GASTRIC ULCER OCCURRING AFTER VAGOTOMY

BY

D. M. MORRISSEY, M.Ch., F.R.C.S.

(From the Department of Surgery, Queen Elizabeth Hospital, Birmingham)

Increasing experience of the late results of vagotomy for duodenal ulcer has shown that the percentage of patients thus treated who are not relieved of their ulcer distress may be as high as 25% (Orr and Johnson, 1949) even though there is a satisfactory reduction in the free gastric acid level. Other authors, reviewing their results after shorter periods, report more favourable figures (Moore, 1947, 1948; Cameron *et al.*, 1949; Healy and Sauer, 1949). Search of the literature for the causes of unfavourable results has revealed only two records of the occurrence of gastric ulceration after successful vagotomy for duodenal ulcer. Orr and Johnson (1949) report one such case in their series of 71 cases, and Dragstedt *et al.* (1949) record two examples of this complication in their series of 197 cases of duodenal ulcer treated by vagotomy alone.

The complication is therefore rare, but is important because of the malignant potentiality of gastric ulcers and because such ulcers are sometimes remarkably painless after vagotomy (Kugel and Janzen, 1949). For this reason it is felt that the following two case reports will be of interest, as they provide further evidence of the inefficacy of vagal section in interrupting the normal course of simple gastric ulceration. These cases come from a series of 79 cases of duodenal ulcer treated by vagotomy alone at the Queen Elizabeth Hospital, Birmingham. Over fourteen

months have elapsed since the most recent of these cases was operated upon, and every case has been followed up at six- and twelve-monthly intervals with barium-meal examinations and an insulin test.

Case 1

A wood machinist aged 40 was seen in the out-patient department in March, 1948, complaining of symptoms of duodenal ulcer of 13 years' duration. From 1935 to 1938 his periods of pain lasted approximately one week, with intervals of freedom up to three months. From 1939 onwards his symptoms were more persistent and were associated with vomiting. Barium-meal examination in 1944 revealed a duodenal ulcer, and from that time he followed a careful dietary regime which controlled his symptoms so that he was able to continue at work. During May, 1946, the ulcer perforated. After operation he was symptom-free for seven months. From January, 1947, his symptoms were more or less continuous. There was no haematemesis or melaena.

Examination revealed a well-built man, weighing 142 lb. (64.4 kg.) and 5 ft. 9 in. (175 cm.) in height, with an abdominal scar from his previous operation. Except for tenderness on palpation of the epigastrium, physical examination revealed no further abnormality. The relevant points from his laboratory investigations and x-ray examination were as follows:

Barium meal and screening: duodenal deformity present; stomach emptying in four hours. Radiograph of chest was normal. Blood count: red cells, 5-6 millions per c.mm.; Hb, 15.7 g.%; C.I., 1.01; white cells, 5,700 per c.mm. A fractional test meal revealed the following: fasting juice 29 ml.; no mucus in any specimen; bile in the 2½-hour specimen; maximum acidity in 1½-hour specimen (0.22 g. of free HCl per 100 ml.); residual juice 2 ml. The histamine test showed maximum acidity in a 30-minute specimen (0.42 g. of free HCl per 100 ml.). The night fasting gastric secretion (maximum acidity 0.43 g. of free HCl per 100 ml.) and the insulin test are graphically presented in Fig. 1.

FIG. 1.—Case 1 (April 10, 1948). Night gastric secretion and insulin test before vagotomy.

FIG. 2.—Case 1 (October 11, 1948). Night gastric secretion and insulin test six months after vagotomy.

On consideration of these findings the case was thought suitable for treatment by vagotomy, but it was felt that, even though there was no evidence of pyloric narrowing, the site of the previous perforation should be inspected.

On April 15, 1948, under general anaesthesia, the left side of the chest was opened after excision of a segment of the eighth rib. Through a 4-in. (10-cm.) incision in the diaphragm, forward from the region of the oesophageal hiatus, the stomach was carefully inspected and the patency of the pyloric canal and duodenum confirmed by palpation. A duodenal ulcer was present, but there was no evidence of gastric ulcer. The diaphragmatic incision was closed and vagal section carried out in the usual manner with resection of 2-in. (5-cm.) segments of the nerves.

