SURGERY OF PEPTIC ULCERATION AND ITS COMPLICATIONS

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Part III

Stomal Ulceration

Stomal ulceration may follow anastomosis of the stomach to any other part of the alimentary canal. I have indicated in my first lecture that by 1954, stomal ulceration had followed in over 50 per cent. of the cases in which I had carried out a gastro-jejunostomy for duodenal ulcer in 1943. In such cases at the second operation, the duodenal ulcer, although often reported as active by the radiologist, is almost invariably healed and inactive, though sometimes stenosed. The only occasions on which I have found activity in the duodenal ulcer was when there was a gastro-colic fistula or stenosis of the gastro-jejunal stoma. The jejunal ulcer is usually on the jejunal side of the stoma, near the efferent loop, but is occasionally quite apart from the stoma and sometimes multiple. If an ulcer is seen on the gastric side and is not on the stoma, it almost invariably means that the patient has had a gastro-jejunostomy for, or in the presence of, a gastric ulcer. In such cases the jejunum is usually intact, but the gastric ulcer may remain active. I have on several occasions seen both an active gastric ulcer and a healthy gastro-jejunostomy in the same field at gastroscopy.

After gastrectomy for gastric ulcer, stomal ulceration is of the greatest rarity. I had personally performed 690 gastrectomies for gastric ulcer before operating on the first case which subsequently developed a stomal ulcer, and in well over a thousand resected in our clinic, this remains the only known case. It was indeed an unusual one in that the gastric ulcer appeared in the prepyloric region on the greater curve side of the stomach. The patient was a man of 45, who had taken aspirin daily for some years. Although the ulcer almost healed under a medical regime, we felt it wiser to resect it in view of its position, and ulcer symptoms recurred within a year. In the follow-up of 611 of our gastrectomies for peptic ulcer by Mr. Colin Craig and Dr. Chippendale, there were no recurrent ulcers among the gastric ulcer resections,

but five proven and one doubtful stomal ulcer among 244 resections for duodenal ulceration—a recurrent ulcer rate of 2.5 per cent. Here again stomal ulceration following gastric resection is, in my experience, invariably in the jejunum and never in the stomach. Occasionally, post-operative deformities mimic a recurrent gastric ulcer, but I have never failed to disprove its presence by gastroscopy or by subsequent operation.

It is interesting to consider why the *stomach* does not undergo *peptic* ulceration after gastrectomy, either for gastric or duodenal ulcer. The problem is a difficult one and its solution depends on the causation of ulcer and ulcer pain. I have pointed out previously (Tanner, 1951) that during digestion there is varying intensity in the concentration of acid in different parts of the stomach after a meal, the acidity probably being higher at the upper level of gastric chyme. I believe that prolonged contact with this concentrated level may play some part in the development of ulcer, and that the time when this level comes opposite the ulcer is the time when ulcer pain occurs. After gastrectomy there is a more rapid descent of the top level of gastric chyme. The acid concentration is diminished because there is a diminished volume of gastric juice, and there is more mixing as a result of the constant inflow of biliary and duodenal juices into the gastric remnant. The jejunum, however, is more susceptible to ulceration. Possibly the unbuffered intercalary secretions play an important part in producing jejunal ulceration, which occurs where the acid concentration is greatest, near the efferent loop, and at the site of greatest weakness, the jejunal side of the suture line scar.

Stomal Ulcer following Gastro-Jejunostomy

It is well first of all to consider the treatment of stomal ulcer following gastro-jejunostomy. Plastic operations on the stoma and local excision of the stoma are of only temporary help. Restoration to

the normal by removing the gastro-jejunostomy has been practised in cases where it was found that the duodenal ulcer had healed. In such cases re-ulceration of the duodenum usually occurs. Restoration to normal is only permissible when the gastro-jejunostomy is giving mechanical, and not stomal ulcer troubles, and when it is known that the gastric acidity has fallen since the original operation. This leaves us with only two orthodox operations for anastomotic ulcer, dismantling the stoma followed by partial gastrectomy, or vagus nerve section. As I have indicated before, the results of the latter operation are not fully known and no one can yet define its indications or limitations. We can, however, state that if there is much distortion of the stoma by ulceration, or stenosis of the stoma, or stenosis of the jejunum (Figs. 20 and 21), the vagotomy should not be used. It should not be used if the stomal ulcer is fixed to the colon, for I have known of a gastro-colic fistula which only became apparent in the convalescence from vagotomy in such a case. I have only used it four times, in patients with X-ray evidence of excellently functioning stomas and minimal scarring, and in these cases the vagotomy has been successful.

In the more common case where there is deformity and scarring from a stomal ulcer following gastro-jejunostomy, or in the rare cases of persistent gastric ulcer following gastro-jejunostomy, a partial gastrectomy is the most satisfactory procedure.

A few technical points may be of interest. Division and closure of the duodenum is usually very easy because of the inactivity of the ulcer. So far as the anastomosis is concerned, I carefully cut through the line of the previous anastomosis in order to dismantle it. If one cuts exactly in the line of the scar it is almost bloodless. After temporarily suturing the opening in the stomach the jejunum is repaired. Any ulcer edge is excised from it and if there is any stenosis, a small longitudinal incision is made through the stenotic part along the bowel. The previously longitudinal opening is closed transversely. It is true that this leaves a rather ugly looking piece of bowel, but it functions perfectly, in fact it is wider than normal jejunum. Resection of the affected jejunum followed by end to end jejuno-jejunostomy is neater, it takes a little longer and is particularly indicated if there is very gross jejunal ulceration, particularly on its mesenteric side, if it is very deformed, or if its blood supply is precarious. At times an unsuspected fistula into the colon is found. The colonic opening is closed transversely. After repair of the jejunum and colon and closure of the opening in the mesocolon, a partial gastrectomy is carried out, followed by a

FIG. 20.—Operative view of grossly dilated afferent jejunal loop due to stenosed stomal ulcer.

fresh gastro-jejunal anastomosis. As gastrectomy for gastro-jejunal ulcer carries a slightly higher stomal ulceration rate than gastrectomy for duodenal ulcer, it is wise to make a higher right section in such cases.

