OESOPHAGEAL STRICTURES AFTER GASTRIC SURGERY AND NASO-GASTRIC INTUBATION

BY

J. A. W. BINGHAM, M.Ch., F.R.C.S.

From the Royal Victoria Hospital, Belfast

The occasional development of an oesophageal stricture without apparent cause following the use of an indwelling naso-gastric tube and, usually, some type of gastric surgery has been recognized for many years. Less often, this condition has followed naso-gastric intubation and either some other abdominal operation or a severe illness not treated by operation. Douglas (1956), when reporting five of these cases, referred to 23 reported in the literature since 1915.

The mechanism of production of these oesophageal strictures and the part played by various factors have previously been discussed by several authors, without any generally agreed or very convincing conclusion having been reached.

A personal experience of four of these cases is recorded below. Study of them throws, I believe, some light on the aetiology of the condition.

Case 1

A man aged 41 was operated on in the Mid-Ulster Hospital for a perforated duodenal ulcer on May 24, 1950, and the perforation closed. Gastric aspiration by means of an indwelling Ryle tube was maintained for 48 hours. During this time the patient was lying flat. Troublesome heartburn started 10 days post-operatively. Otherwise his progress was quite satisfactory until three or four weeks after the operation, when, shortly after he had returned home, he began to experience a little dysphagia. The dysphagia rapidly increased, and he was readmitted on July 7 able to swallow even fluids only with difficulty. Barium-swallow examination showed a narrow stricture involving apparently the lowest few inches of the oesophagus and slight dilatation of the oesophagus above. He was transferred to the Royal Victoria Hospital, Belfast, on July 19, in an advanced stage of starvation, with a slow pulse, hypotension, and extreme prostration and general weakness. A gastrostomy under local analgesia was carried out as a life-saving measure before any investigation of his dysphagia was undertaken. His condition rapidly improved.

At oesophagoscopy on August 7 I found that the mucosa in the lower oesophagus was reddened, oedematous, easily traumatized, and showed superficial ulceration just above a narrow stricture. Gum-elastic bougies were readily passed. It was noted at the time that the appearance suggested peptic oesophagitis.

Following the dilatation, improvement in swallowing lasted for only a few days, and after two further oesophagoscopy dilatations regular self-bougienage by the patient was begun, starting with the smallest sizes and gradually increasing the size until a No. 18 English could be passed. He was soon able to swallow reasonably well, though having to be careful that solids were cut up fine or minced. He was discharged on September 10 to continue with self-bougienage, and found that by passing his bougie, at first daily, later at weekly intervals, and eventually at intervals of several weeks, he was able to swallow satisfactorily.

He continued, though, to be troubled repeatedly with ulcer dyspepsia, including one episode of haematemesis and melaena, and on June 8, 1955, a gastro-enterostomy and vagotomy was carried out by Mr. E. Morrison. Recovery was uneventful, and the patient returned home to continue with self-bougienage.

For a period of some weeks after this operation he again required to pass his bougie as often as two or three times a week, but, apart from this, the operation has proved most satisfactory in every way. Not only has the patient been free from ulcer dyspepsia, but he no longer suffers from heartburn, which had been very troublesome ever since his perforation in 1950, and he has been able gradually to lengthen the intervals between dilatations until these have virtually been discontinued. When last seen he had not required to pass his bougie for eight months, and, provided solids are cut up fine, he has no trouble with swallowing.

When he was first seen it was thought the radiographs showed the oesophageal stricture to extend to the oesophageal hiatus, but after Case 4 of this series had been seen the radiographs were reviewed, and it was then realized that the appearances were consistent with the presence of a hiatus hernia. Further radiographs, taken recently with this in mind, confirmed that a hiatus hernia was indeed present, and that gastro-oesophageal reflux could be produced.

Case 2

A man aged 68, who had had ulcer-type dyspepsia for many years, was operated on in Coleraine Hospital on January 4, 1954, and a perforated duodenal ulcer sutured. Two days after operation a Ryle tube was passed because of vomiting, and aspiration was carried out for at least 48 hours, with the patient lying flat during this time. He was discharged on January 28, but was readmitted on March 5 because of projectile vomiting. A gastro-enterostomy was performed and he was discharged on April 8. On June 15 he was again readmitted because of gradually increasing vomiting immediately after every meal. Barium-swallow examination showed a stricture with hold-up of barium in the lower third of the oesophagus. The gastro-enterostomy was functioning satisfactorily.