Convalescence was uneventful, the patient being discharged on the 11th day. His weight on leaving hospital was 138 lb. (62.6 kg.). By the third month following his operation he was perfectly well, had resumed work, and had gained 14 lb. (6.4 kg.) in weight to 152 lb. (68.9 kg.). On October 1 he complained of epigastric pain after food, relieved by vomiting, and volunteered the information that these fresh symptoms were quite unlike his previous ones. He began to lose weight, and when readmitted to hospital on October 15 he weighed 136 lb. (61.7 kg.). The insulin test was repeated (Fig. 2), and barium-meal examination now showed a gastric ulcer about the middle of the lesser curve, with no delay in stomach-emptying. The duodenal cap was deformed but not tender. His symptoms were not controlled by diet and alkalis, and four weeks later a partial gastrectomy was performed.

At operation the gastric ulcer was readily identified about midway along the lesser curve, as was the scar of the old duodenal ulcer, which appeared to be healed. Examination of the stomach afterwards showed a peptic ulcer about $\frac{1}{2}$ in. (1.2 cm.) in diameter, with yellow necrotic base, penetrating deeply into the stomach wall. Histological section revealed a complete breach of muscular coat and replacement by fibrous tissue without evidence of malignancy. The patient's convalescence was again satisfactory, and he remained symptom-free 21 months later.

Case 2

A man aged 45 had had epigastric pain after meals for 12 years, with periods of freedom from symptoms up to two months. He had been treated by six weeks' rest in bed and dietary measures on two occasions, but pain recurred on resuming full work. There had been no haematemesis or melaena. Examination showed a well-nourished man of 5 ft. 7 in. (170 cm.) in height, weighing 129 lb. (58.5 kg.). Physical examination was entirely negative.

Fractional test meal: "high normal" acid curve with a large volume of resting juice (194 ml.) containing charcoal. Blood count: red cells, 5,090,000 per c.mm.; Hb, 14.1 g.%. A barium meal showed deformity of the duodenal cap, thought to be due to chronic ulceration.

On February 11, 1949, laparotomy was carried out by Mr. B. N. Brooke. A chronic duodenal ulcer was seen on the

anterior wall of the first part of the duodenum with some scarring, but no appreciable stenosis and no evidence of gastric ulcer. In view of this he considered that vagal section was justifiable, and this was effected below the diaphragm.

The patient's immediate convalescence was uneventful, and he was discharged from hospital 12 days later. Three months after the operation he was still symptom-free, had gained 17 lb. (7.7 kg.) in weight, and had resumed his normal occupation of grocer. He was enthusiastic about his progress and was now able to eat a variety of foods which he had not been able to do for many years. He was seen again on July 13, 1949, when he complained of foul eructations and occasional attacks of diarrhoea, but no postprandial pain or discomfort. On August 25 he had a sudden severe haematemesis (he alleged that he vomited about three pints (1.7 litres) of blood-stained fluid), followed by repeated tarry stools.

On admission to hospital next day his red blood cells numbered 2,300,000 per c.mm. and haemoglobin was 44%. He was given a slow transfusion of two pints (1.14 litres) of blood, and after a temporary rise his blood count and haemoglobin began to drop steadily. The stools still contained altered blood on the tenth day after admission. In view of the continued bleeding a further transfusion was given and a partial gastrectomy was performed on September 6 by Mr. Brooke. On this occasion the old duodenal ulcer appeared to have healed, but there were now two small but deep gastric erosions about 3 in. (7.5 cm.) from the pylorus on the lesser curvature. Histological examination of the ulcers revealed a complete breach of the muscle coat and fibrous tissue formation at their bases (Fig. 3). The patient's convalescence was again uneventful. He was discharged from hospital 12 days after operation with a blood count of 4,900,000 red cells per c.mm. and 98% haemoglobin. He has remained symptom-free up to the present time (July, 1950).