Now of this type of operation we have carried out 106 operations as follows:

GASTRECTOMY AFTER DISMANTLING GASTRO-JEJUNOSTOMY

	Cases	Deaths
1. Jejunal ulceration:		
(a) Interval	97	1
(b) For massive haemorrhage ..	2	1
2. Gastric ulceration:		
Interval	9	0
Total:		
(a) Interval	106	1
(b) For massive haemorrhage ..	2	1

Stomal Ulcer following Gastrectomy

Gastro-jejunal ulceration following partial gastrectomy presents a more difficult problem. The first essential in such cases is to ensure that there is no technical reason why an ulcer should appear. They are as follows:

1. Pyloric section and failure to remove the pyloric antrum.
 2. The presence of a Roux form of anastomosis, short circuit between the loops, or a jejunal interposition between stomach and duodenum.
 3. Inadequate gastrectomy (e.g., Antrectomy).
- In the first type of case all that is theoretically necessary is to remove the pyloric segment. In

FIG. 21.—X-ray of another case of jejunal stenosis due to stomal ulcer. The upper shadow of barium is in the stomach and the lower in the afferent jejunal loop, 24 hours after ingestion of the meal.

practice, however, once a stomal ulcer has occurred, recurrent ulceration occurs on minimal stimulation, and so it is wise to make a new anastomosis if there is gross stomal scarring or ulceration. In the second group a Roux anastomosis should be undone and a new loop anastomosis made to take duodenal juices over the stoma once more. A jejunal transplant should be removed. If there is a short circuit of the loops the case can be treated like a stomal ulcer without a short circuit of the loops. A very low gastrectomy should be remade removing at least half, preferably two-thirds of the body of the stomach.

Stomal Ulcer after Gastrectomy without Technical Faults

Until recently we had no means of dealing with this condition apart from higher and higher resections, until in some cases almost a total gastrectomy was made. Plastic operations on scar deformities are useless, they last but little longer than the patients' convalescence, for they do not affect the basic tendency to ulceration.

During the last seven years a new method of dealing with these has arisen, namely vagal re-

section, and to my mind it has entirely altered the outlook of these unfortunate patients. In order to attain success from this operation two things are advisable.

1. The vagus must be well seen and all its branches identified and resected as previously described. The operation may be a little more difficult because of the adhesions and because the nerve becomes more slender and more fragile after gastrectomy.

2. If there is gross stomal deformity or an actively penetrating or a fixed ulcer, it is helpful to excise or refashion the stoma. An ulcer scar is always more prone to re-ulcerate than an operative suture line, probably because its centre is composed of thin epithelium covering fibrous tissue, it is thus more rigid and possibly produces less antipeptic factor.

However, many cases have done well without further resection of the previous anastomosis. In order to deal with a damaged stoma, and to confirm the diagnosis, and because it is generally satisfactory, I usually use an abdominal approach, but under certain circumstances a transthoracic approach is advisable—(a) where a previous

FIG. 22.—(1) Represents a congenital anomaly with gastric mucosa lining the lower oesophagus.
(2) Is the commonest cause of reflux of gastric juice into the oesophagus.

incomplete abdominal vagotomy has been done—(b) where there have been multiple operations and an X-ray shows good stomal function.

Case of C.J., aged 46, who came to me in 1950 with the following history: 1944, Onset dyspepsia; 1947, Partial gastrectomy (Military Hospital); 1949, Perforation stomal ulcer; 1949, Six months later—second perforation of stomal ulcer; 1949, vagotomy; 1950, three months later—third perforation of a stomal ulcer.

He had no dyspepsia before either perforation and was on a strict diet. He was very thin, 105 lb. only. Now I suspected that the vagotomy may well have been incomplete, though no data of the technique used had been given. An insulin test meal was carefully done with X-ray control of the site of the stomach tube. It showed that the vagus was functioning quite normally. Therefore I decided to repeat the vagotomy. X-ray and gastroscopy showed a good functioning gastro-jejunal stoma. I did not want to re-enter the abdomen as he had had three perforations repaired and a vagotomy within 12 months, and so I did a transthoracic vagotomy. The posterior gastric nerve appeared to have been missed at the previous

operation. A subsequent insulin test meal now confirmed that the vagotomy was complete.

Three years have now passed since this operation with no further trouble at all.

This demonstrates the great importance of careful technique and confirmation of the result by an equally careful insulin test meal.

To sum up our results to date in the treatment of stomal ulceration following partial gastrectomy.

No. of cases 20				Proved Recurrent Ulcer
Operation	No.	Deaths		
Vagotomy only	11	0		0
Vagotomy and excise ulcer edge	3	0		0
Vagotomy and remake stoma	6	1		0
Total	20	1		0

The length of follow-up is in all cases over a year, two have passed over five years. In my experience, stomal ulceration after gastrectomy once having started gives little respite, and so I regard even a one-year follow-up as significant.

Those who have treated this type of case know

of the unremitting and severe nature of the disease. I believe that in vagotomy we have a most valuable and dramatic weapon against it. To those who have failed to get satisfactory results I would respectfully suggest that they look to the vagotomy technique and consider the advisability of making a new stoma in the cases where the ulcer is deeply penetrating or the stoma badly deformed.

Peptic Oesophagitis and Oesophageal Ulcer

This diagnosis is made with ever increasing frequency of late years. There is no doubt that much of the increase is more apparent than real, and is due to more accurate diagnosis, together with increasing awareness of the condition. We owe much of our present knowledge to Harrington of the Mayo Clinic, Dunhill and Barrett of London, and to Allison and Johnstone of Leeds. Only ten years ago, few radiologists made special examinations to discover the presence of oesophageal reflux or hiatus herniation, whereas now it is almost a routine addition to the radiological examination of the stomach and duodenum. Endoscopists have been made familiar with the typical oesophagosopic appearance of peptic oesophagitis, the lax gullet full of digestive secretions, the mobile and often patulous cardiac orifice, the congested, granular and sometimes ulcerated and stenosed lower oesophagus. However, peptic oesophagitis may, like other forms of peptic ulceration, be changing in frequency and some of the increase may be real. Evidence at present appears to show that peptic oesophagitis may be due to either (Fig. 22):

1. Reflux of peptic juice into the oesophagus as a result of (a) an oesophageal hiatus hernia, (b) an incompetent cardia, (c) a surgical anastomosis of the oesophagus to the stomach or surgical damage to the cardia.

