can be present and yet be extremely difficult to demonstrate radiologically, the possibility must exist that in many such cases, and perhaps in all, the essential factor is the presence of a hiatus hernia.

If any lesson is to be drawn from these cases it is that when a patient requires gastric aspiration with an indwelling tube he should, if possible, even if there is no reason to suspect the presence of a hiatus hernia, be kept in an upright position and not lying flat.

Summary

Four cases of oesophageal stricture following gastric surgery and naso-gastric intubation are recorded.

The oesophagoscopic appearances in these cases were those of reflux oesophagitis, and in each case careful radiological examination demonstrated that a hiatus hernia was present.

Reasons are given for believing that, while the recumbent position and an indwelling gastric tube play a part in producing oesophageal strictures in such cases by initiating or exacerbating reflux oesophagitis, the essential factor is the presence of a hiatus hernia.

In all four cases treatment started with regular self-bougienage. This has become unnecessary or virtually unnecessary in two cases. In Case 1 this improvement gradually developed after gastro-enterostomy and vagotomy performed for persistent duodenal symptoms, and in Case 4 immediately following Collis's operation of gastroplasty. In the other two cases self-bougienage is being continued.

It is suggested that when gastric aspiration with an indwelling tube is required in any patient the recumbent position should, if possible, be avoided.

I thank Mr. W. M. Brennan, Mr. H. W. C. Bailie, Mr. H. M. Stevenson, and Mr. I. Fraser for referring these cases to me. I am grateful to Dr. F. McDowell for the radiographic demonstration of hiatus hernias in the first three cases when they were reviewed. I am also indebted to Mr. R. D. Wood for help with the illustrations.

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OESOPHAGEAL STENOSIS AFTER PARTIAL GASTRECTOMY

BY

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Benign stricture of the oesophagus consequent on reflux oesophagitis is now a well-recognized condition. Allison (1948), in a review of 74 cases of oesophageal stenosis treated at the General Infirmary at Leeds, emphasized the role of peptic oesophagitis as a cause of stricture formation.

The frequency with which oesophageal stenosis occurs after oesophago-gastric operations has been pointed out by Barrett and Franklin (1949), and by Ripley, Olsen, and Kirklin (1952). In all the cases reviewed by them the operative procedures had interfered with the cardiac sphincter, the cardiac valve, or the "diaphragmatic pinchcock." Since the continence of the cardiac mechanism had thereby been interfered with, and acid and pepsin were able to regurgitate into the lower oesophagus, it is not surprising that reflux oesophagitis followed by stenosis had occurred.

The cases described here are of particular interest in that stenosis of the lower end of the oesophagus followed partial gastrectomy. This is doubly surprising, since the cardiac mechanism apparently had not been grossly disturbed, so there was no special reason for oesophageal reflux to take place. In addition, complete absence of free hydrochloric acid had been attained in both cases after operation; it was therefore unlikely that acid pepsin should cause ulceration of the oesophagus.

It is interesting to note that oesophageal stenosis after various types of operations for peptic ulcer has escaped mention in textbooks of surgery, for review of the literature indicates that this complication does in fact occur following various purely gastric procedures.

Oesophageal stenosis after uncomplicated gastro-enterostomy for duodenal ulcer has been recorded by Larson, Layne, and Howard (1942), and by Allison (1948). A further case of oesophageal stenosis after gastro-enterostomy has been reported by Bergqvist (1946), but this case was complicated by regurgitant vomiting, and duodenostomy had to be performed.

Review of the literature indicates that oesophageal stenosis is rare after partial gastrectomy. Craighead (1954) recorded two cases; in one of these emergency Polya–Hofmeister gastrectomy had been carried out for bleeding duodenal ulcer; the other patient had had a routine Polya gastrectomy for the treatment of a long-standing duodenal ulcer with evidence of early pyloric obstruction. Both patients, however, had had serious post-operative complications. The first of these still had a high level of free hydrochloric acid after operation and had developed stomal ulceration with stenosis, for which jejunostomy and further gastric resection had to be effected; the second patient suffered complete bursting of the wound on the third post-operative day. A somewhat similar case to the former is recorded by Benedict and Daland (1938) in which, after partial gastrectomy, the patient developed efferent-loop obstruction and subsequently oesophageal stenosis.

