episodes of food impaction. Haematemesis had occurred 4
months previously. Figure 25 (page 31) shows the pre-operative barium
swallow. At oesophagoscopy, there was a soft, inflamed stricture at
35 cms. At thoracotomy, there was a thickened, firm area in the
The pre-operative and two post-operative radiographs of a woman of 57 who had an inflamed stricture at 34 cms.

The cardia is now competent. There is no residual stricture to be seen at oesophagoscopy.

The patient is free from symptoms.
oesophagus 3 cm. above the oesophago-gastric junction and there was peri-oesophagitis. Figures 133 and 134 show the post-operative appearances. There is no stricture and the cardia is competent. The patient has been free from symptoms for over 2 years.

c) This man of 55 had a previous history of indigestion and of dysphagia for a year. Figure 135 shows the barium swallow. There was an inflamed stricture at 39 cms. A biopsy showed a fragment of acutely inflamed granulation tissue. The oesophagus was difficult to mobilize and there was a thickened area 3 cms. above the oesophago-gastric junction. Figure 136 shows the post-operative barium swallow. The cardia is competent. The patient has remained free from symptoms for 3 years.

d) A similar case is illustrated in Figures 112 and 113 (page 152).

2. High strictures.

Three patients with high strictures were operated on with good results. The following are the details of one of them:

This was a man of 48 who had heartburn for some years and then developed dysphagia. It would take him an hour to take one course of a meal. Figure 137 shows the barium swallow. There appears to be an ulcer at the site of stricture. At oesophagoscopy, there was an irregular stricture at 28 cms. It was not possible to tell from the endoscopic appearances whether it was benign or malignant. A biopsy showed a fragment of mucosa from the squamo-columnar junction deep to which there was chronic inflammatory infiltrate. The hernia was repaired, together with vagotomy and pyloromyotomy. A myotomy was done below the stricture. Figures 138 and 139 show the post-operative barium swallows. The patient has had no dysphagia or heartburn for 9 months. The oesophagoscoptic appearances are normal.
The illustrations below are two post-operative films of a woman of 54 who had a soft, inflamed stricture at 35 cms. (the pre-operative barium swallow is shown in Figure 25 (page 31). The cardia is now competent. Figure 133 shows a good angle between the oesophagus and the fundus. There is no radiological or oesophagoscopy evidence of residual stricture.
The pre and post-operative barium swallows of a man of 55 who had a stricture at 39 cms. The oesophagus was thickened and difficult to mobilize.

The patient remains free from symptoms after 3 years.
Figure 136 shows a high ulcerated stricture in a man of 48.

Figure 137 is a barium swallow 2 weeks after operation. Some healing appears to have occurred already.

Figure 138 is a barium swallow 3 months later showing only slight residual narrowing. The cardia is competent. At oesophagoscopy no stricture or ulcer is now visible.
There is probably no good reason for doing a myotomy in the case of a high stricture in an adult as the hernia is usually not difficult to reduce.

3. **Stricture midway between the hiatus and the aortic arch on x-ray.**

The patient, a woman of 35, had a history of dysphagia for 11 months following heartburn over a period of 8 years. She had lost 1\(\frac{1}{2}\) stones in weight. Figure 140 shows the barium swallow. There was a soft, very inflamed stricture at 31 cms. (patient's height 4 ft. 11 ins). There was free reflux through the stricture and intense oesophagitis above it. A biopsy from the stricture showed granulation tissue. At thoracotomy, there was a sliding hiatal hernia. A stomach tube was passed. It was held up at the oesophago-gastric junction and, in spite of the radiographic appearances, the stricture could be brought down near to the hiatus. There was chronic mediastinitis with a mass of enlarged glands beside the lower oesophagus. The vagi were isolated and preserved. A myotomy was done midway between the inferior pulmonary vein and the aortic arch and the hernia repaired by Hill's method.

Figure 141 shows the post-operative barium swallow. No stricture is demonstrable. No reflux could be produced.

At post-operative oesophagoscopy a month after the operation, a slight, smooth narrowing was seen. This area seemed mobile showing movement of its walls with respiration. There was no apparent reflux or oesophagitis.

4. **Penetrating ulcer.**

Three patients had a penetrating ulcer at the site of stricture. Two of them have already been described on pages 33 and 154 (Figures 33 and 116). Both were surgical failures. One patient with an ulcer did well. She was a woman of 66 known to have had a hiatal hernia for 6 years and who began to complain of severe pain deep to the
Pre-operative and post-operative barium swallows of a woman of 35 with an ulcerated stricture at 31 cms.

At post-operative oesophagoscopy there is only a slight, smooth, residual narrowing insufficient to cause dysphagia.

Markers have been placed on the cut margins of the oesophageal muscle, the hiatal margin, and the oesophago-gastric junction. The post-operative films showed that the cardia was competent. The lowest marker which is on the oesophago-gastric junction is lying well below the hiatus.

Figure 140

Figure 141
xiphoid. Her life had become a misery. Figures 142 and 143 show only slight narrowing at the cardia but suggest the presence of an ulcer. A large ulcer with a sloughing base on the posterior aspect of the oesophagus was seen at endoscopy. A biopsy from the region of the ulcer contained gastric epithelium. At thoracotomy, there was a sliding hiatal hernia. At and just above the oesophago-gastric junction the oesophagus was hard and grossly thickened. There was a palpable ulcer with a dimple on the external surface of the oesophagus at the ulcer base. There were enlarged para-oesophageal glands. Thickening of the oesophagus extended upwards to a point a little above the level of the inferior pulmonary vein. The oesophagus was mobilized up to the aortic arch. The hernia could be reduced only by dragging on the oesophagus. The vagi were divided. Myotomy was done and the hernia repaired by Hill's method.

The patient has remained well for a year. There is now no ulcer to be seen on x-ray (Figures 144 and 145) or on oesophagoscopy. A small amount of reflux can, however, be produced by the radiologist.

Although this patient has done well so far, it is considered that a penetrating ulcer is a contra-indication to treatment by hernial repair. Even though healing of the ulcer may be achieved, there is too great a likelihood of a residual fibrous stricture.

5. Post-operative stricture.

One such patient was treated by hernial repair and a double myotomy with success. There is still a slight stenosis to be seen at oesophagoscopy. It is quite smooth and not inflamed and has been insufficient to cause dysphagia in the past 3 years.

In spite of this one successful result, it is considered that post-operative and post-recumbency strictures are usually unsuitable for treatment by hernial repair as the pathological changes involve all
Two pre-operative and two post-operative films of a woman of 66 with a stricture at 36 cms. with a deep ulcer at the oesophago-gastric junction. The ulcer is no longer visible on oesophagoscopy.
coats of the oesophageal wall and are likely to be irreversible.

6. Three patients had myotomy and hernial repair who had no stricture. They had large sliding hernias and, in spite of the fact that there was apparently only a mild degree of superficial oesophagitis, it was difficult to bring the oesophago-gastric junction below the hiatus. This is the type of shortening referred to on page 172.

The following patient is typical of this group.

He was a man of 63 who had burning acid regurgitation after meals. He suffered from heartburn at night. He occasionally lost his voice. Figure 146 shows the barium swallow. The cardia was at 38 cms. It appeared to be incompetent and the mucosa above it was inflamed and had superficial erosions on it.

There was a sliding hiatal hernia which was difficult to reduce although there was no real evidence of inflammatory changes in the deeper layers of the oesophageal wall. A myotomy appeared to render the hernia easier to reduce. The vagi were preserved. As the patient was very fat, the hernia was repaired by Allison's method. Access to the median arcuate ligament was difficult.

Figure 147 shows the post-operative appearances. There is no demonstrable reflux. The patient has lost his previous symptoms but has troublesome post-thoracotomy wound pain.

7. One patient, a woman of 60, had no stricture but had free reflux and severe aerophagy. On screening she was seen to be making violent swallowing movements with upward movements at the cardia. The hernia was repaired and myotomy done in an effort to prevent the repeated upward movement of the oesophago-gastric junction. The result of operation was good but no conclusions can be drawn.

Post-myotomy Radiographic Appearances

After circumferential myotomy the characteristic appearance
Pre-operative and post-operative films of a man of 63 who had a large sliding hernia but no stricture. The hernia was difficult to reduce and a myotomy was done.
on barium swallow is that of a local distension of the oesophagus having the appearance of what plumbers call a "wiped joint". On screening it is apparent that this local distension is not present all the time but only in certain phases of swallowing. This appearance is shown in Figures, 131, 132, 133, 134, 136.

It is of interest that while no local bulging is seen on barium swallow after Heller's operation, it was observed after circumferential myotomy above a peptic stricture. It is not seen, however, in the patient in whom myotomy was done below the stricture (Figures 138 and 139, page 185).

During a limited follow-up, there has been no evidence that the local distension of the oesophagus increases with time.

Below the bulge at the myotomy, the oesophagus often appears somewhat narrower than above the myotomy and has a peculiar cylindrical appearance (Figures 132 and 134, pages 181 and 183). Whether or not vagotomy is carried out at the same time as myotomy does not seem to affect the configuration of the oesophagus below the myotomy.

On screening, there has been no evidence that the myotomy interferes with the passage of food down the oesophagus. No patient in whom the stricture was relieved was conscious of dysphagia. Trans-thoracic vagotomy carried out at the same time as vagotomy does not appear to cause dysphagia.

In a number of patients in whom markers were applied to the cut ends of oesophageal muscle, to the margin of the hiatus, and to the oesophago-gastric junction, some interesting radiological appearances have been seen. It has been possible to study the position and movement of the markers in two patients by cineradiography.

The markers on the cut ends of muscle are commonly about 2 to 3 cms. apart (Figure 148). In one patient, however, the markers
A radiograph showing 3 markers. The upper 2 are on the cut ends of muscle at the myotomy. The lower marker is on the hiatus margin. The myotomy markers are, in this case, 3 cms. apart.
remained closer together (Figure 141, page 167.). The local distension of the oesophagus during some phases of swallowing is seen to occur between the cut ends of muscle. It has been difficult to get comparable measurements but the impression gained so far is that there is a tendency for the myotomy markers to move a short distance further apart as judged by comparison of films taken on the first post-operative day and one month later. In some cases it can be seen that while the lower myotomy marker remains in a constant relationship to the marker on the hiatal margin, the myotomy markers move away from one another in phases of swallowing. This is shown in Figure 149 where the distance between the myotomy markers varies from 2 to 3 cms. On cineradiography in two patients, the myotomy markers were seen in one patient to move farther apart during swallowing. This separation of the markers seems to be due mainly to upward movement of the upper marker.

From these findings it would seem that the myotomy is probably fulfilling its intended purpose and that, after myotomy, contraction of the longitudinal muscle produces some distraction of the cut ends rather than a direct upward pull on the oesophago-gastric junction.

The relative positions of the markers on the hiatal margin and the oesophago-gastric junction as seen on radiographs show the effectiveness of Hill's repair in bringing the cardia below the hiatus and fixing it there. The cineradiographs did not show the lower markers clearly and were not helpful in confirming the fixation of the oesophago-gastric junction.

The radiographs of two other patients are shown in Figures 150 - 154. They show once again many of the radiological post-myotomy features.

**Case 1** *(Figures 150 - 152)* The patient was a woman of 57 who had dysphagia with regurgitation of mucus and difficulty in swallowing
Unfortunately this film reproduces badly but it is possible to see 3 markers on each picture. The upper two markers are on the muscle margins at the myotomy. The lower one is on the hiatus margin.

While the lower 2 markers remain in much the same relative position, the distance between the upper 2 markers differs. The distance between those on the left is 3 cms. while on the right it is 2 cms. The markers, therefore, move away from one another in certain phases of swallowing.
She complained also of pain in the right shoulder and right side of the chest. Figure 150 shows the barium swallow. At oesophagoscopy, there was food retention in the oesophagus. There was a very inflamed stricture at 35 cms. It could be moved down to 37 cms.

At thoracotomy there was a sliding hiatal hernia. About 3 cms. above the oesophago-gastric junction there was a thickened area in the oesophagus 3.5 cms. in length. The oesophagus was difficult to mobilize. The pleura on the right side was thickened and adherent to the oesophagus. The vagi were thickened and taut. They were divided. The oesophagus between the cardia and the stricture felt soft. An oesophageal myotomy and pyloromyotomy were done and the hernia repaired by Hill's method. No residual stricture is seen on oesophagoscopy.

The post-operative films (Figures 151 and 152) show that the oesophago-gastric junction (lowest markers) is well below the hiatal margin, there is a good oesophago-gastric angle, and the cardia is competent. The typical "wiped joint" appearance is seen on the right hand picture but not on the left. The left hand picture shows the myotomy markers to be 2.5 cms. apart.

Case 2: This patient, an obese woman of 63, had dysphagia for 7 months. She had repeated episodes of food impaction. There was a long previous history of heartburn and indigestion.

Figure 153 shows the pre-operative barium swallow.

At oesophagoscopy, there was a very inflamed stricture at 31 cms. It felt to contain a firm ring. The mucosa above it was intensely inflamed.