Comment on the Cases

Post-operative insulin test results are not available in Case 2, as the patient was admitted to hospital with haematemesis within the six-months period, but the direct evidence of healing of his duodenal ulcer with the entire relief of symptoms while on a full mixed diet probably indicates that complete vagal section was achieved. The result of the insulin test six-months post-operatively in Case 1 is considered to be compatible with complete vagal section, as there was no rise of the acid level following an induced hypoglycaemia of 28 mg. per 100 ml. The view that the gastric ulcer developed after the vagotomy is based on the x-ray evidence and examination at the time of operation, which of necessity, by reason of the approach used, involved manual retraction of the lesser curve.

Discussion

The addition of these two cases to those already reported gives an incidence of 5 in 347 cases of complete vagotomy for duodenal ulcer. Many of the other causes of unfavourable results, such as recurrence and non-healing of the duodenal ulcer, may be explained on the grounds of incomplete nerve section, but such tests as we possess—the insulin test, lowering of gastric acidity, clinical and radiological healing of the original ulcer—indicate that in these cases the vagal section was complete. Dragstedt thought that gastric stasis may have been a causal factor, as he has not had this complication in any of 262 cases of duodenal ulcer treated by vagotomy and gastroenterostomy; and Orr and Johnson found gastric retention in their case, which also presented with painless haematemesis. There was evidence of gastric retention in my Case 2, but barium-meal examination of Case 1 at the time of onset of ulcer symptoms did not reveal any delay in gastric emptying.

FIG. 3.—Case 2. Photograph of stomach showing two gastric ulcers.

It is difficult to reconcile the occurrence of gastric ulcer after successful vagotomy with current views on the pathogenesis of the condition. There is after vagotomy a lowering of the gastric acid level and particularly of the volume and degree of acidity of the night acid secretion. This problem might be solved if an unequivocal test of complete vagal section were available; and the observations of Brooke (1949) with regard to the insulin test are supported by the findings of Walters and Brownson (1948) and Walters *et al.* (1949) that in their series the clinical results in those cases showing a negative post-operative insulin test were similar to those showing a positive response. Without therefore being able to say that vagotomy has any causal relationship to the gastric ulcers in the cases described above, it does seem that the lowering of acidity brought about by vagal section affords no protection against ulceration developing. Further confirmation is forthcoming from the report of Nuboer (1949), who has described the occurrence of gastric ulceration in some of his cases following resection of the oesophagus for malignant disease, when the vagi were necessarily completely divided.

These two cases are obvious failures following vagotomy in the treatment of duodenal ulcer. The Birmingham series referred to above contains cases which have had other undesirable sequelae of the operation: these will be recorded when the series is fully analysed.

Summary

In a series of 79 cases of duodenal ulcer, two cases developed gastric ulcer following successful vagal section. These two cases are described.

The view is expressed that vagotomy is unsuitable as a treatment of gastric ulceration and confers no protection against the development of a gastric ulcer.

In both cases the gastric ulcers developed within six months of the vagal section.

I am indebted to Mr. B. N. Brooke for permission to publish Case 2, and to Professor F. A. R. Stammers for his guidance and help in the preparation of this paper.

REFERENCES

- Brooke, B. N. (1949). *Lancet*, **2**, 1167.
 Cameron, D. A., Burt, C. W., and Tuttle, W. M. (1949). *J. Mich. med. Soc.*, **48**, 301.
 Dragstedt, L. R., Camp, E. H., and Fritz, J. M. (1949). *Ann. Surg.*, **130**, 843.
 Healy, M. J., jun., and Sauer, P. K. (1949). *Bull. N.Y. Acad. Med.*, **25**, 449.
 Kugel, A. I., and Janzen, J. (1949). *Gastroenterology*, **12**, 993.
 Moore, F. D. (1947). *Arch. Surg., Chicago*, **55**, 164.
 — (1948). *Gastroenterology*, **11**, 442.
 Nuboer, J. F. (1949). *Acta gastro-ent. belg.*, **12**, 380.
 Orr, I. M., and Johnson, H. D. (1949). *British Medical Journal*, **2**, 1316.
 Walters, W., and Brownson, B. C. (1948). *Bull. Chic. med. Soc.*, **51**, 135.
 — and Phillips, S. K. (1949). *Kentucky med. J.*, **47**, 254.