At oesophagoscopy on July 4 I found a stricture at 37 cm. Oesophageal mucosa for a short distance proximal to this was reddened, inflamed, and ulcerated, the appearance being that of reflux oesophagitis. A biopsy specimen showed granulation tissue infiltrated by acute inflammatory cells.

The patient learnt to pass oesophageal bougies himself and was discharged on July 30 to continue bougienage at home. At first daily bougienage was carried out, but it proved possible gradually to decrease the frequency of dilatations, and three and a half years later he required to pass a bougie only at intervals of several weeks, was keeping well, had no dysphagia, except occasionally for meat, and had put on over a stone (6.4 kg.) in weight.

This patient has recently been reviewed and further radiographic examination carried out. The presence of quite a large hiatus hernia with reflux was demonstrated, and above this a slightly strictured lower segment of oesophagus.

Case 3

A man aged 61 had a gastro-enterostomy and vagotomy performed for pyloric stenosis in the Royal Victoria Hospital, Belfast, on June 13, 1957. Gastric aspiration was carried out for 48 hours. He was transferred to a convalescent hospital 12 days later. Shortly after arriving there he began to have dysphagia. Barium-swallow examination on July 5 showed what seemed to be an unusually long, narrow stricture apparently involving the lower half of the oesophagus (Fig. 1).

At oesophagoscopy (by Mr. H. M. Stevenson on July 15) the appearance was of an area of gross oesophagitis, at the lower end of which was a tight, simple stricture. This was dilated easily.

The dysphagia soon became as bad as before, and further oesophagoscopy dilatations were carried out on July 26 and August 15. Any benefit from these dilatations lasted for only very brief periods, and when I saw him on September 19 he was able to swallow scarcely anything but water, and sometimes milk, and was in a state of extreme weakness and debility.

At oesophagoscopy on September 20 I found the condition apparently unchanged, the mucosa at mid-oesophagus being red and oedematous and ending in a stricture. This was dilated.

Thereafter self-bougienage was carried out by the patient, at first three times daily, and when he could pass a size 17 English bougie he was discharged from hospital. Since then daily bougienage has been continued. His condition has improved steadily. He has to cut his food up fine, and there is occasionally a transient hold-up with solids.

The earlier radiographs were regarded at the time as showing the oesophageal stricture extending to the oesophageal hiatus, and a hiatus hernia was not specially looked for. A recent barium examination, carried out with the possibility of a hiatus hernia in mind, demonstrated that below the stricture there was indeed a large hiatus hernia with free reflux, and that the stricture was not nearly as long as had been thought (Fig. 2).

FIG. 1

FIG. 2

FIG. 1.—Case 3. Barium-swallow radiograph (July 5, 1957) showing what seemed an unusually long oesophageal stricture. A hiatus hernia was not specially looked for. FIG. 2.—Case 3. Barium study of oesophagus and cardia (February 27, 1958) revealed a large hiatus hernia.

Case 4

A man aged 39 had a rather harrowing story of peptic ulcer trouble. It can be summarized as follows: 1935, perforated duodenal ulcer sutured. 1942, gastro-enterostomy (Professor P. T. Crymble). 1955, gastro-enterostomy undone and Polya-type gastrectomy performed because of an anastomotic ulcer (Mr. M. Lavery). 1956, perforation of anastomotic ulcer sutured. May 14, 1957, Polya-type gastrectomy converted into Billroth I gastrectomy, a portion of jejunum involved in an anastomotic ulcer being excised (Mr. I. Fraser).