There was a sliding hiatal hernia. There was a thickened segment in the oesophagus extending from the cardia upwards for 2.5 - 3 cms. There were enlarged glands in the mediastinum. The vagi were
preserved. A myotomy was done just above the stricture and the hernia repaired by Hill's method.

Figure 154 shows the post-operative barium swallow.

The local distension of the oesophagus at the myotomy is well seen. The oesophago-gastric junction is shown by the markers to be below the hiatus. There is a good oesophago-gastric angle. There is no reflux. There is no sign of a residual stricture.

Discussion of Circumferential Oesophageal Myotomy

It is of interest from the physiological point of view that circumferential myotomy can be carried out without apparent interference with the mechanism of swallowing. Even when the muscle is divided at two levels, swallowing proceeds normally as far as can be judged on x-ray screening and on the absence of symptoms. It has been stated (Samson Wright, 1965) that "the passage of peristaltic waves along the oesophagus depends on the continuity of the preganglionic vagal nerve supply but not on the integrity of the muscle coat. It is claimed that if the oesophageal wall is divided and the superficial nerve plexus left intact, the peristaltic wave can still pass normally over the oesophagus".

One point in relation to the surgical anatomy emerges. As has been said, only a single myotomy has been done as a rule but, on two occasions, double myotomy was done with division of the muscle layers above the stricture and again just below the aortic arch. In spite of this apparently drastic interference with the blood supply to the oesophagus there was no evidence of complications arising from ischaemia of the portion of oesophagus between the myotomies.

This account of the procedure is, at present, more in the nature of the elaboration of an idea than a statement of proved fact. It is difficult for one surgeon to gain sufficient experience from an adequate
The effect of contraction of the longitudinal muscle of the oesophagus in elevating the oesophago-gastric junction has been considered before. Hurst (1934) suggested that, in some cases, the occurrence of a hiatal hernia might be due to traction of the stomach upwards due to shortening of the oesophagus "in response to some vagal stimulation". Rall et al. (1945) showed that electrical stimulation of the vagus in dogs caused shortening of the oesophagus and that this shortening could, in some circumstances, produce a hiatal hernia. Dey et al. (1946) said that shortening of the oesophagus with production of a hiatal hernia due to traction on the stomach in the dog results from electrical stimulation of the vagus nerve or reflexly from stimuli having their origin in the viscera of the upper abdominal cavity. Inglefinger (1958), referring to axial contraction of the oesophagus states that, on swallowing, some upward motion of the oesophagus takes place and that both traction by laryngeal elevation and actual shortening of the oesophageal tube may be responsible. As far as we are aware, an attempt to limit such an upward pull on the oesophago-gastric junction by oesophageal myotomy has not previously been made.

When two or more procedures are combined, it is difficult to assess the relative importance of each one of them. In this instance it remains uncertain if myotomy has been of value in addition to efficient hernial repair alone.

While, at this stage, the value of the myotomy remains unproved, certain observations may be made. Myotomy produces a limited but sometimes significant elongation of the oesophagus. Judging from films and cineradiographs, markers on the cut muscle margins move apart a distance of 2 to 3 cms. and they appear to move away from one another in some phases of swallowing. Such findings suggest that myotomy reduces
certain forces tending to cause recurrence of the hernia. There have been no complications from myotomy. No case of oesophageal rupture or of diverticulum formation has been found so far. Myotomy does not cause dysphagia.

It is suggested that continued trial of the procedure is justified in properly selected cases. It is emphasized that the procedure does not render the obviously irreducible hernia reducible but that it may be helpful in borderline cases where the hernia is reducible with moderate downward traction on the oesophagus. This applies to cases of classical acquired shortening of the oesophagus and also to those where a large sliding hernia may be difficult to reduce, the oesophago-gastric junction having for a long time occupied a high position in the thorax. The operation is doomed to failure not only in the presence of an obviously irreducible hernia but where the local changes at the site of stricture are irreversible.

Circumferential myotomy may have a limited application in carefully selected cases and may extend the indications for treatment of peptic stricture by hernial repair. Whether or not the procedure has any place in the surgical treatment of uncomplicated hiatal hernia remains to be seen.

It is hoped that, if the operation is carried out in departments better equipped to make manometric and motility studies on the oesophagus, some interesting observations may be made on oesophageal function and the competence of the cardia after circumferential myotomy.
CONCLUSION

This Thesis is a clinical and personal account of peptic stricture of the oesophagus. It is obviously difficult to make any new observations from a purely clinical point of view. Our aim has been to record experience, giving a general picture of the condition, while at the same time elaborating certain personal views and ideas.

Without recapitulating all the aspects of peptic stricture which have been described, a few points of emphasis may be mentioned which have not received much attention in the literature.

The figures for age and sex incidence show the increasing vulnerability of the oesophageal mucosa to reflux with advancing age and that, in the seventh decade, this applies particularly to males. There is a tendency in this age group for the stricture to change its character with the development of deeper ulceration.

Most patients continue to have heartburn after they develop dysphagia. There is little evidence to show that a peptic stricture protects the mucosa above it from acid reflux.

There may be an analogy between some cases of ulcerated peptic stricture and fissure-in-ano accounting for the relief from pain which may follow vigorous dilatation of a stricture.

Nutritional problems are uncommon but are met with in infants, in patients with post-operative peptic strictures, and in patients over 70 years of age.

Peptic strictures are commonly divided into high and low ones. From the point of view of selection for surgical treatment, the main difficulties arise with those lying midway between the diaphragm and the aortic arch. Such strictures should be considered as a separate group.
The aetiology of lower oesophageal web remains in doubt. Whether or not it is a form of peptic stricture, it should be considered as a separate entity from the point of view of treatment. The possibility is suggested that the web is not, as is sometimes claimed, secondary to the associated hernia but that this hernia, which is of distinctive type, is secondary to the presence of the web and due to prolonged overaction of the longitudinal muscle of the oesophagus.

Post-operative and post-recumbency strictures form an important group. They account for most peptic strictures in men under the age of 50. Such strictures are, in our opinion, the result of an acute acid burn of the oesophagus.

Post-vagotomy dysphagia must be included in the differential diagnosis of post-operative peptic stricture. In the patients we have encountered with post-vagotomy dysphagia, the condition was due sometimes to trauma and sometimes to peptic stricture formation but not to cardiospasm.

High peptic stricture with lower oesophagus lined by columnar epithelium is by no means uncommon. To emphasize this fact, a number of illustrations of this condition have been included. As to the origin of columnar epithelium in the lower oesophagus, the evidence tends to favour the view that it is a congenital anomaly. We have described it in babies and shown that it may be found above an ulcerated stricture or in an oesophagus without any evidence of ulceration or stricture. One line of enquiry which might be profitably pursued is the incidence of associated congenital anomalies. We describe patients with gastric-lined oesophagus and associated mental defect, inborn error of metabolism, and duodenal diaphragm. The precise aetiology of the anomaly remains uncertain.
The natural history of peptic stricture is of importance in relation to treatment. As it is not necessarily a progressive lesion, conservative management is often satisfactory. Although, in theory, peptic stricture is preventable by hernial repair, the opportunity for prophylactic surgery rarely presents itself in practice.

The success or otherwise of conservative management depends to a large extent on the manner in which it is carried out. It is satisfactory in more than half of the patients. As far as surgical treatment is concerned, we have placed the emphasis on hernial repair where possible. Circumferential oesophageal myotomy may prove to be a means of extending the indications for hernial repair in properly selected cases.

Little attention has been paid to the action of the oesophageal muscle, especially the longitudinal muscle, in causing or perpetuating elevation of the oesophago-gastric junction. By directing attention to the action of the longitudinal muscle it is hoped to encourage further study of this aspect by others. The effect of circumferential myotomy on the function of the oesophagus opens up an interesting field of enquiry in relation to manometric and motility studies.
REFERENCES


J. Laryng., 54, 621.

J. Laryng. and Otol., 51, 77.

Bruce, J. and Small, W. P. (1959). Dysphagia following vagotomy.


Spontaneous rupture of the pharynx

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Spontaneous rupture of the oesophagus is well known to all thoracic surgeons. Spontaneous rupture of the stomach, though rare, is recognized, especially in the new-born. It is perhaps surprising that, as far as we are aware, spontaneous rupture of the pharynx has not previously been described. The history and clinical findings in the following case report are consistent with this diagnosis.

CASE HISTORY

The patient was an obese housewife aged 50 years. The day her pharynx ruptured, her lunch consisted of chips and gravy followed by a steamed fruit pudding which she had made herself. To use her own words, she 'swallowed a mouthful of pudding unexpectedly'. The pudding seemed to stick in her throat and she was immediately seized with intense pain on the left side of the neck. She was at once aware also of a swelling about the size of an egg on the left side of the neck. She thrust her fingers down her throat to induce vomiting and brought up some blood. The pain became even worse and the swelling increased.

She was rushed to hospital on the pillion of a motorcycle. This was her first experience of this mode of transport.

At the hospital, which she reached one hour after the onset of pain and swelling, she was found to be moderately shocked. She complained of agonizing pain all round her neck, between the shoulder blades, and over the right shoulder blade. There was extensive surgical emphysema of the neck. The pharynx was inspected but no cause was found to account for her condition.

She was transferred to the Thoracic Surgical Unit of the Bradford Royal Infirmary about four hours after the beginning of the episode. The pain was so severe that she was unco-operative. She said she just wanted to die. Her blood pressure was 130/70 mm. Hg. (The blood pressure recorded after recovery was 180/110 mm. Hg.) There was gross swelling due to surgical emphysema which involved both sides of the neck, the face, and the upper part of the chest anteriorly. The surgical emphysema appeared to be spreading.

The only additional history obtained at this stage was that she had suffered from chronic bronchitis and that she had had a goitre for many years.

She was given gastrografin to swallow and a radiograph of the neck was taken two minutes later. Most of the opaque medium passed down the oesophagus but some of it remained in the neck just above the level of the cricoid cartilage. The gastrografin lodging in the neck was irregularly disposed and extended laterally on the left side.

OPERATION. An endotracheal tube was passed without difficulty, and no abnormality of the vocal cords was seen.

An oesophagoscope was passed into the pharynx. There was blood in the lower part of the pharynx. What appeared to be a mucosal tear was seen on the left side. The cricopharyngeal sphincter was not seen and no attempt was made to pass the instrument beyond the site of the lesion. When the oesophagoscope was pressed a little towards the left, several currants entered the lumen of the pharynx apparently through a breach in its wall.

An incision was made along the anterior border of the left sterno-mastoid muscle. Access to the pharynx was impeded by the presence of an adenoma of the left lobe of the thyroid. The adenoma was oval in shape and measured 2 in. (50.8 mm.) in its long axis. It lay posteriorly behind the left lobe of the thyroid and was closely applied to the upper part of the oesophagus. The thyroid adenoma was removed. The left side of the pharynx and upper oesophagus were then exposed. The tissues were oedematous. Beside the pharynx there was some dark-coloured fluid and some pudding. A clean longitudinal split 1½ in. (31.75 mm.) long was found in the left postero-lateral wall of the pharynx, extending from a point immediately above the cricopharyngeal sphincter to the hyoid bone. The rent was quite straight. The edges of the torn mucosa were smooth. They pouted a little. Apart from the rent, there was no sign of any abnormality of the mucous membrane. A finger was inserted into the pharynx and also into the oesophagus through the cricopharyngeal sphincter. No foreign body was found. The mucosa felt normal.

The tear in the mucous membrane was closed with a continuous catgut suture, and a similar suture was used to bring together the pharyngeal muscle. The only difficulty in the closure of the pharynx arose at the upper end of the rupture which was difficult to reach until the submandibular salivary gland had been dissected upwards for a short distance. The extent of the mucosal tear was slightly greater than that in the muscle. The rent in the muscular coat had to be extended a little at each end in order to demonstrate accurately the limits of the mucosal rupture.
On regaining consciousness, the patient expressed relief that the intense pain had gone.

A small tube drain to the suture line was removed after two days. The patient was given crystalline penicillin for 10 days. She was allowed to swallow liquids on the day after operation. The post-operative course was uncomplicated.

After the operation it was possible to enquire more closely into the patient’s previous history. She told a strange story. Apparently she had always had to be careful when eating. No one was allowed to speak to her during a meal. If her attention was distracted from the act of swallowing she was inclined to choke. In spite of this, she had always eaten well and taken normal food. She was over-weight. She had never suffered from anaemia. She was rather a jolly person and not given to complaining about her health.

A barium swallow was given two weeks after the operation. No abnormality was seen in the pharynx. Deglutition appeared to be normal. A small hiatal hernia was demonstrated in the Trendelenburg position but gastro-oesophageal reflex could not be produced. There were some mild tertiary contractions in the oesophagus.