2. A congenital anomaly, either (a) the presence in the lower oesophagus of gastric mucosa so that the ulcer is really in or at the edge of gastric tissue in an organ which externally resembles oesophagus, or (b) a congenitally short oesophagus with stomach high in the chest. These two conditions are basically similar and need similar treatment, despite the fact that a gross hiatus hernia may be present in the second form.

It seems that the cause of the disease is prolonged contact of gastric juice with the oesophageal mucosa. The changes in the oesophagus have been very aptly likened to those which occur in the skin around a gastric fistula. There are, however, many patients with severe oesophageal reflux who have no oesophagitis and who may not even have the most typical symptom, namely heartburn. It may be as in the case of duodenal ulceration,

FIG. 23.—Simple stricture of lower oesophagus, following reflux oesophagitis due to hiatus hernia.

that ulceration can be resisted for years until some factor, perhaps a period of excessive peptic secretion, or a period when there is excessive contact of gastric juice with the gullet, or local damage to the oesophagus occurs which sets off the cycle of ulceration and healing, so typical of ulcer disease. It is notable that some patients date their symptoms from an operation carried out in the Trendelenberg position, or associated with much vomiting, or they date it from a pregnancy, at which time there is at least a temporary period of increased oesophageal hiatus strain. McCreath (1953) has pointed out that many women with heartburn, and erratic symptoms of discomfort in the neck and dorsal regions, globus hystericus, etc., have unjustifiably been considered hysterical while in fact suffering from severe oesophageal reflux.

The main complications of peptic oesophagitis are stricture formation (Fig. 23) and anaemia from persistent bleeding. Acute oesophageal obstruction from impaction of a food bolus or fruit stone above the narrowed area sometimes occurs, and acute perforation is a great rarity. Occasionally hiatus hernia is associated with a gastric or duodenal ulcer. I have found hiatus herniation present in 21 of my cases of carcinoma in the cardia region.

It is possible that reflux oesophagitis predisposes to cancer in this region.

In prophylaxis it is important to avoid gastro-oesophageal anastomosis for cardiospasm or for simple oesophageal stricture.

Treatment of Peptic Oesophagitis

I do not propose to say anything about the medical management.

Surgical treatment is at present in a somewhat confused state, because while we all know a certain cure, namely end-to-end anastomosis of the oesophagus to a Roux loop of jejunum, this is too drastic for most of the cases and simpler and safer methods are under trial. I will try to give a résumé of the methods we have used and of their results. The various methods can be divided into four groups: (1) operations to prevent reflux; (2) operations to reduce the digestive activity of the regurgitated juice; (3) resection of the involved area of the oesophagus; and (4) by-passing of the affected oesophagus.

1. Repair of the hiatus hernia. It is probable that the development of a sliding hiatus hernia changes the mechanism of the cardia, making it incompetent and so is the primary cause of most cases of reflux oesophagitis and oesophageal ulcer. Repair of the hernia would seem to be the most logical step whenever possible. It cannot be done if the gullet is too short and is fixed by chronic ulceration—for in such cases the ulcer may rupture during manipulation and the cardia may not reach the diaphragm. It is, however, remarkable how often an oesophagus apparently 5 to 10 cm. shorter than normal will reach the oesophageal hiatus in the diaphragm without difficulty. Repair of the hiatus is, of course, useless in the cases where stomach mucosa extends up into the gullet as a congenital anomaly.

Repair of the hiatus hernia demands reduction, and then repair of the two stretched retentive structures. The first is the phreno-oesophageal ligament which normally passes from under the diaphragm to the cardia region and is analogous to the transversalis fascia. The second and most important is the muscular margin of the oesophageal hiatus in the diaphragm. The oesophageal opening in the diaphragm becomes somewhat kidney-shaped when herniated because of the presence posteriorly of the aorta. In the past it was the custom to repair the hiatus anterior to the oesophagus. This type of repair gained a bad reputation for recurrence, for the hiatus was merely made triangular with a broad base on its aortic aspect, through which recurrence rapidly occurred. Nowadays we find it infinitely more satisfactory to make a posterior repair, strengthening the hiatus on its aortic side and reducing it to a

narrow anterior opening near the dome of the diaphragm. This renders the course of the oesophagus a little shorter, a great advantage. Following this the phreno-oesophageal ligament is not *repaired*, for it is too thinned to repair in such cases, but it is replaced by a row of unabsorbable sutures between the stomach at the level of the cardia, and the inferior surface of the diaphragm (not merely the peritoneum) around the new oesophageal hiatus.

As most of the early inefficient repairs were by the abdominal approach, this route fell into some disrepute and many surgeons came to prefer the transthoracic route. The transthoracic route is advisable if there is a fixed or penetrating ulcer, if there is a suspicion of oesophageal malignancy, or if there has been a previous abdominal repair which has failed, and perhaps in exceptionally obese patients. In all other cases, I much prefer an abdominal approach. It has a lower morbidity and mortality rate and it is to be remembered that many of the patients requiring surgery for this condition are elderly, obese and are poor surgical risks. It has been stated that it is not possible to get an adequate view of the hiatus by the abdominal approach. This is not so if the correct approach is used. A high mid-epigastric incision is made, and the xiphoid process is removed. The anterior diaphragmatic fibres are divided. The left triangular ligament of the liver is then divided and the liver pulled over to the right, when with adequate relaxation and correct manipulation, a good view of the region of the hiatus is usually obtained. If it is not obtained then it is possible to improve the view by division of the sternum through its middle, up to and into the fourth intercostal spaces. I have used this addition for many conditions, though as a matter of fact have not yet found it necessary in dealing with a hiatus hernia.