According to a press report the New York Academy of Medicine has arranged a series of "refresher" lectures for doctors in New York which will be given over the radio. Believed to be the first of its kind, the series is designed to bring the latest scientific developments in medicine to doctors who are unable to attend medical conventions, who have no hospital connexions, or who are too busy to read through the medical journals. The one-hour programmes, to be broadcast every Thursday evening, will actually be transcriptions of papers delivered at the Academy's regular Friday afternoon lectures. A test broadcast last April on A.C.T.H. and cortisone received such wide response that planning went ahead for the series. The New York radio authorities are now planning to start a similar series of "refresher" lectures for lawyers and are hoping to expand the idea to other professions.

EPIDEMIC OF BENIGN DRY PLEURISY

BY

D. G. H. SYLVESTER, M.B.

Medical Superintendent, St. Mary's Hospital, Paddington, W.2
 (From the Student Health Service)

Since Sylvest's original monograph on Bornholm disease, or epidemic myalgia, was published in 1933, a similar condition has been described in countries as widely separated as Scandinavia, India, and the United States. A diagnosis of benign dry pleurisy has been made in cases with associated pleural friction.

In this country epidemics have been sporadic in appearance and often confined to localized communities such as schools (Attlee *et al.*, 1924; Carter, 1933), children's hospitals and convalescent homes (Lloyd, 1924; Williamson, 1924), or to single households (Howard, 1938), while Pickles (1933, 1939, 1948) has described several outbreaks in village communities in Yorkshire.

Scadding (1946) reviewed the literature on Bornholm disease and described a series of similar cases observed among soldiers in the Middle East. These were characterized by a high incidence of pleural friction (55% of 20 cases), and he consequently named them benign dry pleurisy.

This paper records an epidemic of benign dry pleurisy occurring in two households in the Bayswater district of London during September and October, 1949. Eleven cases in all were encountered, two of which were observed during the course of their illnesses.

Case 4

A healthy medical student aged 22 became ill on October 4: he complained of severe pain in both upper quadrants of the abdomen, which was lancinating and worse on coughing or deep breathing. It radiated first to the right, then to the left shoulder, and was accompanied by headache and sweating.

When first seen on October 5 his breathing was shallow and painful and his temperature 100.8° F. (38.2° C.), but, apart from upper abdominal tenderness, there were no abnormal physical signs. In view of the position and radiation of the pain, the absence of cough or abdominal rigidity, and his clean tongue, a tentative diagnosis of diaphragmatic pleurisy was made.

On October 6 his temperature had risen to 101° F. (38.3° C.) and his respirations were 40 a minute. The pain and tenderness were now localized in the region of the left costal margin. A chest radiograph showed no abnormality at the left base; there was, however, a slight but definite decrease in translucency at the right base, together with slight elevation of the right half of the diaphragm. The total leucocytes were 8,000—polymorphs 6,000 (75%), lymphocytes 1,600 (20%), monocytes 400 (5%).

On October 7 there was a pleural rub in the right axilla which persisted for three days. His E.S.R. (Westergren) was 18 mm. in one hour and the Mantoux reaction was positive to 1 in 1,000 tuberculin. The diaphragm moved normally on screening. Recovery was rapid; by October 11 he was free of symptoms and there was no relapse. A radiograph of the chest taken on November 16 showed no residual shadowing.

Case 10

On October 22, eighteen days after the above patient had fallen ill, a second medical student, aged 26, was seized with pleuritic pain in the right shoulder which wakened him in the night and which spread to the right upper abdomen when he got out of bed. The pain was worse on lying down and prevented him from lying on the affected side. His temperature was 99° F. (37.2° C.) and there were no abnormal physical signs. On October 23 the pain had spread to the left shoulder and