It is interesting at this stage to note how frequently, in patients who subsequently develop oesophageal stenosis, the previous gastro-enterostomy or partial gastrectomy has been followed by post-operative regurgitation or obstruction.

The only case of uncomplicated partial gastrectomy followed by oesophageal stenosis that we have been able to find is that recorded by Straus (1950). From personal communications with many surgeons, however, it would appear that this complication follows subtotal gastrectomy much more often than the literature indicates. For this reason it is felt desirable to record the following cases.

Case 1

A housewife aged 72 had suffered from vague dyspepsia and attacks of vomiting at intervals for 20 years. The condition was fully investigated in 1939, the symptoms being attributed to pylorospasm. Response to medical treatment was satisfactory, and no further serious symptoms occurred until she suffered a relapse in 1953, when the former symptoms of pyloric obstruction recurred. A barium-meal examination at this time showed a low distended stomach, but no ulcer crater. After a further period of peptic ulcer diet the symptoms again cleared up, but vomiting recurred in the spring of 1955, and persisted until the patient was seen on August 11 of that year. At this time her general condition was poor, and her weight had fallen from 8 st. 4 lb. to 6 st. (52.6 to 38.1 kg.). The clinical picture was that of

complete pyloric obstruction, and this was confirmed by barium-meal examination.

After careful restoration of fluid and electrolyte balance, operation was performed on September 2. The stomach was grossly hypertrophied and dilated, and there was an extreme degree of pyloric obstruction. The pyloric narrowing was attributed to the healing of a peptic ulcer. No other abnormality was detected in the abdomen. Partial gastrectomy was carried out, the lesser curve of the stomach being reconstructed so that the gastric remnant formed a smoothly tapering tube which was joined by anastomosis with the jejunum in front of the colon, and with the afferent loop to the lesser curvature. The patient's immediate postoperative progress was entirely uneventful and uncomplicated. After operation a Ryle tube was left in the stomach for 72 hours.

On the twentieth day after operation, when the patient was awaiting discharge, she developed a burning pain behind the sternum which radiated through to the back just below the shoulder-blades, and was associated with hiccup. Swallowing food brought on the pain, and there was some sensation of obstruction at the lower end of the sternum. The pain and oesophageal obstruction were intermittent and were attributed to spasm. Barium-swallow examination at this time showed a fixed smooth narrowing of the lower end of the oesophagus, which was attributed to spasm. Oesophagoscopy showed inflammatory changes in the lower end of the oesophagus with some narrowing. Dilatation of the oesophagus was carried out and the patient discharged.

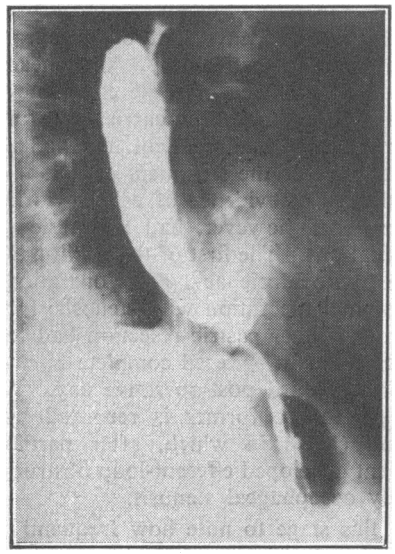

FIG. 1.—Case 1. Barium-swallow radiograph taken nine months after partial gastrectomy, showing an elongated narrowing of lower end of oesophagus, with dilatation above the narrowed segment.