The stomach and other contents of the hernial sac are now reduced into the abdomen. After making an incision through the peritoneum and ligament in front of the oesophagus, a rubber retractor, such as I use for vagotomy is passed through the lesser sac and over the gastric fundus to control the stomach. The hernial sac is withdrawn from the posterior mediastinum—it may be mainly anterior or mainly posterior to the oesophagus. The peritoneal sac with its coat of stretched phreno-oesophageal ligament is cut away and discarded. The stretched crurae of the diaphragm can now be exposed and cleared of all fatty and peritoneal tissue. A stitch is placed in the anterior extremity of the margin of the oesophageal hiatus as a tractor. Sutures of non-absorbable material are now carefully placed through large bites of each crus of the diaphragm.

FIG. 24.—Abdominal repair of oesophageal hiatus hernia. There is a traction suture on the anterior part of the hiatal edge. Sutures have been placed between the margins of the hiatus, posterior to the oesophagus. The right crus is clearly seen.

to draw them together behind the oesophagus (Fig. 24), from the aorta upwards and forwards until the oesophagus is snugly surrounded. It is important to have no shreds of fat or other tissue between the crurae when they are sutured together. The material I use at present is floss nylon. The oesophagus is now fed into the mediastinum until the cardiac orifice lies just below the diaphragm. It is unwise to leave a great length of gullet inside the abdomen, for this will alter the important oesophago-gastric angle of entry—the importance of which has been shown by Barrett. Next, non-absorbable, fairly deep sutures are placed between the cardia region and fascia below the diaphragm round the new oesophageal hiatus. The liver is replaced and the wound closed.

The Results of the Abdominal Repair of a Hiatus Hernia

Repairs posterior to the oesophagus by the abdominal route have been made in 64 cases, one-third being aged over 60. All have been traced, and of 35 operated on between one and four years ago, the present state is as follows:

POSTERIOR REPAIR OF HIATUS HERNIA, ABDOMINAL ROUTE, 1 TO 4 YEARS, POST-OPERATION

	No.	Per cent.
Satisfactory	23	65.7
Improved	7	20.0
I.S.Q.	5	14.3

The reports on the X-rays of 25 patients done over a year ago are interesting when compared with the clinical state. It should be noted that a higher proportion of patients with a poor result have an X-ray examination.

X-ray Report	Clinically		I.S.Q.
	Satis- factory	Im- proved	
No hiatus hernia, no reflux	12	—	—
Hiatus hernia, no reflux ..	2	3	—
Hiatus hernia and reflux ..	1	2	3
Reflux but no hiatus hernia	1	—	1

This shows that there is a tendency for the X-ray and clinical results to coincide roughly, and it is noticeable that all cases without reflux or recurrent hernia on X-ray also had satisfactory results.

Simple phrenic division sometimes relieves

A

B

C

D

FIG. 25.—'Lower partial gastrectomy' (Polya operation) for stenosing peptic oesophagitis. Case M.W. A—July 20, 1949, before operation. B—February 22, 1951, 18 months after operation. C—April 5, 1951. D—February 2, 1954. Symptoms now slight.

FIG. 26.—Upper partial gastrectomy for stenosing peptic oesophagitis. A—Pre-operative X-ray of barium swallow. B—The specimen with only a short segment of ulcerated oesophagus. C—Post-operative barium swallow. Note the absence of any post-vagotomy delay at the pylorus, which has been treated by pylorotomy.

some symptoms of hiatus hernia. The left phrenic is divided despite the fact that most of the fibres round the oesophagus are supplied by the right phrenic nerve. Its effect is not to relax the oesophageal ring, but probably rather by elevation of the left diaphragm to lessen the intra-abdominal pressure and to raise the pressure in the mediastinum so that the hernial contents remain more readily in the abdomen under the diaphragm. It will not cure reflux and is inadequate for oesophageal ulcer. We used it in four senile fragile cases with reflux and discomfort from incarceration of the fundus in the thorax and all declare themselves improved.

2. Reduction of the volume and digestive powers of the regurgitated peptic juices. In 1949, Wangenstein advocated lower partial gastrectomy—as for duodenal ulcer—in the treatment of stenosing peptic oesophagitis. It would be a very useful method if satisfactory, particularly as not a few of the cases also have duodenal ulceration. In a series of five cases of peptic oesophagitis dealt with in this way, three are satisfactory and two are improved (Fig. 25 a, b, c and d). The progress appears to be steady but fairly definite. Partial gastrectomy was used in four other cases of associated duodenal ulcer and hiatus hernia with some reflux oesophagitis, and all have done well.

3. Resection of the Oesophagus and Upper Stomach. If simple resection of the strictured part of the oesophagus is carried out, the reflux will continue more freely than ever and despite the vagal section, oesophagitis will recur. The minimum of *resection* must be of the stricture and a good proportion of the body of the stomach, preferably uniting the gullet to the pyloric part of the stomach (Fig. 26 a, b and c). This is a satisfactory procedure on the whole. In view of the vagotomy it will add to the patient's comfort if a gastro-jejunostomy or pylorotomy is added. (Tanner 1951.) The oesophagus in such cases is usually fibrotic just above the stricture and holds sutures much better than in carcinoma cases. The morality of this operation is less than that of resection for carcinoma.

4. Oesophago-jejunal Anastomosis. The most certain way of curing oesophagitis is by transecting the oesophagus and uniting it to a mobilized Roux loop of jejunum in the thorax. Resection of the strictured part of the gullet is advisable to prevent continued action of gastric juice on it, but total gastrectomy need not necessarily be carried out.

In the post-operative cases, and in the cases with true congenital shortening of the gullet (a very rare anomaly) and in cases in which gastric mucosa replaces the lower oesophagus—the only satisfactory treatment is by resection of the lower oesophagus, uniting the oesophagus either to the

pyloric part of the stomach or to the jejunum. These operations have not the mortality and need not be so extensive as the cancer operations—and they are often easier to carry out.

Case of L.B. An Italian female had cardio-spasm dating from her teens. At the age of 24 an oesophago-gastric anastomosis was made in Padua in 1949. Following this the swallowing became perfect, but she began to suffer from severe retrosternal pain radiating into the back, and acid regurgitations.

