OPERATIVE METHODS IN THE TREATMENT OF PEPTIC ULCER

LECTURE II

By NORMAN C. TANNER, F.R.C.S.

OF late years there has been a trend towards a more liberal use of surgery, in the treatment of peptic ulcer. There are several reasons for this.

(1) The lowering of surgical mortalities.

(2) Diminished post-operative morbidity, pneumonia, empyema, peritonitis, duodenal fistula, etc.

(3) Diminished late ill effects of surgery, *e.g.* persistent ulcer or occurrence of anastomotic ulcer.

(4) Increasing knowledge by the laity of the achievements of surgery.

(5) Failure of medical treatment to control the relapse rate.

We have to be very careful in our judgment of cures for ulcer, whether medical or surgical. Any treatment you may care to make up out of your head, so long as it is reasonably harmless and which entails a fortnight or more of bed rest, and particularly if followed by a convalescent period, will give about a 70 per cent. relief of symptoms for about half to one year. An appendicectomy, with the strong psychological suggestion of an eliminative operation, usually gives a year or more of freedom, but I believe a salpingectomy would give as much relief.

Many different operations have been used, and some measure of success has followed nearly all the operations which have from time to time been popular.

Some operations have perforce been abandoned because they have proved to be inadequate, or hardly worth the risk of laparotomy, *e.g.* local or wedge excision, some because improved procedures have been introduced. Some operations have fallen out of favour because the disease itself has changed somehow in character. In my first lecture I mentioned as an illustration of this change in the nature of the disease, we have the evidence that gastric ulcer used to be common in young women and duodenal ulcer infrequent, now gastric ulcer is rare and duodenal ulcer on the increase. In the same way the duodenal ulcer rate has greatly increased in young men. Now twenty to thirty years ago it used to be said that the incidence of anastomotic ulcer after gastro-jejunostomy for duodenal ulcer was low, perhaps 1 to 2 per cent. Nowadays the figure of 20 to 30 per cent. is often quoted, and that is nearer to my own recurrence rate after gastro-jejunostomy. Knowing the integrity and powers of observation of many of those clinicians who used to quote the lower figure I cannot believe that

they were so overcome by the successes that they did not notice the failures and I believe their figures were accurate. I believe, rather, that coincident with the increase in the incidence of duodenal ulcer, there has been a similar increase in the incidence of anastomotic ulceration in the gastro-enterostomised population—or if you like, there is now an increased risk of developing an anastomotic ulcer after gastro-enterostomy than there was twenty years ago. For all we know this tide may change, perhaps in another twenty years young women will be getting gastric ulcers again, and duodenal ulcer become rare in young men, and perhaps we shall then be able to do gastro-enterostomies with a recurrence rate of 2 to 3 per cent. only. Why these changes should occur one does not know, but they illustrate the fact that the problem of what is the correct operation for peptic ulcer, like the problem of when to operate, does not remain static. There are also geographical variations in the character of ulcer, though the incidence and character tend to become similar in peoples of similar habits and social standards. Perhaps a gastro-jejunostomy in S. India in the hands of Somerville may possibly be done with a lower anastomotic ulcer incidence than the same operation done by me in London.

I think therefore that except for occasional unusual circumstances such operations as pyloroplasty, local excision of ulcer, wedge and sleeve resection are not to be recommended because of the high recurrence rate of ulceration in the scar. Gastro-jejunostomy is an excellent cure for duodenal ulcer. I have never seen an active duodenal ulcer in the presence of a good gastro-jejunostomy. But we cannot use it as a routine because of the high stomal ulcer rate. Though it is occasionally advisable in the treatment of pyloric stenosis, even when done for advanced degrees of stenosis, severe anastomotic ulceration may follow.

Therefore I feel that our elective choice in the surgical treatment of gastric and duodenal ulcer lies between gastrectomy and vagus nerve section.

I would like first of all to consider operation for gastric ulcer. Now one so often sees gastric ulcer in a subacid medium, and indeed the acidity of the average gastric ulcer is subnormal, that I cannot believe that vagotomy will have any great influence on it, particularly as the vagotomy adds stasis and inflammation, both usually inimical to ulcer healing. On the other hand, gastrectomy approaches the ideal as an operation for gastric ulcer, for as I mentioned in my first lecture, after it recurrence is a rarity, and gastric ulcer may recur in the scar of a wedge or sleeve resection even when combined with gastro-jejunostomy.