DISCUSSION

It is necessary to distinguish between, on the one hand, perforation of the pharynx due to disease, foreign body, or the passage of an instrument, and, on the other, a spontaneous rupture. No disease was found in the pharynx in this patient nor was there any sign of a foreign body. The bolus consisted of soft pudding. An oesophagoscope was passed into the pharynx but no attempt was made to pass it beyond the lesion. The rupture in the pharyngeal wall was a clean longitudinal split 1 1/2 in. (31.75 mm.) long in apparently healthy tissues. The lesion was in all respects comparable with that found in spontaneous rupture of the oesophagus.

The swelling of the neck and the onset of pain were synchronous. The sudden onset and intensity of the pain were just as dramatic as the initial symptoms of spontaneous rupture of the oesophagus, although the degree of shock was less. In the latter condition, extravasation may for a time be confused by the pleurae within the mediastinum. There may be some delay before surgical emphysema appears in the neck. In our patient, surgical emphysema developed immediately. The escaping air was not apparently confined by fascial planes.

Spontaneous rupture of a hollow viscus due to raised pressure within it often has a predilection for one particular site. Thus the oesophagus ruptures in its lower part on the left side. Whether or not there is a site of predilection for spontaneous rupture of the pharynx cannot be stated in the absence of comparable cases. However, in a case of pneumatic rupture of the oesophagus described by Cole and Burcher (1961), where a fire extinguisher accidentally discharged in the patient’s face, rupture of the oesophagus occurred at the site characteristic of spontaneous rupture.

Kerr, Sloan, and O’Brien (1953) describe rupture of the lower oesophagus on its left side in a child who had bitten an inflated inner tube. From the point of view of pharyngeal rupture, it is of interest, therefore, that Hood (1957) records a patient in whom rupture of the pharynx and upper oesophagus occurred as a result of blast from a fire extinguisher directed into the nose and mouth. The pharyngeal rupture occurred on its left postero-lateral wall, presumably in the same situation as the spontaneous rupture in our patient.

As to aetiology, the lesion showed all the signs of having been caused by bursting pressure from within the pharynx. It occurred on swallowing. There are three factors of possible significance. The presence of a goitre may have interfered with deglutition. The patient’s strange account of her need to concentrate on the act of swallowing suggests the possibility of some kind of neuromuscular incoordination. A diagnosis of syringobulbia was not established. The radiological finding of tertiary contractions in the oesophagus on barium swallow is perhaps further evidence in favour of disordered function.

SUMMARY

A case is described of rupture of the pharynx during the act of swallowing.

Reasons are given as to why the rupture was considered to be a spontaneous one.

Aetiological factors are suggested.

REFERENCES


RESECTION OF SQUAMOUS-CELL CARCINOMA
OF THE OESOPHAGUS WITH END-TO-END OESOPHAGEAL
ANASTOMOSIS

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Resection of a carcinoma of the oesophagus with end-to-end oesophageal anastomosis is an operation rarely performed. Parker and Brockington (1949) reported 2 patients in whom the operation was done for palliation of dysphagia. One patient lived for 9 months. Kunkel and Kunkel (1958) reported 4 cases of middle-third carcinoma treated in this way. One patient, a woman of 56 years, was alive and well after 9½ years, although her postoperative course was complicated by the development of an oesophago-pleurocutaneous fistula and a broncho-oesophageal fistula. Subsequently she required periodic dilatations though dysphagia was apparently only slight. One patient lived for 9 months, during which time he was able to swallow soft diet without difficulty. He died from recurrence of the disease. The other 2 patients died as a result of anastomotic leaks.

The two main reasons why the operation is rarely done relate to the anatomy of the oesophagus and the pathology of oesophageal carcinoma.

Although anatomists describe the oesophagus as having several curvatures, from the surgeon's point of view it takes the shortest route from the neck to the oesophageal hiatus of the diaphragm, allowing for the space occupied by the other mediastinal structures. This being so, there is no advantage in attempting to re-route the oesophagus in the chest.

The oesophagus can be stretched very little. This is largely due to the fact that its mucosa, though thrown into longitudinal folds when the oesophagus is empty, is without transverse folds. An important additional factor causing difficulty in bringing together the cut ends of a transected oesophagus is the contraction of the longitudinal muscle. This contraction distracts the cut ends and, once the longitudinal muscle has been allowed to contract, it is remarkably difficult to make it assume its former length by simple traction.

The direction of spread of carcinoma of the oesophagus is predominantly longitudinal. Failure to excise an adequate length of oesophagus is the common cause of recurrence of growth at the anastomosis. Miller (1962), after analysis of 405 cases, recommends division of the oesophagus not less than 6 cm. above an adenocarcinoma and not less than 12 cm.
above a squamous-cell tumour. For lower-third tumours Garlock and Klein (1954) advise the use of supra-aortic anastomosis, while for middle-third carcinomas total oesophagectomy is advocated by Nakayama (1959). If such views are to be accepted in all cases, there is no place for limited resection and end-to-end anastomosis, as this operation involves division of the oesophagus close to the growth.

After a further 3 months, her condition deteriorated rapidly. She developed metastases in the occipital bone and in the liver. Death occurred 1 year after operation.

At autopsy there was no recurrence of growth at the suture line, but there were glandular metastases in the mediastinum.

**Comment.**—Although, according to the histological findings, the resection was inadequate,

This paper describes experience with 10 resections of squamous-cell carcinoma of the oesophagus with direct anastomosis. It is hoped to show that, although the operation has obvious limitations, it can, on occasion, be remarkably successful.

**CASE REPORTS**

**Case**

A hypertensive woman aged 69 years had a squamous-cell carcinoma at the junction of the middle and lower thirds of the oesophagus (Fig. 1 A). The growth was short and was excised through a left thoracotomy with end-to-end oesophageal anastomosis on 15 June, 1955.

Histological section showed an anaplastic squamous-cell carcinoma infiltrating the muscle layers up to each end of the specimen.

A barium swallow (Fig. 1 B) 2 weeks after operation showed a localized leak from the suture line. This did not give rise to symptoms.

After 6 months the patient began to have slight dysphagia. A barium swallow 7 months after operation (Fig. 1 C) showed some narrowing at the anastomosis. At oesophagoscopy a smooth stenosis was seen. A biopsy failed to show recurrence of neoplasm. The stricture was dilated. Nine months after operation the patient was able to swallow suet dumplings, meat, and potato pie.

**Fig. 1.—Case 1.** A, Preoperative barium swallow. B, Barium swallow 2 weeks after operation showing a localized anastomotic leak. C, Barium swallow 7 months after operation showing slight narrowing at the anastomosis.

A biopsy failed to show recurrence of neoplasm. The stricture was dilated. Nine months after operation the patient was able to swallow suet dumplings, meat, and potato pie. recurrence at the suture line did not occur. Death was due to mediastinal and more distant metastases. Whether or not more extensive excision would have given a better result is open to question.

Dysphagia was distressing during the last 2 months of the patient’s life.

**Case 2.**—A woman aged 46 years, with a carcinoma at the junction of the middle and lower thirds of the oesophagus (Fig. 2). At left thoracotomy on 8 Jan., 1957 a small carcinoma was resected. End-to-end anastomosis of the oesophagus was easy.

Six months later the patient had transient dysphagia. An adult full-lumen Negus oesophagoscope passed easily down to the cardia. The patient rapidly gained 3 st. in weight. Nine years later she remains alive and well.

Twice in this time she has had food impaction in the oesophagus. On each occasion a lump of meat was stuck at the level of the anastomosis though, at oesophagoscopy, no stenosis could be seen.

Before operation the patient weighed 8 st. 7 lb. Her weight has remained at just over 11 st. since. Apart from the episodes of food impaction she has been entirely free from dysphagia.

Histological section showed the presence of a squamous-cell carcinoma. Both ends of the resected specimen were free from growth.
Comment.—This operation was successful, resulting in long survival in excellent health. Dysphagia has not been a problem.

Case 3.—An emaciated man, aged 74 years, with a carcinoma in the lower third of the oesophagus. On admission to hospital he had dysphagia pneumonitis with a temperature of 102° F. The pneumonitis became worse rather than better in spite of treatment with antibiotics. Operation was performed on 19 Feb., 1958 while the patient was still febrile. At left thoracotomy consolidation of the left lower lobe was seen. The growth was small and hard. A short portion of the oesophagus was removed with end-to-end oesophageal anastomosis. The patient made a rapid recovery. He had no symptoms until he died 6 years later at the age of 80. The cause of death is not known.

Histological section showed a squamous-cell carcinoma which had penetrated through the full thickness of the oesophageal wall and invaded the paraoesophageal tissues. Mediastinal glands removed did not contain metastases.

Comment.—This operation was successful, resulting in 6 years’ survival in excellent health. It had the advantage of being a relatively quick and easy operation in an elderly man with pneumonitis due to dysphagia.

Case 4.—A man aged 56 years had a carcinoma of the oesophagus, the upper limit of which was close to the lower margin of the aortic arch (Fig. 3). At left thoracotomy on 18 June, 1958, the growth was found to involve the pleura on its right side, and to be associated with enlarged mediastinal glands. The growth was resected with difficulty and end-to-end anastomosis done. A barium swallow 2 weeks after operation showed a substantial, though localized leak at the suture line.

After only 3 months the patient developed dysphagia with a hard, tight stricture. Biopsy at this stage showed no evidence of recurrence of growth at the anastomosis. Temporary relief of the dysphagia followed dilatation of the stricture on three occasions in the next 3 months. Five months after operation a Souttar’s tube was inserted. The patient then gained a stone in weight. Dysphagia became increasingly severe in the last 2 months of life and the patient died 1 year after operation.

Histological section of the resected specimen showed a squamous-cell carcinoma infiltrating through the full thickness of muscle coats but not reaching the ends of the specimen. No metastases were found in mediastinal glands.

At autopsy, the patient was found to have recurrence of growth at the anastomosis.

Comment.—The growth involved the pleura on its right side. In view of the recurrence at the anastomosis it is apparent that longitudinal resection was inadequate. The patient’s comfort during his 1 year of life was marred by dysphagia. A better result might have been obtained by more radical surgery.

Case 5.—A mentally deranged man aged 72 years had a carcinoma in the middle third of the oesophagus. The lesion was approached by a right thoracotomy on 13 Aug., 1958. It extended from just above the vena azygos to a point 4 cm. below the vein. The growth was mobilized, but growth had to be left behind adherent to the aortic arch. During dissection of the growth there was much bleeding. The main mass of the growth was excised and end-to-end oesophageal anastomosis carried out. The immediate postoperative radiograph showed a bulging to the right of the upper mediastinal shadow, presumably due to a haematoma. The patient died on the eleventh postoperative day from disruption of the anastomosis.

Histological section showed a moderately differentiated squamous-cell carcinoma which had infiltrated through the muscle layers.

Comment.—The lesion was inoperable because of involvement of the aortic arch. Disruption of the anastomosis was due to the formation of an infected haematoma, a technical fault.

Case 6.—A man aged 45 years had a carcinoma in the upper third of the oesophagus (Fig. 4). Bronchoscopic examination showed bulging of the posterior wall of the
trachea. At right thoracotomy on 30 June, 1959 the
growth was found to extend from the vena azygos up¬
wards to within a short distance of the apex of the pleura.
It was difficult to separate the growth from the trachea.
Malignant spread appeared to have occurred in tissue
adjacent to the left main bronchus. The carcinoma was
resected and end-to-end oesophageal anastomosis carried
out.
Postoperatively there was surgical emphysema in the
neck and chest wall for a few days. Otherwise, recovery
was uneventful.
Five weeks later the patient had recurrence of dysphagia.
A stricture at the suture line was dilated and the patient
taught self-bougineage. He was then treated with cobalt-
beam therapy.

![Fig. 4.—Case 6. Barium swallow showing a carcinoma in the
upper third of the oesophagus.](image)

After 6 months the patient was passing his bougie only
once every 3 weeks. He discontinued bougineage 18
months after operation. He is alive and free from
symptoms 61 years after operation.

Histological section of the resected specimen showed
the presence of a poorly differentiated squamous-cell
carcinoma which had infiltrated through the entire thick¬
ness of the oesophageal wall. The ends of the specimen
were apparently free from growth.

**Comment.**—Local resection and postoperative
radiotherapy for a carcinoma in the upper third of the
oesophagus resulted in long survival in excellent
health. The problem of dysphagia was overcome.

**Case 7.**—An emaciated man of 38 years, with a car¬
cinoma of the oesophagus just below the level of the aortic
arch. A left thoracotomy was done on 7 Sept., 1960. The
pleura on the right side was involved and the growth
extended into the right lung. A portion of the right lung
was removed with troublesome air-leaks. It seemed
likely that growth had been left behind in the right lung.
The carcinoma was resected and oesophageal anastomosis
carried out.

One month later the patient was swallowing soft foods,
but could not swallow meat. He was treated with high-
energy radiation. During this treatment the dysphagia
increased and remained a problem for the rest of the
patient’s life. At oesophagoscopy there was a hard
stricture which did not respond well to dilatation. A
Southar’s tube was inserted with temporary partial relief
of the dysphagia.

Three months after the operation a large metastasis
was palpable in the left humerus.
The patient died 5 months after operation.

Histological section of the resected specimen showed a
squamous-cell carcinoma with metastases in mediastinal
glands.