On oesophagoscopy, the lower dilated oesophagus was seen to be filled with gastric juice and there was ulceration at 38 cm. Medical treatment gave no relief and so in October 1951, an abdominal Billroth I partial gastrectomy was carried out to lower the gastric acidity. Relief from this was incomplete and subsequent oesphagoscopy still showed a congested bleeding lower oesophagus. Therefore, in January 1952, the lower oesophagus was exposed and removed by a transthoracic route, and the end of the oesophagus connected to a mobilized Roux loop of jejunum. Eighteen months after this, she had only minor troubles.

Results of Treatment

I am giving, of course, an interim report. I hope to be more certain of the relative efficiency of the various procedures in five years' time, but a review at the present time is valuable.

Perforation of Peptic Ulcer

All of us present at this lecture tonight have been brought up to believe that early surgical closure is the best treatment for acute perforation of a peptic ulcer. We know how it has greatly reduced the mortality of perforated ulcer compared with the days before Heusner first successfully closed a gastric ulcer perforation in 1892. We have learned that avoidance of delay in effecting closure is the most important single factor in reducing the mortality. Latterly, we have found the mortality of this complication drop to a new low level with greater care in pre-operative preparation of the patient, with better anaesthesia and with the introduction of the antibiotics. For many years past, an occasional patient has been treated with success by conservative measures because he was too ill or arrived too late for surgery, or because the diagnosis was missed, or surgery was unavailable. Occasional cases have also been treated by immediate gastric resection because of associated massive ulcer haemorrhage, or because the ulcer was malignant, and I think that at times we have been agreeably surprised to find how well they did. Nevertheless, with simple suture giving even improving results, it has required some temerity for the advocates of routine conservative treatment

of perforation on the one hand, or of routine gastrectomy on the other hand, to try to demonstrate the good points of these methods.

Conservative Treatment of Acute Ulcer Perforation

Now as far as conservative treatment is concerned, it has been shown that this will succeed in what Mr. Hermon Taylor (Taylor, H., 1951) described as one of its objects, namely to avoid those complications which are associated with the *operation* for perforated ulcer, e.g. wound infection, disruption or hernia, anaesthetic and pulmonary complications and post-operative adhesions. Added to this it has been shown that in the hands of devoted clinicians a low mortality comparable with that obtained by simple suture may be obtained. Nevertheless the method has not been universally adopted, partly because it has not *lowered* the mortality of perforation and partly because of the great demands on the nursing and medical attendants for prolonged and anxious observation. There is also always the possibility of a disastrous misdiagnosis of the cause of the acute abdominal catastrophe. My present opinion is that the method is unsuitable for routine usage. There is no doubt, however, that we owe a great debt to the pioneer work of those surgeons who have improved the technique of the conservative, or as Hermon Taylor calls it, the 'aspiration treatment' of ulcer perforation. By their work they will have saved many lives among those patients with acute ulcer perforation, for whom either by reason of unfitness for anaesthesia or by geographical situation, surgery is inadvisable or unavailable.

Immediate Gastrectomy for Ulcer Perforation

In 1951 in the Macarthur lectures, I remarked (Tanner, 1951): 'So far as urgent gastrectomy is concerned . . . Many figures have been presented showing that resection can be done with a low mortality. So far, however, and I have looked carefully to find them, no figures have appeared to show that the over-all mortality of all cases admitted with perforation of peptic ulcer has been lowered or even kept level by performing gastrectomy on some of them. . . . I long to see some straightforward and unbiased figures on this subject, because it is worthy of trial.' Since that time, we have had no proof that immediate gastrectomy will lower the over-all mortality of ulcer perforation, and indeed when such low mortalities for simple suture as those achieved by Avery Jones and his colleagues at the Central Middlesex Hospital are put forward, it is obviously going to be very difficult to better them. Nevertheless, in view of the fact that some 6 per cent.

of perforated apparently simple gastric ulcers turn out to be malignant, and because of the increased post-operative complication rate for perforated *gastric* ulcer, we have, since 1950, carried out immediate gastrectomy in the treatment of perforation of large pre-pyloric or mid gastric ulcers, in addition to what might be called the orthodox group, namely sealed off perforations with only localized peritonitis, concomitant haemorrhage and perforation and perforated gastric carcinoma. It has since been shown, notably by Professor A. G. R. Lowdon when he was at Edinburgh, that immediate gastrectomy in expert hands does not appear to lead to a higher mortality than simple suture of perforation (Lowdon, 1952).

J. Gilmour (Gilmour, 1953) in a recent review of his cases confirmed the increased mortality of perforation with increasing age and with delay in surgical intervention, but he also made two other significant observations. The first was that the mortality chiefly affected the patients with *chronic* peptic ulcer. The second was that in a late follow-up of cases of ulcer perforation treated by simple suture, one-quarter of the acute ulcer perforations had severe relapses of symptoms, and three-quarters remained well, but of 65 chronic ulcer perforations all had relapsed, two-thirds requiring further operative treatment. This indicated that unless immediate gastrectomy could offer a significantly lowered mortality from perforation it was wiser to continue with simple suture in acute ulcer cases. In the case of chronic ulcer, however, immediate gastrectomy would have much in its favour in view of the poor late prognosis.

After careful weighing-up of these points we decided at St. James' Hospital, London, in May 1953, to undertake immediate partial gastrectomy under the following conditions.

1. In general the patient should be between the ages of 30 and 60. Exceptions to this might be the patient under 30 with symptoms dating back over many years, and the patient over 60 in excellent general condition.

2. Length of history: (a) Gastric ulcers—Resect all which fall into the above age qualification, provided the patient is considered fit enough; (b) Duodenal ulcers—Resect when a patient under the age of 45 has a history of at least one year and a patient over 45 with over six months' dyspeptic symptoms. Resect in all cases where there is evidence of bleeding, either past or present. Resect in all cases where there is evidence of pyloric stenosis.

3. The general condition of the patient will naturally influence the decision as to treatment by resection, simple suture or intravenous drip and gastric suction.