The patient was extremely ill for some days after this last operation, and gastric aspiration was carried out for five days, with the patient lying flat. A few days later he began to be troubled with hiccup and severe heartburn. As soon as he went home, a little over three weeks after operation, he experienced some dysphagia for the first time. This continued and became progressively worse. When I saw him on January 9, 1958, he was unable to swallow solids at all. Barium radiographs, shortly before this, had shown a hiatus hernia with readily produced reflux, and above this a stricture at the lower end of the oesophagus. (It was after seeing this case that the earlier cases were re-examined

and were also found to have hiatus hernias.) It is worth noting that barium-meal examination three months before his last gastric operation had not shown any sign of an oesophageal stricture. A hiatus hernia was not looked for at that time.

At oesophagoscopy (January 12) the appearance was that of a reflux oesophageal stricture, the mucosa just proximal to the stricture being reddened, oedematous, and slightly ulcerated, and bled easily on trauma. The stricture was dilated. A biopsy specimen showed smooth muscle with a marked chronic inflammatory infiltrate.

The patient began self-bougienage and was able to swallow a reasonable diet. However, he continued to have heartburn, and in view of his age and the chances of his reflux stricture becoming progressively worse, I decided that, instead of treating his condition purely by dilatation, I would carry out Collis's operation of gastropasty (Collis, 1957). I have recently been using this operation with most gratifying results in some cases of hiatus hernia with reflux oesophagitis, stricture formation, and shortening of the oesophagus. The operation was performed on January 28.

Up to the time of writing, two and a half months later, the patient has done well. Since the operation he has had no more heartburn, has required no further dilatations, and his swallowing has improved steadily, though he still has to be a little careful with solids. On recent radiographic examination reflux through the hiatus from the portion of stomach below the diaphragm could not be produced.

Discussion

Possible reasons for the development of oesophageal strictures in this type of case have been discussed by others. Straus (1950), for example, suggested that they may be the result of trauma from the tube, but this view has usually been discounted. Certainly the finding of acute inflammatory changes, with redness, oedema, and ulceration of the mucosa, weeks and even months after the removal of the indwelling tube, as in the present cases, clearly is against this view. Douglas (1956), in reporting five cases similar to those described in this paper, suggested that the most important factor in the production of these strictures was probably the patients' vomiting. This, however, is not very convincing, for some of these patients did not have a great deal of vomiting, and the effect of gastric aspiration with an indwelling tube is to prevent vomiting post-operatively.

Once it was recognized that the four cases described above all had hiatus hernias these difficulties disappeared. As already mentioned, the oesophagoscopy appearances in these cases were identical with those found in the reflux oesophagitis and reflux oesophageal stricture so often complicating hiatus hernia. The association in each case of a hiatus hernia and these oesophagoscopy findings makes it appear certain, therefore, that the oesophageal strictures in these cases were due to the hiatus hernias and the result of gastro-oesophageal reflux. This is not to say, though, that the operations and indwelling gastric tubes did not play a part. Reflux oesophagitis certainly often develops in association with hiatus hernia without any prolonged period of recumbency and without an indwelling tube. But reflux occurs more readily in the recumbent position, and almost certainly more readily still if an indwelling tube is present at the same time. Therefore this combination of recumbency and an indwelling tube in a patient with an unrecognized hiatus hernia could well start off reflux oesophagitis where none had been present before or markedly exacerbate a pre-existing mild oesophagitis. The temporary increase in dysphagia in Case 1 following gastro-enterostomy and vagotomy, when the patient was again confined to bed and an indwelling gastric tube was again employed, provides support for the view that such a regime may exacerbate reflux oesophagitis in a patient with a hiatus hernia.

It cannot, of course, be stated with certainty that similar cases described by others have had unrecognized hiatus hernias. But as in the first three cases described here the hernias were at first unrecognized, and as a hiatus hernia

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 up-right 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.

REFERENCES

- Collis, J. L. (1957). *Thorax*, 12, 181.
Douglas, W. K. (1956). *Brit. J. Surg.*, 43, 404.
Straus, G. D. (1950). *Arch. Otolaryng. (Chicago)*, 51, 165.

OESOPHAGEAL STENOSIS AFTER PARTIAL GASTRECTOMY

BY

K. C. McKEOWN, M.Ch., F.R.C.S., F.R.C.S.Ed.
*Senior Consultant Surgeon, Darlington Memorial Hospital
and Friarage and Rutson Hospitals, Northallerton*

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