At autopsy there was a fibrous stricture infiltrated by
active, somewhat undifferentiated, epidermoid carcinoma.
This had extended into a bronchus. In addition, the
ghosts of old cell nests were seen, presumably the result of
radiotherapy.

**Comment.**—Treatment failed completely. The
operation did not succeed either in adequately
removing the growth or in giving relief from dys¬
phagia. As the growth invaded the right pleura and
lung, was associated with metastases in mediastinal
glands, and produced a large distant secondary in
bone after only 3 months, it seems unlikely that a
better result would have been achieved by more
radical surgery.

**Case 8.**—A frail, emaciated woman of 89 years, with
a malignant stricture in the lower third of the oesophagus.
At left thoracotomy on 13 Sept., 1960, a small hard
growth was resected with end-to-end oesophageal
anastomosis. A firm lump was felt through the diaphragm.
The diaphragm was incised and a malignant gland mass
removed from the gastrohepatic omentum. The patient
made a rapid and uneventful recovery and went to a
geriatric hospital 2 weeks after operation. She died 6
months later. According to her doctor she remained free
from dysphagia. He attributed her death to generalized
arteriosclerosis.

The growth was an undifferentiated squamous-cell
carcinoma infiltrating the muscle coats but not reaching
the ends of the specimen.

**Comment.**—In view of the patient’s age, her
death from unrelated causes, and her freedom from
dysphagia, the operation may be regarded as satis¬
factory.

**Case 9.**—A woman aged 57 years. Oesophagoscopy
had been carried out elsewhere and a malignant stricture
dilated. This procedure had been followed by severe pain
between the shoulder blades. At right thoracotomy on
4 Oct., 1961 there was a carcinoma of the middle third of
the oesophagus associated with mediastinitis. The growth
was difficult to mobilize and, owing to the peri-oesopha¬
gitis, it was not easy to define its longitudinal extent. The
carcinoma was excised and the cut ends of the oesophagus
anastomosed.

Histological examination showed a squamous-cell
carcinoma which extended to the upper end of the resected
portion of oesophagus.

Oesophagoscopy was performed a month later. There
was an irregular stricture with slough at the suture line.
Two biopsies were taken. One consisted of a mass of
bacteria and leucocytes surrounding a piece of suture
material. The other showed chronic inflammatory reaction
in the submucosa.

The patient began to have dysphagia 2 months after
operation. There was a slight degree of narrowing at the
anastomosis. Just above this there was some irregular
tissue, a biopsy from which showed squamous-cell car¬
cinoma. At this stage a malignant gland was felt in the
left supraclavicular fossa.

The patient was given radioactive cobalt-beam therapy
to the oesophageal lesion. At the end of this treatment
she could swallow any food except meat.

The patient then remained well for 8 months with only
slight dysphagia. A further oesophagoscopy was done.
There was a soft web-like stricture which dilated easily. Further biopsies showed no malignant recurrence.

Four months after operation the gland in the left side of the neck was treated with radiotherapy. Shortly after this a secondary in the left tonsil was also treated with radiotherapy.

Nineteen months after operation a malignant mass was removed from the left side of the neck.

At the end of 2 years the chest radiographs showed recurrence of growth in the mediastinum. In spite of this the patient was able to swallow quite well. Eventually, she became increasingly short of breath owing to tracheal and bronchial compression. Death occurred 2 years and 7 months after operation.

Comment.—The results of surgery were poor. There was, however, evidence of glandular involvement in the neck as early as 2 months after operation. The failure of the operation was due to the development of secondaries in the neck, the left tonsil, and, finally, in the mediastinum.

Excision of the growth was inadequate. It is interesting, and perhaps of importance, that though recurrence was proved to have occurred above the suture line 2 months after operation, radiotherapy apparently destroyed the local recurrence, which was not found again in the subsequent 2 years and 5 months of the patient’s life.

Case 10.—A woman aged 52 years had a carcinoma in the middle third of the oesophagus. At right thoracotomy on 28 Aug., 1962 the growth was found to extend from the level of the azygos vein for a short distance downwards. It was separated with difficulty from the region of the tracheal bifurcation and from the pericardium. The oesophagus was mobilized from the apex of the pleura down to the diaphragm. The carcinoma was resected and end-to-end oesophageal anastomosis carried out.

The patient was given high-energy radiation postoperatively. At the end of the course she was swallowing well.

Ten months after operation a malignant recurrence in the left side of the neck was treated with radiotherapy. Later the patient’s condition deteriorated rapidly. She had a large secondary in the region of the left sacro-iliac joint and another in the left lung. She died 16 months after operation.

Histological section showed a poorly differentiated squamous-cell carcinoma. Examination of the lines of section showed one end to be infiltrated by growth in the subepithelial layer.

Comment.—The results of surgery were poor. Local excision was inadequate, but local recurrence did not take place, possibly because of the effect of postoperative radiotherapy.

RESULTS

The results of surgery are summarized in Table I. As far as the results are concerned, the patients fall into three groups:

1. One patient with an inoperable tumour died from disruption of the anastomosis.
2. Four patients had good results. One, with a carcinoma at the junction of the middle and lower thirds of the oesophagus, is alive and free from symptoms after 9 years. One with a carcinoma in the upper third is alive and well after 6 1/2 years. The other 2 patients died at the age of 80 and 90 years respectively, the former having lived in good health for 6 years.
3. In 5 patients the results of surgery were poor. Survival ranged from 5 months to 2 years and 7 months. In 3 patients there was histological evidence of inadequate excision. Of these, 1 died after a year without recurrence in the oesophagus, 1 had early local recurrence successfully treated with radiotherapy and 1, who also had postoperative radiotherapy, did not develop local recurrence.

Recurrence at the anastomosis took place in the other 2, though in neither was growth found at the ends of the resected specimens. In both the growth had extended into the pleura or into the lung.

The essentials for success with this operation are correct selection of cases and sound operative technique.

Table I.—Summary of Treatment and Results

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (Years)</th>
<th>Surgery only</th>
<th>Surgery and Radiotherapy</th>
<th>Survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>69</td>
<td>+</td>
<td>-</td>
<td>1 yr.</td>
</tr>
<tr>
<td>2</td>
<td>46</td>
<td>+</td>
<td>-</td>
<td>Alive after 9 yr.</td>
</tr>
<tr>
<td>3</td>
<td>74</td>
<td>-</td>
<td>+</td>
<td>6 yr.</td>
</tr>
<tr>
<td>4</td>
<td>56</td>
<td>-</td>
<td>+</td>
<td>1 yr.</td>
</tr>
<tr>
<td>5</td>
<td>72</td>
<td>-</td>
<td>+</td>
<td>1 yr.</td>
</tr>
<tr>
<td>6</td>
<td>45</td>
<td>-</td>
<td>+</td>
<td>Alive after 61 yr.</td>
</tr>
<tr>
<td>7</td>
<td>38</td>
<td>-</td>
<td>+</td>
<td>6 mth.</td>
</tr>
<tr>
<td>8</td>
<td>80</td>
<td>-</td>
<td>+</td>
<td>6 mth.</td>
</tr>
<tr>
<td>9</td>
<td>57</td>
<td>-</td>
<td>+</td>
<td>7 yr.</td>
</tr>
<tr>
<td>10</td>
<td>52</td>
<td>-</td>
<td>+</td>
<td>1 yr. 4 mth.</td>
</tr>
</tbody>
</table>

CASE SELECTION

Although the surgeon might wish to reserve this operation for very old or very ill patients, this form of case selection does not apply. The practicability of the operation is dictated entirely by the size and nature of the growth. There are several factors of importance in the selection of cases.

1. Radiology.—The length of a carcinoma of the oesophagus cannot be accurately assessed radiologically. However, the barium swallow is of some help. If the lesion is obviously longer than 5 cm. or if it casts a soft-tissue shadow, resection with end-to-end oesophageal anastomosis need not be considered.

2. Oesophagoscopy.—The lesion should be seen as a smooth, hard stricture. It should not be fungating or show deep ulceration. Some indication of the length of the stricture may be gained by passing a Chevalier Jackson bougie through it and withdrawing the bougie until its shoulder is felt to catch on the lower limit of the stricture.

3. Operative Findings.—The lesion must be not more than 4 or 5 cm. in length. The oesophageal wall above and below the growth should feel soft. Thickening of the wall may be due to malignant infiltration, to inflammation and oedema in association with an ulcerated carcinoma, or to muscle hypertrophy above a stricture. It is difficult to distinguish macroscopically between them. Not only is the possibility of malignant infiltration a contraindication to limited resection, but inflammation and oedema of the oesophageal wall make it difficult to bring the cut ends together. Ideally, therefore, one looks for a short, hard growth with soft, lax oesophageal wall above and below it.

4. Anastomosis.—Important information is obtained during the performance of the anastomosis. If there is any tendency for the sutures to cut out, the attempt at end-to-end anastomosis should be abandoned and another type of operation done. Thus the
surgeon must keep an open mind about the advisability of this operation until the final stages of the anastomosis.

TECHNIQUE

After resection of even a short length of oesophagus, anastomosis of the cut ends must always be done under a certain amount of tension. The technique of anastomosis plays a large part in making the operation possible without disastrous anastomotic leaks. In the standard oesophageal anastomosis it is customary to begin with a posterior row of sutures through the muscle coats. With even slight tension at the suture line the muscle coats do not hold stitches well. In carrying out end-to-end oesophageal anastomosis the standard method is, in our opinion, difficult. The following technique is suggested.

The oesophagus above the growth is first of all cleaned out at oesophagoscopy.

Upper- and middle-third growths are approached by right thoracotomy and lower-third growths by left thoracotomy. We have not made a practice of opening the abdomen. Pylorospasm has not been encountered in spite of vagotomy, though we have perhaps been fortunate in this.

The growth is mobilized. A little extra length can be obtained by freeing the oesophagus above and below the lesion. This applies particularly to separation of the oesophagus from the posterior wall of the trachea and from the region of the aortic arch and tracheal bifurcation.

The muscle coats of the oesophagus are then infiltrated with local anaesthetic over all the accessible length of oesophagus. This helps considerably in preventing contraction of the longitudinal muscle.

Stay sutures are placed through the whole thickness of the oesophageal wall above and below the proposed lines of section. During division of the oesophagus the stay sutures are kept taut, the lower ones being pulled in an upward direction and the upper ones in a downward direction, so that distraction of the cut ends is prevented.

The muscle coats are divided circumferentially above and below the palpable extent of the growth, leaving the mucosa intact for the time being. When the mucosa is displayed in this manner the mucosal extent of the growth can be more readily seen. The mucosa is then divided, usually within ½–1 cm. of the apparent limits of the growth. No clamps are used.

The anaesthetist threads a Ryle's tube down the inside of a rectal tube so that the lower end of the Ryle's tube projects about 2 in. from the front end of the rectal tube. The rectal tube, with its contained Ryle's tube, is then passed through the mouth down the oesophagus and threaded into the lower portion of the oesophagus and on into the stomach.

An assistant grips the upper and lower segments of oesophagus and approximates them by sliding them towards one another along the rectal tube. By appropriate rotation of the upper and lower parts of the oesophagus around the tube, the surgeon is enabled to place all his stitches on the side of the oesophagus facing him, and an awkward back layer is avoided. For the anastomosis we use two layers of continuous 0 catgut on a 30-mm. intestinal needle. The first layer picks up all coats of the oesophageal wall and is locked on alternate stitches. Taking the stitches through all layers gives them a very good grip. As the continuous suture progresses the cut ends come together gradually, and tension on any one stitch is reduced to a minimum after the first two or three have been successfully inserted.

It is advisable to stop the first continuous suture a short way before its completion and insert three interrupted sutures in the last part of the anastomosis, leaving them untied until all three have been placed. If one tries to complete the continuous suture it is difficult to be sure that the last stitches pick up the mucosa. The first continuous suture is completed after the interrupted stitches have been tied. The oesophagus is again rotated around the tube and exactly the same procedure followed with a continuous suture through the muscle coats. This time interrupted stitches are not required.

The rectal tube is felt for through the intact diaphragm as it lies in the upper part of the stomach. The projecting Ryle's tube is grasped by the surgeon while the anaesthetist withdraws the rectal tube, leaving the Ryle's tube in position.

POSTOPERATIVE STRicture AND DYSPHAGIA

Of the 9 patients who left hospital significant dysphagia occurred in 4. In 1 patient (Case 1) dysphagia developed after 9 months and was due to recurrence in the mediastinum. In 2 (Cases 4 and 7) there was early postoperative dysphagia. In both the growth was found at operation to have extended transversely into lung or pleura. One patient (Case 6) had early postoperative dysphagia which was completely overcome by a temporary period of self-bougination.