4. Perforated stomal ulcer—Simple suture in all cases.

5. Perforated gastric carcinoma—Resect if operable and the patient fit enough, otherwise simple suture combined, if thought advisable, with appropriate palliative procedure.

The period under review, six months, is too short to make a significant effect on our over-all mortality. Our clinical impression is that contrary at least to my own expectations, the over-all mortality is diminished. A clinical impression is always more reliable when supported by hard mortality figures. I give our figures for the four years since October 1949. The over-all surgical mortality is 8.6 per cent. but including the 18 cases too ill for surgery it is 14.5 per cent. The one death following gastrectomy was a patient who died of coronary thrombosis three weeks after a gastrectomy for concomitant haemorrhage and perforation.

PERFORATED PEPTIC ULCER—IMMEDIATE RESULTS

	No. of Cases	Operation Deaths	Mortality Per cent.
Simple suture, P.D.U.	133	10	7.5
Simple suture, P.G.U.	20	4	20.0
Simple suture, P.G.J.U.	5	1	20.0
Emergency gastrectomy:			
P.D.U.	25	1	4.0
P.G.U.	2	—	—
Total (Surgical)	185	16	8.6
No operation	18	13	72.0
Total (all cases)	203	29	14.5

However, perhaps one of the most significant occurrences concerns the very remarkable improvement following urgent gastrectomy in two patients with late perforations and frank pus in the peritoneal cavity. The second case merits recounting.

A.B., aged 63, an obese bronchitic man with old rheumatic heart disease and a long ulcer history was admitted in poor condition 24 hours after perforation. His systolic blood pressure was around 60 mm. Hg. but after $1\frac{1}{2}$ hours resuscitation he was recovered enough for surgery. His was a type of case in which simple suprapubic drainage of the peritoneal cavity might have been considered the optimum treatment by many. However, a severe perforation of a vast stenosing posterior penetrating duodenal ulcer was discovered. In view of the extreme difficulty of closure of the perforation, my colleague, Mr. Andrew Desmond, carried out a partial gastrectomy. His condition the next day was considerably better and he has continued to make an excellent recovery.

It should be added that about a third of these

gastrectomies for acute perforation have been carried out by surgical registrars, though of course not until they have had very good experience of upper abdominal surgery, and they have unusual opportunities of gaining experience of this work at St. James' Hospital. I need hardly add that in view of the very favourable progress of our cases, we are in favour of continuing the present policy of resection of at least the chronic ulcers in the treatment of ulcer perforation.

Massive Gastro-Duodenal Bleeding as a Result of Peptic Ulceration

I pointed out in my first lecture that Sir Gordon Gordon-Taylor in his 1945 Lettsomian Lectures had enunciated the principles of selection of cases for surgical treatment and had discussed the results of surgery. I can add but little on these aspects, and so instead will give some of our up to date results in the treatment of this complication.

Some of you may know that at St. James Hospital, we have since 1941 tried to stabilize our medical management of massive gastro-duodenal bleeding, but have from time to time after careful consideration, varied our indications for surgery and then finally reviewed the results obtained during the different periods (Tanner, 1949). By the end of 1949 we found that our best results were obtained by early and fairly frequent surgery—to be exact, surgical intervention in 60 per cent. of the cases. It occurred to us that the improvement might be in part due to advances in chemotherapy and increases in the numbers of our senior staff since the war. Therefore in 1950 and 1951 we reverted to 'selective surgical intervention using surgery in only 19 per cent. of the cases. By this term we mean that certain criteria which are known to indicate a dangerous form of bleeding are also indications for early surgery. In 1952 and 1953, we have reverted again to a modified form of the Finsterer (Finsterer, 1947) régime. By the Finsterer régime, we mean roughly that if a patient is admitted with massive gastro-duodenal bleeding and has a history of chronic peptic ulceration, he is operated on at once. Finsterer argues that such early surgery carries little more risk than interval surgery, and the patient with chronic ulceration needs an operation anyhow. If, on the other hand, the patient has only a brief history or no history of ulceration, he is treated conservatively, but if bleeding is repeated or continues, exploratory operation is at once undertaken.

By 'Finsterer over 45' we mean much the same as the Finsterer régime, except that in view of the extremely low mortality in cases of haematemesis under 45, we only operate under that age if the haematemesis is repeated in the hospital, even

FIG. 27.—Graph showing the varying numbers of cases of haematemesis and melaena admitted to St. James' Hospital, month by month.

though the patient may have a chronic ulcer history.

Before proceeding to our results it is well to see the type of case we treat, because it will at once be seen that we deal with a high percentage of gastric ulcers—the type in which surgery is most often required.

CASES OF MASSIVE HAEMATEMESIS AND MELAENA, 1941-53				
Gastric Ulcer	..	..	..	649
Duodenal Ulcer	..	..	..	478
Gastro-jejunal Ulcer	..	..	..	63
Oesophageal U.	..	..	..	8
Gastritis	..	..	..	97
Hiatus hernia	..	..	..	18
Portal hypertension	..	..	..	41
Carcinoma of stomach	..	..	..	45
Carcinoma of oesophagus	..	..	..	1
Simple gastric neoplasm	..	..	..	9
Gastric diverticulum	..	..	..	1
Aneurysm aorta	..	..	..	1
Pancreatitis	..	..	..	2
Carcinoma of pancreas	..	..	..	3
Uraemia	..	..	..	1
Swallowed foreign body	..	..	..	1
Retrograde intussusception	..	..	..	1
Uncertain	..	..	..	71
				1,490

In addition, it will be seen that many of the patients are elderly and this is another factor to bear in mind when considering our results. In our last two year series 55 per cent. of the cases were aged over 60 years of age, 27 per cent. over 70 and 5 per cent. over 80. The influence of age on mortality can also be seen. In our last series the mortality of massive ulcer haemorrhage under the age of 70 was 3.6 per cent. but over the age of 70 it was 25 per cent.