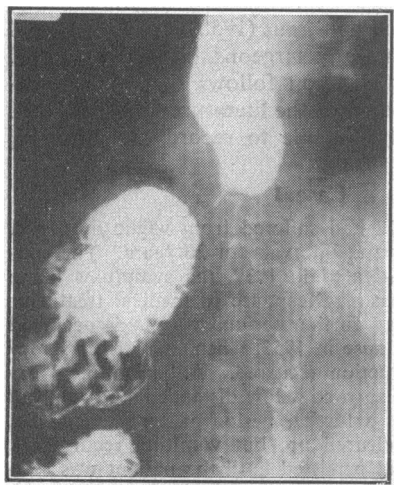

FIG. 2.—Case 1. Oblique (anterior-posterior) view of oesophageal stricture, showing no evidence of new growth.

After discharge from hospital the patient's general condition improved, and within three months her weight had increased by 21 lb. (9.5 kg.). She still, however, complained at intervals of attacks of the same retrosternal pain, with dysphagia. Further dilatation of the oesophagus produced a very temporary benefit, and, though her general condition improved and there was further increase in weight, the attacks of pain became more persistent and the difficulty in swallowing more constant.

Barium-swallow examination on June 11, 1956, showed narrowing of the distal 2 in. (5 cm.) of the oesophagus with dilatation above the narrowed segment (Fig. 1). There was increased peristalsis, and the narrow portion showed a beaded appearance when the bolus was actually passing. There was no filling defect to suggest new growth (Fig. 2), and on placing the patient in the Trendelenburg position no reflux of barium occurred. The gastric remnant showed no abnormality and the meal passed through the stoma quite normally.

Because symptoms persisted the patient was admitted on June 20 for reinvestigation. The *x*-ray appearances were unchanged, and oesophagoscopy showed inflammatory changes in the lower end of the oesophagus with narrowing of the lumen and small superficial longitudinal ulcers on the crests of the mucosal folds. After dilatation of the oesophagus, specimens of gastric juice were obtained. These showed no free acid; the total acid was 10 clinical units, and the *p*H 6.5.

It was decided to explore the lower end of the oesophagus, and this was done on June 26 through an oblique thoraco-abdominal approach along the line of the eighth left rib. The gastric remnant was found to be lying neatly below the spleen, and there was no evidence of obstruction of the efferent or afferent loop. The gastrojejunostomy stoma was entirely free and normal in every respect and there was no evidence of either stomal ulceration or of jejunitis. The oesophageal hiatus was rather loose and admitted two fingers into the sac of a small sliding hernia. The lower end of the oesophagus was swollen and oedematous, though perioesophageal adhesions were not extensive.

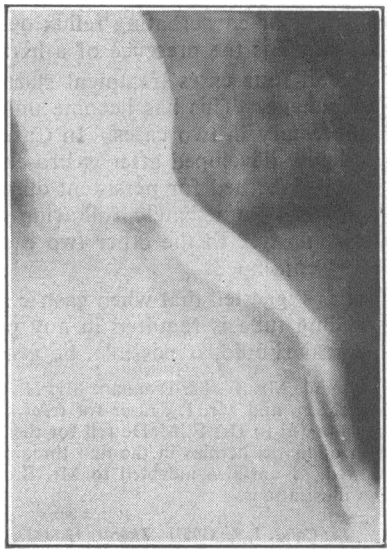

FIG. 3.—Case 1. Barium-swallow radiograph, after dilatation and conversion of Polya gastrectomy to one of Billroth I type, showing a greatly increased lumen at site of former narrowing in lower third of oesophagus.