Why does a gastrectomy succeed in preventing ulcer ? It is usually said that the ulcer bearing area and the scar is removed, the gastric acidity is reduced by the reduction in the area of acid-secreting cells and by the neutralisation of acid gastric juice by the alkaline duodenal juices, and the hormone stimulus of the pyloric antral mucosa—the

gastrin of Edkin, which normally stimulates the fundus to secrete acid is also removed. But gastrectomy leaves a longer scar than was there before. The whole ulcer-bearing area is not removed, for several centimetres of the lesser curve are often left, and even if an ulcer is left and a gastrectomy done below it the ulcer tends to heal. Furthermore gastric ulcer may occur in subacid or even an achlorhydric medium and so reduction of the acidity is unlikely to be the most important factor. It is an interesting problem and is bound up with many unknown factors concerning the causation of peptic ulcer.

To consider this problem it is well to return to first principles. What is the cause of ulcer and of ulcer pain ? I personally believe that the evidence is all in favour of the theory that ulcer and ulcer pain

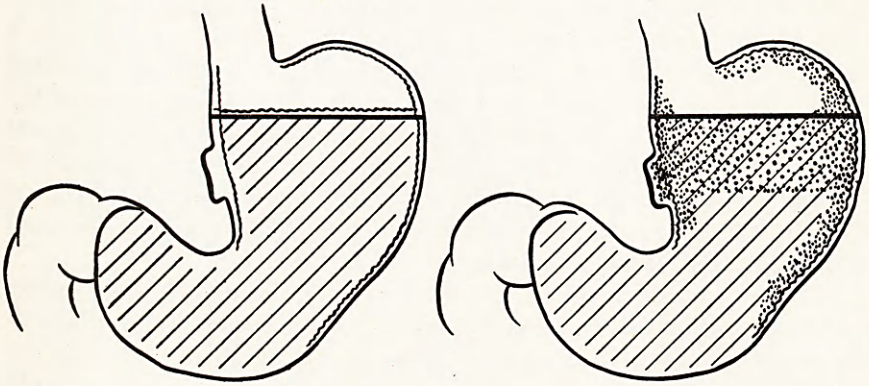

FIG. 12.—A diagram suggesting the possible distribution of the greatest concentration of gastric juice shortly after a meal.

are due to the stimulation of the stomach and ulcer crater by gastric, particularly acid gastric juice. In even a so-called anacid mucosa, acid is secreted from the remaining peptic cells but is small in quantity and immediately buffered by mucus and gastric content.

Now my suggestion is that the time of ulcer pain is related to the time when chyme of the maximal acid concentration is in contact with the crater. Normally when food is taken, there is not at first a complete admixture, but the food lies in layers, that is to say, pudding above meat. The peptic juice pouring from the stomach wall forms at first a greater concentration between food and mucosa where is is quickly buffered by combining with food protein and the juice also pours down from the fundus to mix with the upper food layers, where it forms its greatest concentration.

If one passes a Ryle's tube into the stomach immediately after a meal, one can take specimens from different parts of the stomach more or less simultaneously. Here is such a test meal result (Fig. 8).

Unfortunately we have no method of judging accurately the variation in acidity within the stomach at any particular phase of digestion, but this picture shows a probable state of affairs (Fig. 12).

Now my theory is that the pain-food relationship depends on the time after a meal when the greatest acid concentration comes into

contact with the ulcer crater. We know that there is a rough tendency for pain to come later after food the farther down the stomach the ulcer lies. If one takes X-ray pictures of a patient with a lesser curve ulcer, it can be seen that at the time of pain onset this broad upper acid level is roughly opposite the crater.

Eventually the lower layers of food will have almost left the stomach, but acid juice is still being secreted so that the final parts of the meal are food plus much acid gastric juice. This is the time, about two hours after a meal, when the duodenum comes into contact with the most acid part of the gastric chyme, and this is the time when duodenal ulcer pain usually commences.

Now why has *bed rest* sometimes such an immediate effect and such a healing effect on ulcer ? Well the relation of the upper acid level of food to the crater becomes changed. Lying on the back or side a lesser curve ulcer may suddenly be covered in food rather than bathed in acid juice pouring down from the fundus, and different parts of the stomach, perhaps the greater curve will then have to take the brunt of the upper acid level. Furthermore in recumbency the upper level is widened in extent, thinned and gastric peristalsis will make a completer food mixture, so that there is less tendency for a concentrated upper acid level of juice to appear.