Stricture at the suture line is due either to malignant recurrence or to fibrosis. Recurrence of growth at the anastomosis is an indication of inadequate
excision of the tumour. Stricture is a well-recognized complication of the surgical treatment of congenital oesophageal atresia and is likely to occur where the anastomosis is under tension. Similar circumstances obtain after limited excision of an oesophageal carcinoma, but with correct case selection there should be only slight tension at the suture line. In most of our patients a barium swallow was given 2 weeks after operation. The films suggest that stricture follows local breakdown of the anastomosis with formation of granulation tissue and subsequent fibrosis. There was usually a localized escape of barium of varying degree. When there is minimal escape of barium (Fig. 5) it is unlikely that fibrous stricture will develop. A somewhat larger, though still localized, leak occurred in 1 patient (Case 2) (Fig. 6 A). This leak did not cause symptoms. A barium swallow 5 months later (Fig. 6 B) shows the anastomosis intact. Three years later (Fig. 6 C) the mucosa is seen to be quite smooth and without stricture. On the other hand, in 1 patient (Case 4) where there was a much larger leak, though still contained within the mediastinum (Fig. 7), a tight fibrous stricture was found 3 months later.

**DISCUSSION**

Is the operation ever justifiable? Experience with only 10 patients leaves this question unanswered. The most optimistic impression of the results is obtained by removing from discussion patients in whom the lesion was inoperable. In this way 4, and possibly 5, of the patients may be ignored. They were patients in whom irremovable carcinoma had to be left behind, or where the growth had invaded the pleura and lung, or where distant metastases were discovered shortly after the operation. There remain 5 patients, in 4 of whom the results of surgery were good, and only 1 patient in whom it appeared in retrospect that more radical surgery might have given a better result. Taking this perhaps biased view, it might be claimed that the operation is justifiable subject to the strict case selection already outlined.

From the evidence of much larger series it must be accepted that failure to excise an adequate length of oesophagus is the main cause of local recurrence. On the other hand, the results of even the most radical surgical procedures are disappointing. Successful surgery depends more on the nature of the growth than on the type of operation performed. With a small hard growth of the upper or middle third of the oesophagus, limited excision with end-to-end anastomosis may well be justifiable. The problem of the small lower-third carcinoma is more difficult. However, Cases 2 and 8 show that limited excision may give good results.
This short series gives some support to the view that limited excision with postoperative radiotherapy is a form of treatment worthy of consideration. The efficacy of radiotherapy is inversely proportional to the volume of the tumour. It is logical to consider, as has been done particularly with carcinoma of the breast, that local excision and radiotherapy may be preferable to either treatment by itself. Two of our patients are of interest in this connexion. One (Case 9) had local recurrence which was apparently eradicated by radiotherapy. The other (Case 10), in whom there was histological evidence of inadequate excision, did not develop recurrence at the anastomosis.

Surgical treatment for oesophageal carcinoma should achieve freedom from dysphagia. A disadvantage of limited excision with end-to-end anastomosis is that the incidence of postoperative dysphagia is probably greater than with other procedures. The method carries not only the risk of local recurrence but also the risk of dysphagia from fibrous stricture formation at the suture line. Dysphagia may, of course, occur from peptic stricture following oesophagogastrostomy but is rare after oesophagojejunostomy. When a stricture does form after end-to-end oesophageal anastomosis it is often web-like and responds well to dilatation.

The operation, therefore, has certain disadvantages. It is limited in its application, being suitable only for the somewhat rare, small hard growth. The operation contravenes the rule of wide excision in cancer surgery. It is probably associated with a higher incidence of postoperative dysphagia than oesophagogastrostomy or oesophageal replacement with jejunum or colon.

It has advantages. It is a relatively short procedure and, when practicable, is suitable for very old and very ill patients. When it is successful the patient is left with a normal alimentary tract, apart from the possible effects of vagotomy. His nutrition is excellent. He does not suffer from anaemia of either sideropenic or megaloblastic type. There are no late sequelae from derangement of calcium metabolism. He does not suffer from oesophagitis. The 3 long survivors in the present series are the best results of surgery for oesophageal cancer which the author has achieved by any type of operation.

It cannot be overemphasized that any attempt to extend the indications for this operation is doomed to failure.

**SUMMARY**

Ten patients are described in whom a carcinoma of the oesophagus was resected with end-to-end oesophageal anastomosis. In 4 patients the operation was combined with postoperative radiotherapy.

The selection of suitable cases is outlined.

A technique of anastomosis is described.

The advantages and disadvantages of the operation are discussed.

**REFERENCES**


GASTRIC ULCER IN ASSOCIATION WITH HIATAL HERNIA

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Collier, Hurst, and Sheaf in 1929 reported two cases of gastric ulceration associated with hiatal hernia. In both the ulcer was on the lesser curvature in the “isthmus” of stomach between the hernia and the abdomen. Since that time gastric ulceration at the level of the diaphragm, in association with hiatal hernia, has become well recognised. The ulcers described by Collier et al. were large, chronic, and penetrating. Such ulcers are easy to recognise by radiography, but recognition at an earlier stage may be more difficult. A gastric ulcer may develop in a so-called gastric-lined oesophagus, or in the herniated portion of stomach. Such ulcers have been well described by Barrett (1950, 1954), and by Allison and Johnstone (1953). When the presence of a gastric ulcer is suspected, particular attention should be paid to the stomach at the level of the diaphragmatic hiatus both on preoperative radiographs and during the operation itself. At this site a non-penetrating ulcer may easily be overlooked unless specially sought.

The following case-records illustrate the features of this ulcer, and the type of hernia found in the first four cases is illustrated in fig. 1. This is described by Johnstone (1955) as a “sliding hernia resembling para-oesophageal type due to increased herniation of the greater curvature”.

Case-records

Case 1.—An obese woman of 51 had for some years suffered from heartburn. In October, 1957, she began to have severe pain under the left costal margin. The pain was most severe immediately after meals. It persisted and was but little relieved
by alkalis. She became pale and listless. Haemoglobin 41%. Occult blood was present in the stools. A barium swallow (fig. 2) showed a hiatal hernia and a gastric ulcer at the level of the diaphragm. At oesophagoscopy, some oesophagitis was seen near the cardia with superficial linear erosions of the squamous mucous membrane, but no deep oesophageal ulceration. At operation (Feb. 18, 1958) an indurated area on the lesser curvature of the stomach was found, 1 1/2 in. from the cardia, and adjoining the anterior margin of the hiatus. Haemorrhagic stippling appeared on its peritoneal surface when it was touched. The indurated area was not adherent. There was very little muscle tissue between the anterior border of the hiatus and the central tendon of the diaphragm. After reduction of the hernia, the patient experienced rapid relief from her pain. Tests for occult blood in the stools were negative a week later. The anaemia disappeared. A barium meal 14 days after operation showed no gastric ulcer.

Case 2.—An obese women of 69 had a history of postural heartburn for many years. For 2 years she had had severe burning pain in the epigastrium especially after meals. In April, 1957, she fainted and had haematemesis and melena. She was tender under the right costal margin. Hb 49%. Occult blood present in the stools. A barium swallow (fig. 3) showed a hiatal hernia with a gastric ulcer at the hiatal level. At oesophagoscopy, little oesophagitis was apparent. At operation (May 14, 1957) there was found to be very little muscle tissue at the front of the hiatus. There was an indurated area on the lesser curvature 1 1/2 in. from the cardia, lying against the anterior margin of the hiatus, but no adhesions at this point. The hernia was reduced. The patient had no further pain. Her anaemia rapidly disappeared. A barium meal 15 days later did not show a gastric ulcer. The patient has been symptom-free for a year.

Case 3.—An obese, dyspneic woman of 60, with diabetes, was seen in 1954 because of severe epigastric pain which was persistent and not relieved by alkalis. Her gallbladder had been removed a year previously without relief. A subsequent X-ray showed a hiatal hernia but not a gastric ulcer. She continued to have epigastric pain and some heartburn, but because of her poor general condition was not operated upon. She was admitted on Oct. 13, 1956, with severe pain and ten-
Fig. 2—Barium swallow in case 1.

derness in the left hypochondrium. For 3 months she had had repeated vomiting, occasionally of altered blood. Hb 44%. Oesophagoscopy showed moderate oesophagitis near the cardia but no ulceration of the oesophagus. At operation on the day of admission a non-adherent indurated area was felt on the lesser curvature 2 in. from the cardia and lying against the front of the hiatus. The hernia was reduced. A barium meal 17 days later did not show a gastric ulcer. The patient has had no pain for 18 months. The anaemia is cured.

Case 4.—A woman of 70 had for many years complained of severe pain in the epigastrium and along the right costal margin. It occasionally radiated to her back. Her gallbladder had been removed without relief of symptoms. Hb 54%. A barium swallow showed a hiatal hernia but did not demonstrate a gastric ulcer. Oesophagoscopy showed no evidence of oesophageal ulceration. At operation on Oct. 23, 1957, the anterior border of the hiatus was felt as a firm, fibrous crescent. On the lesser curvature of the stomach, adjacent to the front of the hiatus and about 1½ in. from the cardia, there was an indurated area—more obvious on palpation than on inspection. Haemorrhagic stippling appeared on its peritoneal surface when this was touched. There were no adhesions. The hernia was reduced. A barium meal 15 days later did not show a gastric
ulcer. The patient has had continued relief from her pain.

Case 5.—
A woman of 64 was admitted because of severe upper abdominal pain of a year's duration. 2 days after admission her pain increased, and radiography demonstrated gas under both domes of the diaphragm. The patient was known to have a hiatal hernia. No oesophageal ulcer was seen at oesophagoscopy. At operation (March 1, 1955) a para-oesophageal hiatal hernia was found: it contained stomach and greater omentum and there was some torsion of the stomach. At one point the greater curvature lay opposite the anterior margin of the hiatus, and here there was a perforated gastric ulcer of the greater curvature, adherent to (but not penetrating) the neck of the sac. The front of the hiatus felt hard, and consisted of compressed, indurated muscle tissue and firm central tendon. The hernia was reduced without difficulty and the perforation closed. The patient died suddenly of pulmonary embolism 14 days after operation. At necropsy, when the stomach was opened, the site of the ulcer could not be seen with the naked eye.

Discussion

Diagnosis.—After a long history of pain, indigestion, and heartburn, the patient develops more severe pain in the epigastrium or along either costal margin, sometimes going through to the back. The pain is inadequately
relieved by alkalis and spontaneous remissions are infrequent. Haemorrhage from the stomach may cause profound anaemia.

A gastric ulcer may be demonstrated by radiography, but its anterior position may make this difficult.

**Operation and its effects.**—The ulcer is commonly on the lesser curvature 1–2 in. from the cardia and the peritoneum over it is often not adherent at the neck of the hernia. During manipulation of the hernia, its contents are reduced into the abdomen. The gastric ulcer then lies some distance below the level of the hiatus. Its presence may easily be overlooked if attention is concentrated exclusively on repair of the hernia. The lesser curvature should be carefully palpated, because the ulcer is often discovered more readily by palpation than by inspection.

After reduction of the hernia the ulcer heals rapidly.

**Anatomy of the esophageal hiatus.**—With a finger in the hiatus, one feels that, whereas its lateral and posterior margins are formed by a soft rolled edge of muscle, its anterior border is relatively sharp and firm. The central tendon of the diaphragm is in close proximity to the front of the hiatus and gives this margin its unyielding character. In the presence of a hiatal hernia, the muscle
tissue at the front of the hiatus tends to be reduced, or compressed into a firm band at the back of the central tendon.

The most traumatic part of a hernial orifice is generally held to be its sharpest and firmest margin. An important cause of trauma to the hernial contents at the neck of a femoral hernia is the lacunar ligament originally described by Gimbert. In hiatal hernia the firm anterior margin of the hiatus seems to play an equally traumatic part.

The portion of the central tendon lying immediately in front of the oesophageal hiatus is, moreover, its strongest part. As described by Blair (1923), the central zone of the diaphragm "is found to be occupied by four well marked diagonal bands radiating outwards from a central point of decussation like the bars of a St. Andrew's cross. This central point appears as a thick node of compressed tendinous strands situated in front of the oesophageal aperture" (fig. 4). There is therefore no possibility of enlargement of the hiatus in an anterior direction at the expense of the central tendon. To relate the anatomy of this hernial orifice to the more familiar external inguinal ring, the arrangement is reminiscent of the inter-crural fibres which are described by Scarpa as "opposing further divergence of the tendinous pillars".

A portion of the lesser curvature about 1–2 in. from the cardia usually lies against the firm anterior margin of the hiatus, and it is at this point that the gastric ulcer commonly develops. In case 5, where the stomach had rolled upwards and undergone some degree of torsion, the greater curvature was at one point adjacent to the front of the hiatus; and here an ulcer had developed. It is suggested, therefore, that this type of gastric ulcer arises, not in relation to a particular part of the stomach, but on whatever part lies against the anterior margin of the hiatus.

The cause of the ulcer appears to be trauma. This is suggested by its exact localisation to that portion of the stomach lying against the firm anterior border of the hiatus, and by the fact that it heals and remains healed on reduction of the hernia.