It was decided that the oesophagitis was possibly due to the reflux of alkaline jejunal secretion in view of the *p*H of the specimen of gastric juice previously obtained. To confirm this, a further gastric specimen was obtained and the *p*H was found to be 7. In view of these findings and for reasons discussed below, it was decided to convert the Polya-Hofmeister gas-

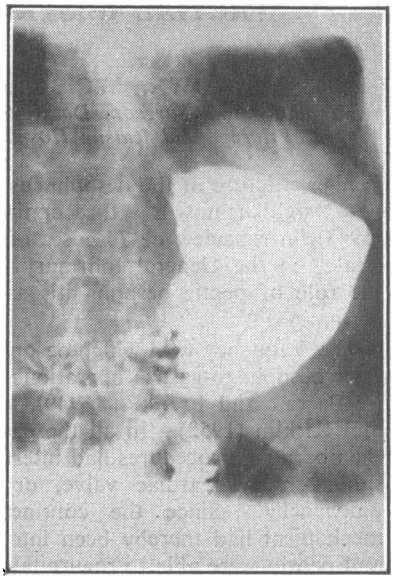

FIG. 4.—Case 1. Barium-meal radiograph showing normally functioning Billroth I gastrectomy.

trectomy into one of the Billroth I type. The gastro-jejunostomy stoma was excised, retrograde dilatation of the oesophagus was carried out, and an anastomosis was performed between the re-formed stomach remnant and the duodenal stump. The gastro-jejunostomy stoma, excised *in toto*, was sent for section. This showed no evidence of stomal ulceration.

Post-operatively, a naso-gastric tube was kept in for five days, and the patient made a completely uninterrupted recovery. The retrosternal pain cleared up at once and she was able to swallow with ease. Barium-swallow examination on November 9, 1956, showed no evidence of obstruction at the lower end of the oesophagus (Fig. 3), and the gastrectomy was working normally (Fig. 4).

The patient has been reviewed at intervals for the past two years and has been entirely symptom-free; she is still gaining weight. Examination by barium swallow has been done on several occasions and no recurrence of the oesophagitis or of oesophageal narrowing has occurred.

Case 2

A schoolmaster aged 64 had had occasional attacks of heartburn for some years, but no serious trouble until two months before his admission to hospital. As a result of serious domestic and personal worries he had been smoking and drinking to excess. He developed epigastric pain, which came on after food and was not relieved by food, with occasional night pain. Vomiting started some days before his admission to hospital; it was persistent and severe, and on several occasions he vomited blood.

On admission on October 31, 1954, his condition was very poor; he was dehydrated, and the haemoglobin estimation was 79% (11.9 g. per 100 ml.). The stomach was distended and there was visible gastric peristalsis and a succussion splash. Blood was frequently present in the vomit, and occult blood tests of the stools were constantly positive. Fractional test meal showed hyperacidity. Barium meal examination on November 9 showed a distended stomach with increased peristalsis. No barium was passing through the pylorus, and there was tenderness and deformity in the region of the duodenal cap, with gastric residue at 24 hours.

After four weeks' medical treatment the patient's condition improved. At operation on December 1 gross pyloric obstruction was confirmed. The narrowing had occurred round an ulcer exactly on the pylorus, which penetrated deeply into the pancreas and was surrounded by oedema and fibrosis. A partial gastrectomy was carried out with removal of three-quarters of the stomach, and after refashioning part of the lesser curvature an anastomosis of the afferent loop to the lesser curve was carried out, using exactly the same technique as in Case 1. Post-operative progress was entirely uneventful and uncomplicated. After operation a test meal showed complete achlorhydria. Section of the ulcer confirmed that it was a pre-pyloric ulcer with no evidence of malignancy.

On the day of discharge from hospital, three weeks after operation, the patient found that he could not take normal meals because of difficulty in swallowing. Solid food appeared to stick at the lower end of the sternum and there was a sensation of heaviness in the upper part of the epigastrium. On occasion a mouthful or two of undigested food was regurgitated.

These symptoms persisted, and after five months his weight had fallen from over 12 st. to 8 st. 10 lb. (76 to 55.3 kg.). During this period the patient had failed to report to hospital or to his own doctor, but on June 4, 1955, he was readmitted in emergency in an emaciated condition. He was unable to swallow anything except fluids, and bedsores were developing.