It is interesting to see the effect of external circumstances on the incidence of ulcer bleeding—and so I have made a graph of the varying numbers of cases of haematemesis admitted to St. James' Hospital, month by month (Fig. 27). You will notice in particular the abrupt rise at the time of the commencement of the 'fly-bomb' attacks on London during the war. The sudden drop which follows is associated with a temporary evacuation of many wards when the hospital was damaged and the increased evacuation from London later no doubt also had some effect. There is also an interesting peak in December, 1952.

I would now like to show you the present state of our long term results in varying our indications for surgery from time to time. We usually

GASTRO-DUODENAL HAEMORRHAGE, 1941-53

Peptic Ulcer and Gastritis Groups

(Incidents of Bleeding)

Period	No. of Cases	Aged over 60 Per cent.	Operated on Per cent.	Over-all mortality	Average age at death Per cent.
I. 1941-1943. Operations avoided until late	193	33	5	10	62
II. December 1943-December 1947. Selective intervention	372	34	12	12	64
III. January 1948-December 1949. 'Finsterer'	215	43	59	7.5	65
IV. January 1950-December 1951. Selective intervention	274	46	19	10	70
V. January 1952-December 1953. 'Finsterer over 45'	266	55	30	9.5	69

change—if we think a change worthy of trial, at the end of December after a two year period.

Comments on Results

What do these figures show? First of all, it is evident that our patients with gastro-duodenal bleeding are of increasing age year by year. In particular you will notice the jump in average age of patients treated from 34 per cent. aged over 60 to 43 per cent. between periods two and three. I have pointed out the remarkable increase in mortality found with increasing years, no matter whether you take 50, 60, 70 or 80 as the dividing line. Indeed, we have not lost one patient under 40 in the last 1,000 ulcer and gastritis cases, and the one we had aged 40 was a man who steadfastly refused surgery until he became unconscious. Therefore I regard the improvement between Groups II and III as very significant, bearing in mind both the increased age of the patients and the lowered mortality.

Some of this improvement may have been due to the ending of the war and to the increasing availability of penicillin. It was not due to increased availability of blood—which we had in adequate supply. It was not due to improved anaesthesia, for we have stabilized that variable factor by using local anaesthesia throughout the whole period.

Reversion to selective surgical intervention in 1950 increased the over-all mortality slightly, though there was still a creep-up in average age. A return to 'Finsterer over 45' has slightly diminished our mortality, but this group contains a very high number of octogenarians and you will see that the average age of the patients has risen abruptly and 55 per cent. are aged over 60. Therefore I again regard the reversion to more frequent surgery as a significant improvement in our results.

No doubt our better results obtained when we greatly widen the indications for surgery are in part due to the advanced ages of the patients—the oldest series, I believe, ever recorded. It is also bound up with the fact that a high percentage of

the bleeding ulcers are gastric—a type carrying higher mortality, and in which surgery is particularly advisable.

Diagnosis of the Cause of Bleeding

A knowledge of the cause of bleeding is of the greatest value in deciding whether to operate on a particular case. It will be seen from my table of all the causes of bleeding, that in over 50 per cent. of the cases, the cause lies in the stomach. Therefore in all cases in which immediate surgery is not considered necessary, and indeed in some cases prior to immediate surgery, a gastroscopic examination is made within one to three days of admission to hospital. In addition to good views of the stomach being obtained in most cases, air is gently blown into the gullet during extraction of the instrument, excellent views of any oesophageal varices may be obtained, and at times hiatus hernia can be diagnosed. It is not, of course, a reliable instrument in diagnosing oesophagitis because a little bleeding will obscure the view, and only small areas are seen. If an ulcer is present in the stomach, its position, size and depth and chronicity can often be judged. The ulcer may still be oozing, or the crater may be filled with clot. Often the sealed vessel end can be seen. On one occasion we saw an aneurysm in an ulcer crater ballooning up with each heart beat, but this was a case which had not yet bled. Furthermore, if an atrophic gastritis is seen, we know we can exclude duodenal ulceration as a cause of the bleeding. In a number of the cases where the causative lesion is not evident, an atrophic gastritis is found. At operation—which we always conduct under local anaesthesia, the cause of the bleeding is usually evident. It is not uncommon to find that the ulcer is small and shallow and containing a vessel end almost as big as the ulcer crater. Similar cases have been described as cases of rupture of atheromatous gastric vessels, but I believe they are in reality gastric ulcers which have almost healed but there is failure of epithelialization over the sclerotic vessel—which acts almost like a foreign body in preventing healing. In

FIG. 28.—Progress of aneurysm in duodenal ulcer crater to a fatal termination, despite rapid healing of ulcer after partial gastrectomy in which the duodenum and antral muscularis were preserved. There is a probe through the rupture in the aneurysm.

such cases the most careful examination of the stomach and duodenum must be made, not only with the fingers but with the eyes. Sometimes only the bristle-like vessel end or a little local rigidity in the wall can be felt, or a fine local scar or blush with local glandular enlargement can be seen. The upper part of the posterior wall of the stomach must be examined with especial care.

If an ulcer is discovered a partial gastrectomy should be carried out. Local resections are often as time consuming and as difficult as a gastrectomy—it is difficult to excise the whole edge, and late recurrence is always a likely possibility. I have used sleeve resection only once and found two ulcers. Care must be taken in cases of multiple ulceration that the highest ulcer is removed.

At times, but in my experience extremely rarely—the ulcer cannot be found despite the most careful search, and a presumptive diagnosis of gastric bleeding is made. In such cases, provided the bleeding had been truly massive, then a partial gastrectomy followed by gastro-jejunal reconstruction should be done. At times a small ulcer will

be found in the specimen, or several small erosions. Even if some of the gastritic erosions remain in the fundus the bleeding will lessen because of the diminished blood supply to the fundus consequent on the gastrectomy. I have never found it necessary to do a total gastrectomy for a non-malignant bleeding gastric lesion.

Not infrequently it will be found at operation that bleeding from the ulcer is temporarily arrested. The problem arises as to whether in such an eventuality it is safe to leave a hazardous-looking duodenal ulcer in the hope that, cut off from the gastric chyme, bleeding would not recur and the ulcer would rapidly heal. Now I believe that this is a reasonable procedure to follow in most cases, though I was unfortunate on the only occasion that I followed it.