These cases show that gastric resection is not necessary in the treatment of this type of ulcer. This opinion is based on experience with non-penetrating ulcers. If the ulcer is penetrating, resection of part of the stomach might be necessary (Barrett 1954); but, so long as the hernia can be satisfactorily reduced, surgery should presumably be as conservative as possible.
Summary

Five cases are described in which a gastric ulcer was associated with a hiatal hernia.

The ulcer developed where the stomach was in contact with the anterior margin of the oesophageal hiatus, and was apparently caused by trauma from this structure.

The presence of such an ulcer may easily be overlooked, even on radiography.

In these five cases the ulcer was cured without resection of the stomach. It healed as soon as the hernia was reduced.

REFERENCES

Allison, P. R., Johnstone, A. S. (1953) Thorax, 8, 87.
— (1954) ibid. 42, 231.
ULCERATION OF THE ESOPHAGUS IN ASSOCIATION WITH A TUMOR OF THE PANCREAS

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Reprinted from THE JOURNAL OF THORACIC AND CARDIOVASCULAR SURGERY
St. Louis

Vol. 48, No. 2, Pages 200-204, August, 1964

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(Printed in the U. S. A.)
ULCERATION OF THE ESOPHAGUS IN ASSOCIATION WITH A TUMOR OF THE PANCREAS

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Although a number of patients with the Zollinger-Ellison syndrome have been successfully treated by excision of the tumor, Zollinger and Craig recommend total gastrectomy for arrest of the ulcerative process. By removing the acid-producing mucosa, total gastrectomy prevents further ulceration in the stomach, duodenum, or upper jejunum. In the case of Zollinger-Ellison syndrome to be described, an ulcer developed in the esophagus several years after total gastrectomy.

CASE REPORT

The patient, a man 54 years of age, was seen elsewhere in 1950. He had a long history of dyspepsia. An ulcer of the first part of the duodenum was demonstrated radiologically. Hyperchlorhydria was present. Perforation of the duodenal ulcer occurred in 1951. In 1952, a partial gastrectomy of Polya type was carried out for a chronic duodenal ulcer penetrating the pancreas. One third of the stomach was removed. An x-ray study in 1954 showed a large sternal ulcer. A higher partial gastrectomy with a Roux-en-Y gastrojejunal anastomosis was performed in 1955. In 1956 a gastrojejuno-ileo fistula was closed at a further operation.

In January, 1957, the patient was transferred to the Bradford Royal Infirmary. He was weak and emaciated. He complained of intense and persistent pain over the left costal margin, over the precordium, in the left shoulder, and in the left side of the neck. A barium meal showed a very large ulcer, the base of which, on the left side, appeared to be lying against the inner aspect of the chest wall. The radiologist noted the presence of free gastro-esophageal reflux and suspected the presence of a small hiatal hernia.

In February, 1957, a total gastrectomy was performed. There was a huge gastric ulcer penetrating the diaphragm. A jejunal loop was brought up in the manner of Roux and anastomosed to the lower end of the esophagus. A T jejunojejunal anastomosis was made below the transverse mesocolon. It was difficult to determine whether or not there was a small hiatal hernia as the tissues in the region of the hiatus were grossly edematous. Histological section showed the gastric ulcer to be simple and chronic.

Postoperative Course.—The patient resumed his work one year after operation and remained at work till shortly before his death 6 years later. After an interval of 3 years he developed a megaloblastic anemia which was controlled by vitamin B₁₂ injections.

Two and a half years after operation, the patient had one episode of dysphagia. A barium swallow showed no lesion in the esophagus and no delay in the passage of the barium from the esophagus into the jejunum. Four and a half years after total gastrectomy, the dysphagia recurred and a barium swallow revealed a stricture of the esophagus at the level of the aortic arch (Fig. 1).

Received for publication March 4, 1964.

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At esophagoscopy, the upper part of the esophagus was found to be intensely inflamed, with linear superficial erosions of the squamous mucosa. At 26 cm. from the incisor teeth, there was a tight stricture. An attempt was made to obtain biopsies from below the level of the stricture. However, no mucosa was found in two biopsies which consisted of necrotic material with acute inflammation.

The patient complained of regurgitation of fluid which he described as acid and which burned his throat.

During the next year, esophagoscopy and dilatation of the stricture were carried out three times with, on each occasion, temporary relief of the dysphagia. The patient continued to complain of “acid” regurgitation.

The patient was re-admitted to hospital in November, 1962. His condition had begun to deteriorate one month previously when he developed diarrhea, abdominal pain, and swelling of both ankles. On admission, he complained of epigastric pain and of heartburn when lying down. The diarrhea persisted. An additional feature was that he felt very thirsty and was found to have diabetes. The liver was enlarged, hard, and irregular, and there was some ascites. No malignant cells were found in the ascitic fluid but a needle biopsy of the liver showed the presence of an adenocarcinoma. Esophageal juice was aspirated but no free hydrochloric acid or bile was found in the specimen obtained. The patient died on Jan. 6, 1963.
AUTOPSY FINDINGS

The relevant findings related to the esophagus and the pancreas.

Pancreas.—There was a rounded tumor in the body of the pancreas, about 5 cm. in diameter. Histological section of the tumor showed it to be an islet cell carcinoma of trabecular or ribbon pattern. There was moderate cellular pleomorphism but relatively few mitoses.

The head of the pancreas was normal in appearance.

Liver.—The liver contained many large metastases which were seen histologically to be metastases of islet cell carcinoma of similar structure to the pancreatic tumor.

No abnormality was found in the parathyroid, adrenal, or pituitary glands.

Esophagus.—(Figs. 2 and 3). There was ulceration and narrowing of the esophagus at the level of the aortic arch. At this point, there was some thickening of the esophageal wall. Above the ulcer, the esophagus was macroscopically normal and was shown histologically to be lined by normal squamous epithelium.
Formalin had been introduced into the esophagus by tube immediately after death in order to preserve its lining. Section through the ulcer showed chronic ulceration. The epithelium was replaced by a surface of chronic granulation tissue below which was dense fibrosis.

Below the ulcer, the esophageal lining was of different macroscopic appearance from that above the ulcer. Histological sections showed the esophagus below the ulcer to be lined by an intact columnar epithelium (Fig. 4). This mucous membrane closely resembled flattened small intestinal mucosa and showed many goblet cells and numerous Paneth cells at the bases of the tubular glands. No oxyntic cells were seen.

At the esophagojejunual anastomosis, the flattened glandular mucosa lining the esophagus below the ulcer was in continuity with typical villous small intestinal mucosa.

**DISCUSSION**

Presumably the esophageal ulcer and stricture were due either to acid-pepsin or to alkaline digestion of the squamous mucosa.

The gastrectomy was complete as shown by histological sections through the esophagojejunal anastomosis. There was, therefore, no gastric mucosa to produce acid. The lower esophagus was lined by columnar epithelium. Columnar epithelium in the esophagus in association with a hiatal hernia, often referred to as “gastric lined oesophagus,” is usually considered not to produce digestive juices. The question arises as to whether or not this epithelium, under the in-
fluence of an ulcerogenic pancreatic tumor, was capable of producing acid. This seems unlikely. No oxyntic cells were found in it. Also, the mucosa was not gastric in type but bore a close resemblance to small intestinal mucosa. However, as stressed by Planteydt and Willighagen, intestinal metaplasia of gastric mucosa is common, so that the possibility of the presence of oxyntic cells at an earlier stage cannot be dismissed.

The esophageal ulcer may have been due to alkaline reflux. If, as seems probable, columnar epithelium resistant to digestion was already present in the lower esophagus at the time of total gastrectomy, an ulcer due to reflux of bile and pancreatic juice would occur at its upper limit. Bingham, McKeown, and Cox have all described patients in whom esophageal stricture occurred after partial gastrectomy and they postulated that the lesions were due to reflux of bile and pancreatic juice. This may be the simple explanation for the stricture in our patient, in which case no relationship between the esophageal ulcer and the pancreatic tumor need be assumed. Esophagitis may occur after total gastrectomy, especially if a jejunal loop is brought up in continuity. However, it is uncommon when a Roux loop is used. Such esophagitis and stricture formation occur early and are in the proximity of the esophagojejunostomy. In our patient the stricture took four and a half years to develop and was situated at a considerable distance above the anastomosis.

The stricture of the esophagus was, therefore, quite unusual, and its development in association with an ulcerogenic tumor of the pancreas tempts one to assume a relationship between the two lesions. The findings suggest that, before carrying out total gastrectomy for a Zollinger-Ellison syndrome, it may be wise to make sure that the esophagus has a normal epithelial lining by taking biopsies from different levels in the esophagus. If columnar epithelium is found, it may be worth considering planning the operation so that the esophageal anastomosis is made above the upper limit of the abnormal mucosa.

The occurrence of an esophageal ulcer in this patient suggests that it is necessary to keep in mind the possibility of an ulcerogenic tumor of the pancreas in the presence of bizarre ulceration of the esophagus as well as in the presence of ulceration of the stomach, duodenum, and upper jejunum.

**SUMMARY**

A case is described in which a stricture of the esophagus at the level of the aortic arch occurred 4½ years after total gastrectomy for a Zollinger-Ellison syndrome. The esophagus below the stricture was lined by columnar epithelium.

**REFERENCES**

Hiatal hernia

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Inguinal hernia, so beautifully illustrated and animatedly discussed almost 200 years ago, still has a very definite recurrence rate after repair. In the repair of both inguinal and hiatal hernia room has to be left for the passage of an anatomical structure, the spermatic cord and the oesophagus respectively, through a muscular opening without subjecting that structure to compression. After repair of the hernia an opening remains which is a potential source of recurrence. In hiatal hernia the problem is more complicated in that we are dealing not only with the protrusion of an abdominal viscus but with a very delicate and only partially understood valvular mechanism at the oesophago-gastric junction with a boiling cauldron of acid on one side of it and a vulnerable mucosa on the other. Surgery is eminently successful in the relief of obstructions whether they be in bowel, in the biliary or urinary tract, or in a heart valve. It is far less successful in the reconstruction of one-way valvular mechanisms where co-ordinated muscle action plays such an important part. I make this preliminary comment in order to explain why any surgeon claiming 100% success in the treatment of hiatal hernia is a prevaricator and any physician expecting him to achieve such success is an uncritical optimist.

Having said this, I must hasten to add that, in my opinion, the results of repair of uncomplicated hiatal hernia are good. The question immediately arises as to what criteria are to be used to judge results. In the case of inguinal hernia success or failure may be decided merely by asking the patient to cough. In hiatal hernia, one has to assess results on symptoms or post-operative radiological findings or both. Herein lies a difficulty. It is well recognized that, although clinical success may be achieved in 80-90% of patients, the radiological cure-rate is much lower. Pearson & Gray (1967) reported 83% clinical successes as against radiological success in only 40%. Raphael et al. (1965) said that although most reports indicate that the majority of patients experience symptomatic improvement after operation, rates of recurrence based on roentgenographic evidence vary from 1 to 48%. Although this discrepancy between symptomatic and radiological results is disconcerting, I think that, for practical purposes, it is the patient's assessment of the operation which is the important thing. In the case of an internal hernia, her heart does not grieve for what her eye does not see.

The results of hiatal hernia repair depend to a large extent on case selection. Rex et al. (1961) claimed that in a 10-year study of medically treated patients, 82% with minimal symptoms improved on medical therapy alone. The very best surgical results are to be obtained by operating on patients who do not really require surgery. From what I have read in the literature, there is a tendency to describe the results of surgery in hiatal hernia while failing to detail the types of hernia operated on or to mention, almost as an aside, that complicated cases associated with stricture have been excluded from the series. I think that the results of surgery must be subdivided into those obtained in uncomplicated hernias and those obtained in the presence of the various complications of hiatal hernia, especially peptic stricture, which adversely affect the rate of cure.

The incidence of hiatal hernia in the general population is unknown. All reported series have undergone some selection. I have chosen a highly selected group of patients, namely those I have operated on in the last 3 years. In spite of the difficulties inherent in attempting to draw conclusions from such a series, I have felt that you would prefer opinions based on actual case records rather than vague impressions as seen through a surgeon's rose-coloured spectacles.

The series consists of 117 patients. Of these, eighty-one may be classified as uncomplicated hernias. By this I mean reducible hernias either sliding or para-oesophageal, the sliding hernias being usually associated with oesophagitis but without evidence of stricture at oesophagoscopy. Thirty-six patients are classified as complicated.

Complications

No less than twenty-nine had peptic stricture of the oesophagus. Two presented as strangulated
hernia, two were associated with an oesophageal web, two with a penetrating ulcer at the level of the hiatus, and in one there was an achalasia of the cardia in addition to the hiatal hernia.

About peptic stricture I shall say more later but at this stage I would like to say a word about some of the other complications.

(1) Of the strangulated hernias, one, a man of 58, came to hospital late and died of pre-renal uraemia. One patient who presented with pain and vomiting was thought from the radiological appearances to have a strangulated hiatal hernia. There was indeed a hernia but the symptoms and X-ray appearances were produced by pyloric stenosis and eventration of the left dome of the diaphragm.