It was thought that the patient had developed a carcinoma of the oesophagus, and after intense medical treatment he became fit enough for barium-swallow examination to be carried out on July 20. This showed a stricture in the lower third of the oesophagus (Fig. 5). Oesophagoscopy showed an inflammatory narrowing at the lower end of the oesophagus but no evidence of growth. After dilatation had been carried out, specimens of gastric juice were taken, and these showed complete absence of free hydrochloric acid. After dilatation there was no improvement in the dysphagia, and the oesophagus was re-explored on July 22. A thoraco-abdominal incision was made along the line of the seventh rib from the mid-axillary line on the left side, over the costal margin to the midline on the anterior abdominal wall, and the chest cavity and abdomen were widely opened. No growth was found, but there was thickening and oedema of the oesophagus, suggestive of oesophagitis. There was no evidence of diaphragmatic hernia. For reasons discussed further below, it was decided to convert the Polya–Hofmeister into a Billroth I gastrectomy. The gastric remnant was re-formed and joined by anastomosis with the duodenal stump, and the jejunal stoma carefully repaired. After this operation a Ryle tube was kept in the stomach for five days, and the only other point of special notice was that a further oesophageal dilatation was carried out.

After operation the patient's condition improved steadily. The total proteins on September 12 had risen to 6.2 g. per 100 ml. (albumin 3.5 g., and globulin 2.7 g.). *X*-ray examination on February 17, 1956, showed very slight narrowing of the lower end of the oesophagus with a normally functioning gastrectomy (Fig. 6). There was no evidence of any hold-up, and the patient said he had no difficulty with swallowing. He has been seen from time to time since this operation, and his good progress has been maintained.

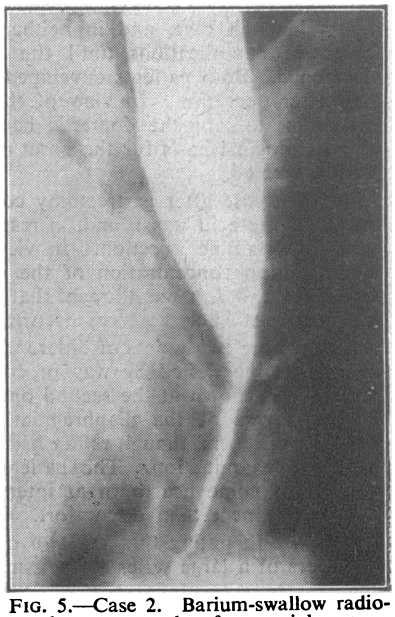

FIG. 5.—Case 2. Barium-swallow radiograph seven months after partial gastrectomy, showing simple stricture of lower end of oesophagus, with considerable dilatation above the narrowed segment.

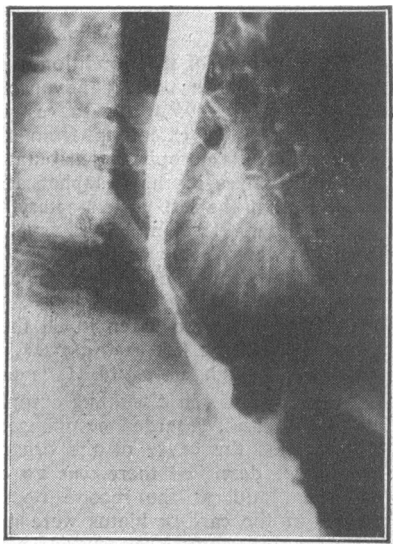

FIG. 6.—Case 2. Barium-swallow radiograph after the Polya gastrectomy had been converted to one of Billroth I type. The oesophagus had not been dilated post-operatively.

Discussion

A review of the histories of these two cases reveals some quite remarkable similarities.

Both patients were in advancing years, one being 64 and the other 72 years of age. They were both in a very debilitated condition in which vomiting and loss of weight had been marked features.

Pyloric stenosis was present in each instance. In Case 1 there had been intermittent pyloric obstruction for many years, attributable to pylorospasm, but in the year prior to operation the obstruction was constant and almost complete, and the patient's weight had fallen to 6 st. (38.1 kg.). In Case 2 the symptoms of pyloric obstruction did not begin until two months before admission to hospital, but the rapid deterioration in his condition was aggravated by serious domestic worries and over-indulgence in alcohol and tobacco.