H. T., aged 60, was admitted to St. James' Hospital from a mental hospital, having had recurrent haematemesis and melaena for fourteen days. He had schizophrenia, a large bed-sore, and had attempted suicide. We operated because bleeding was repeated after transfer to us. A

huge posterior penetrating duodenal ulcer involving the region of the bile ducts was found. The prepyloric region was opened and it was found that clear bile and no fresh blood came back from the duodenum, and so presumably the bleeding had ceased. After removing the antral mucosa, the sero-muscularis of the antrum was closed, and a partial gastrectomy performed. Following this he improved day by day, but on the eighth day, massive melaena recurred. I felt that the ulcer could hardly be active after this space of time and so we treated him conservatively thereafter. He died of repeated bleeding about a week later.

At autopsy, the ulcer indeed was found to have practically healed (Fig. 28), the small unhealed part being entirely occupied by a ruptured aneurysm, the rupture of which had no doubt led to the repeated bleeding.

Another conservative method for bleeding posterior duodenal ulcer favoured at times and which we used for a period, was duodenotomy, arrest of bleeding by suture together of the ulcer edges—followed by gastro-jejunostomy. I used this on eight occasions, but in two patients, the bleeding recurred and one died of it. I do not use this method now.

After recounting my experiences with the various régimes in the treatment of ulcer haemorrhage, you will see that I am inclined to favour surgery. That does not mean that I am advocating surgery

universally. There is a considerable variation in the type of case treated in different places, and our preponderance of elderly gastric ulcer cases makes surgery particularly suitable. It is not without significance that we have such a volume of work that we are able to keep in practice for these operations.

I have gone over a very wide field and so have, perhaps, only been able to give an over-all impression of our present practices in dealing with peptic ulcer and its complications.

BIBLIOGRAPHY

- BRAIN, R. H. F., and STAMMERS, F. A. R. (1951), *Lancet*, **i**, 1137.
 CAPPER, W. M., and BUTLER, T. J. (1951), *Brit. med. J.*, **ii**, 265.
 FINSTERER, H. (1947), *Wien, med. Wschr.*, **97**, 3.
 GILMOUR, J. (1953), *Lancet*, **i**, 870.
 GORDON-TAYLOR, G. (1945), *Trans. med. Soc. Lond.*, **64**, 197.
 LOWDON, A. G. R. (1952), *Lancet*, **i**, 1270.
 MCCREATH, N. D. (1953), Personal communications.
 MIMPRISS, T. W., and BURT, St. J. M. C. (1948), *Brit. med. J.*, **ii**, 1095.
 MORONEY, J. (1951), *Lancet*, **i**, 993.
 POLLARD, H. M., BOLT, R. J., RANSOM, H. K., and ORE-BAUGH, J. E. (1952), *J. Amer. med. Ass.*, **150**, 1476.
 QUINN, W. F., and GIFFORD, J. H. (1950), *California Med.*, **72**, 18.
 SHANKS, S. C. (1952), *Trans. med. Soc. Lond.*, **67**, 136.
 SWYNNERTON, B. F., and TANNER, N. C. (1953), *Brit. med. J.*, **ii**, 841.
 TANNER, N. C. (1949), *Ibid.*, **i**, 110.
 TANNER, N. C. (1951), *Langenbeck's Arch. u. Deutsch Z. Chir.*, **267**, 369.
 TANNER, N. C. (1951), *Edin. med. J.*, **58**, 288.
 TANNER, N. C. (1952), *Ann. Roy. Coll. Surg.*, **10**, 45.
 TAYLOR, H. (1951), *Lancet*, **i**, 7.
 WANGENSTEEN, O. H. (1949), *Surg. Gynec. and Obstet.*, **88**, 509.

References continued from page 576—Simon Behrman, B.Sc.(Hon.) Lond., M.R.C.P., Lond.

17. FETTERMAN, G. H., and MORAN, J. J. (1941), *Arch. Path.*, **32**, 251.
18. CORDAY, E., ROTHENBERG, S. F., and PUTNAM, TRACY J. (1953), *Arch. Neurol. Psych.*, **69**, 551.
19. WOLF, H. G., and LENNOX, W. G. (1930), *Ibid.*, **23**, 1097.
20. WOLF, H. G. (1936), *Physiol. Rev.*, **16**, 545.
21. BEHRMAN, SIMON (1951), *Arch. Ophthalm.*, **45**, 458.
22. WALSH, F. B., and KING, A. B. (1942), *Ibid.*, **27**, 1.
23. ASKENAZY (1952), *Folia Psych.*, **55**, 55.
24. Personal Case.
25. STROBOS, R. R. J., and MOUNT, L. A. (1953), *Arch. Neurol. Psych.*, **69**, 118.
26. SWEET, W. H., and BENNETT, H. S. (1948), *J. Neurosurg.*, **5**, 178.
27. DENNY-BROWN, D. E. (1951), *M. Clin. Nth Amer.*, **35**, 1457.
28. DUNNING, H. S. (1953), *Arch. Neurol. Psych.*, **70**, 531.
29. HYLAND, H. H. (Case 3) (1933), *Ibid.*, **30**, 342.
30. KING, A. B., and LANGWORTHY, O. R. (Case 1), (1941), *Ibid.*, **46**, 835.
31. DUTAILLIS, P. (1949), *Rev. Neur.*, **81**, 997.
32. SHAPIRO, R. (Case 1) (1952), *Radiol.*, **58**, 94.
33. LE BEAU, M. J. (1949), *Rev. Neur.*, **81**, 1029.
34. ABBOTT, K., quoted by HAMBY, W. B. (1952), 'Intracranial Aneurysms', Thomas, Springfield, U.S.A.
35. SKILLICORN, S. A., and AIRD, R. B. (1954), *Arch. Neurol. Psych.*, **71**, 367.
36. CHAO, W. H., KWAN, S. T., LYMAN, R. S., and LOUCKS, H. H. (1938), *Arch. Surg.*, **37**, 100.