(2) The association of upper oesophageal web with hiatal hernia was described by Smiley, McDowell & Costello (1963). Such webs are easy to miss radiologically and, without oesophagoscopy, the dysphagia may be attributed to the hernia. In one patient there were two oesophageal webs, an upper and a lower. The upper one was split through the oesophagoscope and the lower one was excised at thoracotomy. Incidentally, lower webs are easy to miss even at oesophagoscopy. They are extremely mobile and move down in front of the oesophagoscope. At thoracotomy, they may be difficult or impossible to feel. On one occasion, when operating on a lower web and being unable to feel it, I had the anaesthetist pass a large stomach tube. The tube appeared to pass well down into the stomach and, at first, I thought the diagnosis was wrong. On closer inspection, it was found that the tube had pushed the web down ahead of it. I have seen exactly the same thing in a more recent case. I have never seen this happen with a peptic stricture so that I tend to support the view that, though such webs are usually associated with a hiatal hernia, they are not peptic strictures. The web is readily removed by submucosal dissection.

(3) In the patient with a hernia and achalasia of the cardia, all the symptoms are relieved by Heller's operation and repair of the hernia (Fig. 1).

The figure of thirty-six complicated hernias does not include those associated with duodenal ulcer, disease of the biliary tract, or diverticulitis regarding which my figures are imprecise. However, I hope to have shown how frequently complications exist which must inevitably affect the results of surgery.

Indications for surgery

The high incidence of complicated cases in this series may be a reflection of a certain restraint which has been exercised in the selection of patients for surgery and this brings me to the question of case selection.

![Fig. 1. Achalasia of the cardia associated with hiatal hernia.](image)
stomach is at the level of the cardia. Occasionally, I think it is worth while trying to get the patient to sleep face down (Fig. 3). An offender in increasing reflux is the Goldthwaite belt for sore back. Sometimes the belt has become a habit rather than a necessity and it may be discarded with benefit.

(2) Secondly, I have taken as an indication for surgery the persistence of heartburn with, in addition, dysphagia due to acute oesophagitis.

![Fig. 2. Lateral X-ray with the patient lying on her back showing the height of the barium column in the stomach above the level of the cardia.](image)

![Fig. 3. Lateral X-ray with the patient lying face down showing the oesophagus lying above the barium in the stomach.](image)
as seen at oesophagoscopy but without stricture formation. There were twelve such patients. Rightly or wrongly, it was felt that operation in such patients might prevent progressive changes in the deeper layers of the oesophageal wall leading to peptic stricture.

This raises the problem of when repair of a hernia should be advised as a prophylactic measure against peptic stricture. In practice, the patient with a peptic stricture usually presents as such. Only occasionally does one witness by serial X-rays the development of a stricture in an initially uncomplicated hernia. I think, however, that acute oesophagitis together with discomfort on swallowing is an indication for surgery and also I think that a baby with a hernia or gastro-oesophageal reflux who regurgitates blood-stained fluid should be operated on.

(3) Thirdly, strangulation is an obvious indication for surgery. A history of episodes of pain often relieved by vomiting in the presence of a large hernia is, I think, an indication to operate in order to avert strangulation. The most dangerous type of hernia in this respect is the one which contains about half the stomach and in which a persistent fluid level is seen on the straight X-ray of chest, especially the lateral view. Paradoxically, when all the stomach is in the chest, the risk of strangulation, though present, seems to be less, presumably due to the fact that the portions of bowel entering and leaving the hiatus are less bulky.

(4) Another lesion demanding surgery is the peptic ulcer which goes on penetrating remorselessly and may involve the pericardium or the aorta. Such ulcers may be at or near the squamo-columnar mucosal junction, on the lesser curvature at the level of the hiatus, in the herniated portion of stomach or in a gastric-lined oesophagus. In this series there were two patients with an ulcer at hiatal level (Fig. 4). These ulcers often bleed. They are of interest as I believe that the only treatment they require is repair of the hernia which moves the ulcer away from the margins of the hiatus (Davidson, 1958).

(5) A fifth indication for surgery is bleeding. Haematemesis may be a sufficient indication on its own. Anaemia presumed to be secondary to a hernia may respond quite well to treatment with iron but recurrent anaemia is often a factor influencing the decision to operate.

(6) Lastly, in this series, peptic stricture of the oesophagus was a frequent indication for surgery.

To recapitulate, the indications for surgery were failure of medical treatment, acute oesophagitis and dysphagia without actual stricture formation, strangulation or a history of episodes of pain with a large hernia, penetrating ulcer, haematemesis and sometimes recurrent anaemia, and peptic stricture of the oesophagus.

Peptic stricture

This is a large problem. As I have said, there were twenty-nine strictures in this series. This figure does not include all the peptic strictures I have seen in the past 3 years as, because of my approach to the management of this lesion, a number did not come to surgery. I am afraid that I am in a somewhat bemused state concerning the correct treatment of this lesion. Perhaps I may illustrate my difficulties by describing some types of stricture dealt with. Peptic strictures seem to me to fall into three groups from the clinical point of view.

(1) Firstly, there is the stricture which, at oesophagoscopy looks inflamed and oedematous and feels relatively soft and mobile. This is the type of stricture which, theoretically, should respond well to control of reflux by repair of the hernia. In practice, the behaviour of such strictures is somewhat unpredictable. In this series, there are instances of at least three types of response.

(a) Patients in whom successful repair of the hernia was achieved with disappearance of the stricture.

(b) Patients in whom the hernia recurred soon after operation but in whom the dysphagia was nevertheless relieved (Fig. 5).
(c) Cases where neither successful repair of the hernia nor relief of the dysphagia was achieved (Fig. 6).

2) A second type is the high stricture with a segment of columnar-lined oesophagus above the hernia. There were at least seven such patients. They again respond in different ways to hernial repair. Some were cured. Others were converted from a long inflamed stricture to smooth, rather web-like strictures which, in contra-distinction to their pre-operative state, were much more amenable to dilatation (Fig. 7). Some remained as tight as ever.

I am obviously unable to distinguish between strictures which are going to respond to repair of the hernia and those which are not.

3) Then there is a group of very hard strictures which neither respond well to dilatation nor disappear after attempted repair of the hernia. Three such strictures in this series developed after an operation for duodenal ulcer. This type of stricture is, I think, a very definite entity. It is possibly related to the stricture which may follow soon after delivery especially in hyperemesis gravidarum as described by Abbey Smith & Nelson (1965). Dysphagia usually comes on about 3 weeks after an operation, often an operation for duodenal ulcer but also after a variety of other procedures. A hiatal hernia has not necessarily been present before the operation. My own view is that this stricture is due to an acute acid burn of the oesophagus during recumbency under anaesthesia. Just as it is recommended that alkali be given by mouth before anaesthesia for labour in order to prevent Mendelson’s syndrome, so, I think, we may come round to the policy of giving alkali immediately before operation on patients known to have a hiatal hernia or who have a peptic ulcer or hyperchlorhydria. The following case record is typical of this type of stricture:

Perforation of a chronic duodenal ulcer occurred in a man of 32. The perforation was closed. Three weeks later he developed dysphagia which became severe and led to emaciation and dehydration. The barium swallow showed the tapering column of barium characteristic of this
striction. The patient has done well for 2 years on self-bouginage. It is of interest—that, although no hiatal hernia was seen at several previous X-rays over a period of some years, there is now a hernia. It is debatable whether a hernia played a part in the occurrence of stricture or whether it was, in fact, secondary to severe changes in the oesophageal wall produced by ulceration and oesophagitis.

Of the twenty-nine strictures, relief of dysphagia by reduction of the hernia was achieved in eighteen, improvement in five, and in six the stricture remained as tight as before. In view of these figures, I think that what has been called a ‘hopeful’ repair of the hiatal hernia associated with stricture is often justified especially as there is a great difference in the severity of the operation between simple reduction of the hernia and excision of the stricture with colon or jejunal replacement.

My own attitude towards peptic stricture is as follows but this is a strictly personal view. I try to reduce the hernia if at all possible. There is no doubt that in a number of patients where the X-ray appearances and the findings at oesophagoscopy seen ominous, the hernia may be easier to reduce than one might expect. This is particularly so when there is a high stricture with a gastric-lined oesophagus when the actual stricture may be easy to reduce. I have not practised Collis’ operation to any extent. If the hernia seems quite irreducible or the patient is unfit or very old, I try to manage the case by dilatation before considering excision and replacement with colon or jejunum. Dilatation via the oesophagoscope gets rather a bad press. I would emphasize that, if repeated dilatation is going to succeed, there must be a close liaison between the patient and the surgeon. It is no use dilating the stricture and sending the patient away to go through the laborious business of seeking a further appointment through the usual channels. The patient must be able to ring up and arrange for dilatation within a day or two of recurrence of dysphagia. Also, with a co-operative patient, I am sure that self-bouginage has a very definite place.

Sometimes there is no alternative to excision of the stricture but, in practice, I do few such operations for this lesion. It is indicated, I think, for a very hard stricture not responding to dilatation or where there is a penetrating ulcer which is progressive. A peptic stricture in childhood which does not respond to repair of the hernia and where the child fails to thrive and remains seriously underweight is, I think, an indication for excision of the stricture and colon replacement.

Peptic stricture of the oesophagus may undoubtedly undergo malignant change. One patient of mine had had a peptic stricture since childhood and developed a carcinoma at the site of stenosis at the age of 42. How much one should be influenced in treatment by the possibility of malignant change I do not know.

Anaemia

In this series there were sixteen patients with significant anaemia. Six of these had haematemeisis. In one patient, although there was a hernia, the anaemia was due to bleeding from a
leiomyoma of the stomach. In two patients bleeding occurred from an ulcer at the level of the hiatus. In nine patients in whom the anaemia was presumed to be related to the hernia, the hernia was invariably a fairly large one and its repair resulted in cure of the anaemia in seven.

**Results**

In the hernias I have classified as uncomplicated 77-5% were asymptomatic after hernial repair, 15% were improved, and 7-5% were no better. When there were complications, especially stricture, only 61% were asymptomatic, 25% were improved, and as many as 14% were no better.

The main reasons for poor results were persistence of reflux or recurrence of the hernia, and persistence of dysphagia. Results were poor in a group not always mentioned but which I would describe as instances of wrong diagnosis or ill-advised surgery. It is only too easy to blame symptoms on a blameless hernia. When, after operation, some patients, as in this series, express dissatisfaction because of buzzing in the ears, nightmares, numbness round the ankles and black dust in the water, one realizes that one should never have operated on them at all.

**Discussion**

I have purposely avoided discussion of surgical technique. My own preference is for the trans-thoracic or thoraco-abdominal approach as I find it difficult to know how otherwise one can cope with the considerable number of patients with stricture and peri-oesophagitis. However, many believe otherwise and discussion of this matter is singularly unrewarding. I must admit that post-operative wound pain, which may on occasion, be severe and persistent, is a disadvantage of the thoracic approach. There is no time to describe my own not altogether successful efforts to solve this particular problem.

I cannot understand those who would undertake the surgical treatment of hiatal hernia without routine oesophagoscopy. Even the best radiologist cannot reveal all. At oesophagoscopy, one can see the site of the oesophago-gastric junction, get an idea of the function of the cardia and certainly see the moderate and severe degrees of oesophagitis with superficial erosions of the squamous mucosa. One can find a gastricleined oesophagus not apparent on X-ray. One can determine the level of the stricture and if it is hard or soft, fixed or mobile. One can take a biopsy to exclude neoplasm. Inevitably one will discover from time to time associated lesions such as webs which have been missed on X-ray.

In spite of all I have said concerning case selection, I am sure that in many instances we know not what we do. The oesophagus is a very emotional organ. In no surgery that I practise do psychological factors play such a big part. The fat woman of 55 who has, as they say, 'let herself go', who no longer feels needed by her grown-up family and her disinterested husband, may seek comfort from the priest, the psychiatrist, the beautician, or the surgeon. Equally baffling is the woman with the doting husband who does all the chores. We claim cure of heartburn by repair of a hernia when all we may have done is to provide circumstances in which a woman who feels neglected may again become an object of sympathy. We accept blame for failures when the symptoms are in fact related to business worries or domestic disharmony. The dermatologist must be a psychologist and so must the oesophageal surgeon. It is very difficult to know when an oesophagus is being digested by acid and when it is blushing for shame, pining for love, quivering with anxiety, desolate with grief, or merely reflecting downward trends on the stockmarket.

I hope that I have succeeded in emphasizing the complexity of the problem, at least as far as I am concerned, while at the same time showing that the results of surgery in properly selected patients are good.

**References**


Postoperative Stricture of the Oesophagus

This paper concerns the stricture of the oesophagus which may develop within 2 or 3 weeks of an operation that has not directly interfered with the cardia or the oesophageal hiatus of the diaphragm. Fortunately the lesion is not common, but papers describing a limited number of cases have appeared in the literature. The knowledge that the incidence of the stricture is low is no consolation for the unfortunate patient in whom it occurs. The stricture is often intractable and may remain a problem for the rest of the patient's life.