The original operative technique of partial gastrectomy was the same in both instances. It may be of significance to emphasize that the left gastric artery was ligated near its origin, but in such a way as to leave undisturbed the left inferior phrenic (and oesophageal) branches. The lesser curve of the stomach was reconstructed quite extensively to allow anastomosis of the afferent loop to the lesser curve to lie easily in the left paracolic position below the spleen.

Both cases were managed after operation in exactly the same manner. An indwelling Ryle tube was present for 72 hours in each case, and in neither were there any post-operative complications until the oesophageal symptoms developed. Both patients developed symptoms on the 20th day after operation. In view of the time of onset of the symptoms and of the domestic background in each case, a psychological basis for the onset of the symptoms was at first suspected.

In each case after gastrectomy complete absence of free hydrochloric acid was found, in response both to test meal and to histamine injection. In view of the fact that the hydrogen-ion concentration of the gastric juice varied between 6.5 and 7, it was thought that recurrent peptic ulceration was not likely to have occurred.

There were two points of difference in the histories which should be mentioned by way of contrast. The patient in Case 1 was shown at the second operation to have a small sliding hernia at the diaphragmatic hiatus which would admit two fingers, though reflux had not been demonstrated by *x*-ray examination. The patient in Case 2 had complained of some heartburn at intervals, but vomiting was more prominent than discomfort.

It is worth noting that the two cases here described are members of a large series at present under review, in which the pre-operative preparation, operative technique, and post-operative management have been standardized. It would thus appear that the incidence of two in a series of 1,000 cases gives an accurate view of the frequency of this complication, the incidence of which is therefore 0.2%.

Aetiology

The aetiology of this condition is not understood, though various theories have been advanced to explain its occurrence. Straus (1950) has suggested the possibility of a thrombophlebitis extending from the coronary vein upwards along its oesophageal tributaries and causing inflammatory changes in the oesophageal wall. Another possibility is that the left gastric artery may have been ligated at too high a level and that as a result the oesophageal branches were obstructed and the blood supply to the lower end of the oesophagus thereby impaired, producing an area where stenosis might well occur. As previously mentioned, great care had been taken in all the cases in the series to avoid ligating the left gastric artery at too high a level, so that this, though a possibility, is unlikely.

Mosher (1945), in discussing oesophageal stenosis in cases of cardiospasm, regarded oesophageal and perioesophageal infection as the cause of the organic narrowing. In the cases here described there was no evidence of subphrenic infection, and, at the second operation, changes in the region of the cardiac hiatus were in no way prominent.

The trauma of the indwelling Ryle tube must be considered as a possibility. The frequency of urethral stenosis following the indwelling urethral catheter provides an interesting clinical parallel. Following a review of 82 cases of oesophageal stenosis studied at necropsy, Bartels (1935) felt that the naso-gastric tube was not an aetiological factor, but stated that it might act as an aggravating factor. Olsen (1948), Benedict and Sweet (1948), and Mason and Ausband (1952) all emphasized the significance of naso-gastric tubes in contributing to oesophagitis and stenosis. In the cases here described, however, it will be remembered that while the naso-gastric tube was left *in situ* for 72 hours after the Polya operation, on the other hand the tube was left in for five days when the operation was converted to a Billroth I gastrectomy. As oesophageal stenosis followed the first operation but not the second, it must be deduced that the presence of the tube does not play a fundamental part in the production of stenosis.

The final aetiological factor to be considered is that of reflux. The occurrence of peptic ulcer of the lower end of the oesophagus associated with either gastric or duodenal ulcer has been recorded on so many occasions that the vulnerability of the oesophagus to acid-pepsin is accepted without question. In the cases here described the possibility that minor degrees of reflux might have occurred is accepted, in that one case had a sliding hernia and the other had pre-operative symptoms suggestive of reflux. What cannot be accepted is that acid - pepsin played a part as the causative factor. After partial gastrectomy both cases had complete achlorhydria, and estimation of the hydrogen-ion concentration showed that this varied between 6.5 and 7. It would appear, therefore, that if reflux did take place the reaction of the fluid being so near neutrality could not of itself harm the oesophageal mucosa. Since the activity of pepsin at the *p*H's under review would be negligible (Fig. 7), a different explanation is required. Since trypsin is a most powerful proteolytic enzyme and acts in an alkaline, neutral, or slightly acid medium, it seems quite possible that tryptic activity could severely damage the lower end of an oesophagus, causing oesophagitis and stenosis. In the cases under discussion the hydrogen-ion concentration of the fluid obtained from the stomach and lower end of the oesophagus provided a very suitable medium for tryptic activity. It may well be, therefore, that in these cases a combination of factors has been involved and that tryptic activity, rather than acid-pepsin activity, was the primary factor in causing reflux oesophagitis followed by stenosis.

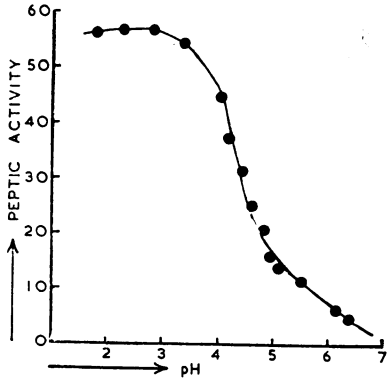

FIG. 7.—Graph showing the peptic activity of gastric juice at various hydrogen-ion concentrations. Note that peptic activity declines from a maximum at *p*H 2.1 to negligible activity at *p*H 6.0.

Treatment

It is not possible to be in any way dogmatic in regard to the treatment of this unusual condition. It is apparent, however, that after simple dilatation of the oesophagus recurrence takes place with great rapidity. In Case 2 dilatation was carried out under direct vision, but the oesophagus was oedematous and congested, so that no benefit could have been expected or anticipated, and no benefit in fact occurred. In Case 1 repeated dilatations were carried out in the first instance, but recurrence of stenosis took place with such rapidity that they were abandoned.

The failure of dilatation was presumably due to the fact that the conditions giving rise to the oesophagitis were still operative, so that the inflammatory condition persisted and stenosis recurred.

It was felt that reflux was the most likely cause of oesophagitis in the cases here described. Since both cases showed a complete achlorhydria, it was thought that acid or peptic digestion was unlikely to be the operative factor. On the other hand, the conditions for tryptic activity were ideal. It was therefore felt necessary to prevent jejunal contents from reaching the lower end of the oesophagus. The obvious means of attaining this end was to convert the Polya gastrectomy into a Billroth type. If reflux did thereafter occur, the contents would be gastric, achlorhydric, and free of jejunal content.

In both cases this manœuvre was successful. The oesophagitis and dysphagia cleared up rapidly, and though review has taken place at frequent intervals for the past two years, no evidence of recurrence of stricture has occurred. This seems to confirm that reflux of jejunal content, and probably tryptic activity, were the cause of the oesophagitis which led to stenosis.

Summary

Two cases of an unusual complication of subtotal gastrectomy are described.

The possible causes of this complication are discussed.

It is suggested, on theoretical grounds and as a result of the line of treatment adopted in these cases, that the reflux of the jejunal contents, and possibly tryptic activity, may be the operative factors in causing the oesophagitis which led to stenosis.

I thank Dr. P. N. Coleman, consultant pathologist, for the section reports and also for his work in association with Mr. S. Taylor, F.I.M.L.T., our chief technician, in preparing the photographs. I also thank Professor Ian Aird for his kind interest and help, and Dr. W. Irvin and Dr. A. K. Lamballe, who have taken the radiographs of these cases.