I shall describe 7 patients. The first will be described in detail as showing most of the features characteristic of this stricture. I shall also comment on certain facts which seem to indicate the time at which the damage giving rise to the stricture is done.

Case 1

A man of 43 with a long history of duodenal ulcer was admitted to hospital with a perforation. This was closed by suture and omental graft. A Ryle's tube was passed before operation and left in the stomach for 2 days after the operation. After its removal, the patient vomited persistently. The tube was reinserted and kept down for 4 more days. At this stage, the patient said his gullet felt hot and raw. A gastrografin swallow 8 days after operation showed no delay in the emptying of the stomach. About 3 weeks after operation, he began to have difficulty in swallowing. The dysphagia was progressive until he had great difficulty in swallowing even liquids. There was some delay in the investigation of the dysphagia. A barium swallow 3 months after operation (Fig. 1) showed a complete hold-up of barium. The stricture was a tight one and the tapering appearance of the column of barium suggested extensive changes in the oesophageal wall.

The patient was admitted to the Thoracic Surgical Department in an emaciated and dehydrated condition. At esophagoscopy, the lumen was seen to narrow in a funnel-shaped manner. The instrument could be passed to 37 cm. from the incisor teeth. Beyond this, the narrowing continued down to a tight stricture which could not be approached by the esophagoscope. It was not possible to pass a bougie through the stricture. The patient was maintained on intravenous fluids. A few days later esophagoscopy was repeated and bougies were passed successfully. After this the patient improved dramatically; he ate voraciously during the day and raided the refrigerator during the night. In 11 days he gained 22 lb. in weight, a good deal of which consisted of oedema fluid in his legs.

Two further dilations were done through the esophagoscope and the patient was then taught self-bougination. A barium swallow was given 2 months later (Fig. 2). This showed a hiatal hernia.

The patient did well on self-bougination and reached his normal weight in 3 months. He has returned to work as a commercial traveller. He carries his bougie in a plastic sheath in his brief case out of which it projects for some distance. Recently, while in Glasgow, he was stopped and questioned by the police after a shooting affair: they thought the bougie might be some kind of firearm. The patient described with relish the look on their faces when he took out the supposed weapon and swallowed it!

Comments on Case 1

In the common kind of peptic stricture of the oesophagus associated with hiatal hernia, a complete hold-up of barium on the barium swallow film is unusual. This X-ray appearance seems to be more common in the type of stricture we are describing (see also Case 5).

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Severe weight loss is characteristic of the postoperative stricture. In the common type of peptic stricture, on the other hand, very many patients maintain their weight in spite of troublesome dysphagia.

In this patient, the findings at oesophagoscopy were interesting in that they were reminiscent of the state of affairs sometimes found with corrosive stricture. A barium swallow 5 months after the operation showed a hiatal hernia. A hernia had not been seen on previous films. It is difficult to know whether the hernia was present before the operation or was the result of changes initiated at or after the operation. In many cases described in the literature, a hernia was discovered only after the onset of the dysphagia. In some cases there had been no previous films; in others, previous X-ray reports did not disclose a hernia, and whether or not a hernia had been present before the formation of the stricture remained uncertain.

In this patient, a man with a long history of duodenal ulcer and probably hyperchlorhydria, it is easy to imagine...
the manner in which the peptic stricture occurred as a result of gastro-oesophageal reflux. In the following group of 3 patients, the explanation is less obvious: the stricture developed soon after operations designed to suppress the gastric acid.

Case 3

This patient, a woman of 66, developed dysphagia after discharge from hospital following a two-thirds Polya gastrectomy for oesophagus lined by columnar epithelium. Reduction in the size of the hiatus resulted in almost complete relief from dysphagia.

Case 2

A man of 67 began to have difficulty in swallowing nearly 3 weeks after a three-quarters Polya gastrectomy. He was operated on in the belief that there was a hiatal hernia, the repair of which might result in improvement in the stricture. At operation, the presence of a hernia seemed doubtful. The hiatus was lax, and the stricture lay a short distance above the oesophagogastric junction, apparently at the upper limit of a segment of duodenal ulcer. There had been distressing vomiting for a few days after operation. The dysphagia was persistent and progressive. A barium swallow 18 months later (Fig. 3) showed a tight stricture at the oesophago-gastric junction. Dilation of the stricture through the oesophagoscope proved difficult and ineffective. At operation, there was no hiatal hernia. The oesophagus was difficult to mobilise from the mediastinum. Immediately above the diaphragm there was a peculiar hard hour-glass constriction of the oesophagus.

Fig. 3
The muscle layers at this point had almost disappeared and their remnants were firmly adherent to the submucosa. The findings suggested a previous destructive process involving all coats of the oesophagus. An attempt at excision of this stricture has not been successful. Dilation of the residual stricture via the oesophagoscope is now easy but is required at intervals of about 6 months.

Case 4

A man of 68 complained of dysphagia soon after partial gastrectomy for duodenal ulcer. He had been known to have a hiatal hernia but had not previously suffered from dysphagia. The barium swallow (Fig. 4) showed an irregular stricture and a hiatal hernia. The stricture was at a distance of 36 cm. from the incisor teeth. It allowed easy passage of bougies. An attempt to relieve the dysphagia by repair of the hernia has not been successful. Dilation of the stricture is required at intervals averaging 9 months.

Of the 3 patients, postoperative vomiting occurred in one, and a hiatal hernia was known to exist in another. The three were probably intubated but intubation, though probably an important assault on the patient, often receives scant mention in the case records.

Case 5

A man of 83 underwent gastroenterostomy without vagotomy for duodenal ulcer and pyloric stenosis. He was readmitted 2 months after operation because of dysphagia which had been present for some time; he was having great difficulty in swallowing even liquids. A barium swallow showed a complete obstruction to the passage of barium. Although previous X-rays had not revealed a hernia the later films suggested that there might now be a hiatal hernia.

Case 6

The patient, a man of 75, had a previous history of perforated duodenal ulcer but without previous dysphagia. He was not known to have had a hiatal hernia. He
JAMES S. DAVIDSON

developed a very hard, tight stricture soon after prostatectomy. The X-ray (Fig. 5) shows the stricture and a hiatal hernia.

Case 7

The patient, a man of 61, had a laparotomy for a suspected carcinoma of the stomach. On X-ray he had a cup and spill type of stomach due to an unexplained elevation of the left dome of the diaphragm. Laparotomy was negative. The surgeon mentioned the fact that he did not find a hiatal hernia. Two and a half weeks after operation the patient developed dysphagia and quickly became emaciated. X-ray (Fig. 6) showed a stricture and a hernia. During the past 5 years he has maintained a good weight on self-bougination.

Discussion

In seeking a cause of the stricture one is up against the same obscurities which cloud the aetiology of gastric and duodenal ulcer. It is not known why some patients can tolerate oesophageal reflux for long periods without sustaining oesophageal damage while others may rapidly and quite suddenly form an oesophageal stricture.

We have to assume that a major factor in the stricture formation must be gastro-oesophageal reflux.

One would hope to find in the case records a common factor which would explain this postoperative stricture. Various things have been incriminated, for instance the existence of an unrecognised hiatal hernia or a segment of "gastric-lined" oesophagus, postoperative vomiting, or intubation. Not one of these possible causes is present in every case. Indeed, the only common factor is operation and recumbency under general anaesthesia.
I have described 7 patients of whom only one was female. In 5, the operation was for duodenal ulcer. One patient, though operated on for prostatic enlargement, also suffered from duodenal ulcer. Two striking features are, therefore, the association with duodenal ulcer and the predominance in males: this accords with other series in the British literature. The total number of patients described in separate papers by Bingham (1958), Cox (1961), Douglas (1956), Hurst (1961), and McKeown (1958) is 16, of whom 13 were men. 14 suffered from a peptic ulcer which was, almost without exception, duodenal and with a high incidence of perforation and pyloric obstruction. It seems that this rapidly developing postoperative stricture is most likely to occur in a male after an operation for duodenal ulcer. This suggests that it is due to acid-pepsin digestion of the oesophagus in a person susceptible to peptic ulceration. The patient need not have been intubated. He need not have had a hiatal hernia or postoperative vomiting.

It is of interest and of some importance to know when the damage which gives rise to the stricture is inflicted. I think there are two clues, viz. the time of onset of the dysphagia, and its occurrence after operations designed to reduce the gastric acidity.

The time of onset

In this series, dysphagia began soon after discharge from hospital—on an average, 3 weeks after operation. This pattern is repeated in the literature. Thus of Bingham's 4 cases, the first had dysphagia in 3 weeks, the second was discharged in April and had X-ray evidence of a stricture by June, the third was discharged after 12 days and had dysphagia very soon after, and the fourth developed dysphagia in 3 weeks. McKeown's 2 patients had difficulty in swallowing after 3 weeks. One of Hurst's patients had dysphagia in 2 weeks and the other just after discharge, i.e. in about 3 weeks. In Cox's 3 patients who had a partial gastrectomy, dysphagia began within 20 days. This timing is remarkably constant, whereas other factors are inconstant. It is tempting to relate the relatively constant time of onset to the other constant, viz. the operation, and to conclude that the oesophageal lesion is initiated during the operation.

The nature of the operation

The second clue is the fact that the stricture may develop after a partial gastrectomy which has successfully reduced acid secretion. If, as seems likely from the type of patient in whom it most often occurs, the lesion is due to acid reflux, it is strange that it should form after the acid has been removed. The explanation often given in such circumstances is that the stricture now follows tryptic and alkaline digestion of the oesophageal mucosa. This is something of a switch, for when the lesion follows closely upon closure of a perforation, it is considered to be due to acid reflux but, after partial gastrectomy, an opposite chemical reaction is invoked. Yet, in both circumstances, the time of onset of the dysphagia is the same. It would suggest that after partial gastrectomy the stricture is still the result of an acid burn of the oesophagus, sustained between the induction of the anaesthetic and the resection of the stomach. The situation resembles a corrosive stricture in which all the damage occurs during one relatively brief contact between the corrosive and the mucosa. The pH of the gastric juice in the postoperative period must vary from patient to patient and if the damage were inflicted during the postoperative period, one would expect the stricture to develop at varying
periods of time after the operation, depending on the degree of acidity or alkalinity of the gastric juice. As we have seen, this is not so.

If this view of the timing of the oesophageal damage is correct, certain inferences may be drawn. Nagler and Spiro (1963) have shown that, when a patient is recumbent, introduction of acid into the stomach through a tube lowers the pH of the oesophagus and causes heartburn. This does not happen if the patient is propped up even as little as 10°. An intragastric tube is, therefore, most dangerous during the period of recumbency under anaesthesia and in the immediate postoperative period. As I believe that the stricture is due to an acid burn of the oesophagus inflicted during the early part of the operation, an intragastric tube is potentially dangerous if it is in position during the operation. Passed after the operation, when the acid has been reduced and the patient is propped up, it may well be harmless. Also, if my view is correct, it would be logical to give alkali by mouth to peptic ulcer patients immediately before operation.

Postoperative vomiting occurred in only a minority of the patients and is not the cause of the lesion, though it might be a sequel.

An analogy seems to exist between this stricture and the lesion in pregnancy described by Abbey Smith and Nelson (1965) which follows closely on delivery, especially in hyperemesis gravidarum. Here again the patient presents with a severe, often intractable, stricture, and the onset of dysphagia bears the same time relationship to labour as the postoperative strictures to operation. The patient vomits during pregnancy but has no dysphagia: difficulty in swallowing begins only after delivery. I suggest that the stricture may be the result of an acute burn of the oesophagus during labour, when the patient is recumbent, has marked elevations of intra-abdominal pressure, and is sometimes anaesthetised. The very different circumstances of vomiting during pregnancy and reflux of acid during labour may explain why the burn is post-partum.

In this group of 7 patients, a hiatal hernia was found in 5 during investigation of the postoperative dysphagia. Of them, one patient had been known to have a hernia before operation; the others had been X-rayed before operation but a hernia had not been discovered. In Case 7, in whom a hernia was found associated with the stricture, a hernia had not been discovered previously either radiologically or at operation. The fact that this stricture follows quickly upon operation means that the time of its origin is known fairly accurately. Also, in this group of patients, previous X-rays have usually been taken in the course of investigation of the duodenal ulcer. In the common type of peptic stricture, the patient presents with both the stricture and the hernia. There are often no previous X-rays. It is assumed, no doubt correctly, that in the great majority of patients the hernia precedes the stricture. Study in detail of a larger series of postoperative strictures might shed light on the vexed question of whether the hiatal hernia is always the primary lesion or may sometimes be secondary to changes in the oesophageal wall resulting from a penetrating oesophageal ulcer.

Summary

Postoperative stricture occurs most often in men after an operation for duodenal ulcer and appears to be associated with acid reflux in persons susceptible to peptic ulceration. It is
due to an acid burn of the oesophageal mucosa during the operation.

In procedures for the reduction of gastric acidity the presence of an intragastric tube seems to be a potential danger during the operation but possibly not at a later stage.

The burn of the oesophagus might be prevented by the preoperative administration of alkali by mouth.

REFERENCES