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ADRENALINE IN HYPERTHYROIDISM AND INSULIN HYPOGLYCAEMIA

BY

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There are many similarities between the features of acute insulin hypoglycaemia and of hyperthyroidism. Tachycardia, weakness, finger tremor, sweating, and flushing can occur to a similar extent in both conditions. Furthermore, the similarity between the features of hyperthyroidism and those of phaeochromocytoma is striking; indeed, these disorders are not infrequently confused. All three conditions are known or thought to be related to an increase of secretion of adrenaline from the adrenal medulla. Cannon *et al.* (1924) reported an increase of adrenaline secretion in cats in response to insulin hypoglycaemia and showed that this was a mechanism for raising the lowered blood sugar. It has been suggested that adrenaline is responsible for many of the symptoms of hypoglycaemic reactions (Bell *et al.*, 1956). Similarly, in man, von Euler and Luft (1952) and French and Kilpatrick (1955) demonstrated a considerable increase in urinary adrenaline excretion following hypoglycaemia.

An association between hyperthyroidism and changes in adrenaline metabolism has long been suspected. Hyperthyroid subjects have a striking increase in sensitivity to adrenaline and noradrenaline (Rosenblum *et al.*, 1933; Peltola, 1951; Schneckloth *et al.*, 1953; Brewster *et al.*, 1956). Many observers have noted changes in adrenaline content of adrenal glands in animals in which hyperthyroidism was produced by giving thyroxine (Goodall, 1951; Hökfelt, 1951; Pekkarinen *et al.*, 1951; Durlach *et al.*, 1955). Accordingly, urinary catechol excretion has been measured in a number of patients with hyperthyroidism and the results are compared with those found in insulin hypoglycaemia and in normal subjects.

Methods

The urinary catechol excretion was measured in 12 euthyroid subjects, in 17 patients with hyperthyroidism of varying degree, and in two patients with hypothyroidism. The estimation was repeated in two patients previously hyperthyroid who had become euthyroid following treatment with radioiodine.

In all but one of the patients with hyperthyroidism the estimation was performed while they were in-patients and thus at rest in bed. The normal subjects, who were mainly hospital and university staff, were performing their usual duties. Urine was collected for 24 hours into a dark bottle containing hydrochloric acid.

Two methods of extraction were used, either aluminium hydroxide (von Euler and Hellner, 1951) or aluminium oxide (von Euler and Orwen, 1955). The latter method required modification as our assay was done on the rat-uterus preparation (Diller, 1958). Recovery by both methods varied between 65 and 90%. No correction was made for this.

Adrenaline assays were performed, using the oestrous rat uterus stimulated by carbachol (Gaddum and Lembeck, 1949). Values are given as L-adrenaline. The assay was checked in two ways. Firstly it was shown that all adrenaline-like activity was destroyed by boiling an aliquot of the extract at alkaline *p*H. Secondly, addition of known amounts of adrenaline to the extract after measurement produced only additive effects and no potentiation or masking was found. If either of these requirements was not met another extract was prepared from a further urine collection.

Total catechols were measured in the same extract on the cat's blood pressure, and the noradrenaline content was found by using the adrenaline value from the uterus assay (Burn *et al.*, 1950). Values are given as DL-noradrenaline.

Attempts were also made to measure adrenaline and noradrenaline by a fluorimetric method (von Euler and Floding, 1955), so that the number of estimations could be increased. However, it was found that there was much non-specific fluorescence in all the urines that were studied. This produced more inaccuracy when the level of catechols was in the physiological range. Similar results with fluorimetric methods are described by Pitkänen (1956).

Radioiodine tracer studies were performed in all of the patients with thyroid disorders. They were given 25 μc. of ^{131}I and the thyroid uptake was measured after 4 and 48 hours. Protein-bound ^{131}I was measured at 48 hours, using trichloracetic acid as protein precipitant (Goodwin *et al.*, 1951).

Results

The clinical diagnosis of hyperthyroidism was confirmed by radioiodine studies; in all of them the four-hour uptake was more than 40% of the dose and the 48-hour protein-

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