Experimental evidence is fairly strong that constant gastric hypersecretion stimulated perhaps by injections of histamine in beeswax may lead to peptic ulceration, without any other factor. It may be that in some individuals prolonged stimulation from the more acid level of the gastric chyme may lead to ulcer in the corresponding level of gastric mucosa.

Now if there is anything at all in this I think we can see better why a stomach after partial gastrectomy may not re-ulcerate. First of all the constant entrance to the stomach of duodenal juices propelled by jejunal peristalsis will tend to increase the mixing effect, and neutralise the acid level.

Secondly the relation of the upper food level to the lesser curve is changed, the gastric remnant being more full at first and then the upper food level descends rapidly owing to the faster note of emptying, so that no one part of the lesser curve suffers a constant stimulus of being opposite the high acid concentrate level.

However, I must not spend too long building an intricate superstructure on my as yet flimsy theory. Let me say, however, that there is a rich field for research in this subject.

As I have mentioned very high lesser-curve gastric ulcer sometimes presents a difficult technical problem. Most ulcers which are almost as high as the cardia may be included in the resection by removing a tongue-shaped piece of the lesser curve, the so-called Pauchet modification (Fig. 13). It is neither necessary, nor I think desirable to resect nearly all of the stomach in these cases.

In the case of ulcer encroaching on the cardia then I think gastrectomy below the ulcer as recommended by Madelener and

FIG. 13.—Posterior view of stomach with a high lesser curve innocent gastric ulcer, removed by the Pauchet method.

Florcken is sufficiently well established to justify it, provided one has œsophagoscopic biopsy or absolute evidence of innocence.

For the average case of ulcer involving the body of the stomach, it is unnecessary to do an excessively high resection, transection at a convenient level above the ulcer is safe in view of the rarity of stomal ulceration.

There has been a revival of interest in the Billroth I form of gastrectomy in the treatment of gastric ulcer, because of the impression that unpleasant post-gastrectomy or dumping symptoms are less after gastro-duodenal than after gastro-jejunal anastomosis. My impression of the Billroth I operation has been favourable on the whole, and though post-gastrectomy symptoms may follow it they are usually relatively mild and transient. This form of gastrectomy has the additional advantage of having no jejunal loops to get into trouble and when performed for gastric ulcer it is a smaller operation. I have also noticed an incidence, though very small, of pancreatitis as an early and even more rarely a late complication of partial gastrectomy completed by gastro-jejunal (Polya) anastomosis. This may be due to the partial defunctioning of the duodenum which results from the operation. It has also been suggested, with some slight evidence, that biliary disease is commoner than normal after Polya gastrectomy. It is possible that the passage of food through the duodenum is physiologically advantageous to the biliary and pancreatic systems, perhaps by aiding the expulsion of their juices. If this be so, then the Billroth I operation would be physiologically more sound. Unfortunately it may be a hazardous procedure in duodenal ulcer cases and I do not use it except for gastric ulceration.

Now as to duodenal ulcer. Here the problem used to be that of gastro-jejunostomy versus gastrectomy. With the rising incidence of gastro-jejunal ulcer this problem has become simplified. Gastro-jejunostomy is the finest of all cures for a duodenal ulcer, but unfortunately a rising percentage of cases get anastomotic ulceration. We therefore now keep gastro-jejunostomy for the very occasional poor risk severely obstructed cases, in which the undoubted risk of developing anastomotic ulceration is overclouded by the immediate needs of the case. For most cases gastrectomy has fairly satisfactorily filled the bill hitherto. Time has shown it to have certain disadvantages. The first is obviously the primary mortality. This has diminished from something near 6 to 10 per cent. to the region of 1 to 2 per cent. or less during the last fifteen years, as a result of improvement in the treatment of pulmonary and peritoneal infection, of acute bronchial obstruction with lung collapse, improved anæsthesia, etc. Technical faults causing death should be an extreme rarity and ideally should be eliminated. One still sees too many reports of duodenal stump leakage, particularly after gastrectomy for duodenal ulcer. The two commonest causes of leakage are inadequate closure of the stump and back pressure on the duodenum leading to duodenal distension and rupture. I believe that scrupulous care in closure of the duodenal

stump can always give an adequate closure. The closure should never be hurried. In support of this I can claim well over a thousand personal gastrectomies for simple ulcer, without a single disruption of the duodenal stump. In the rare case of ulcer of the second part

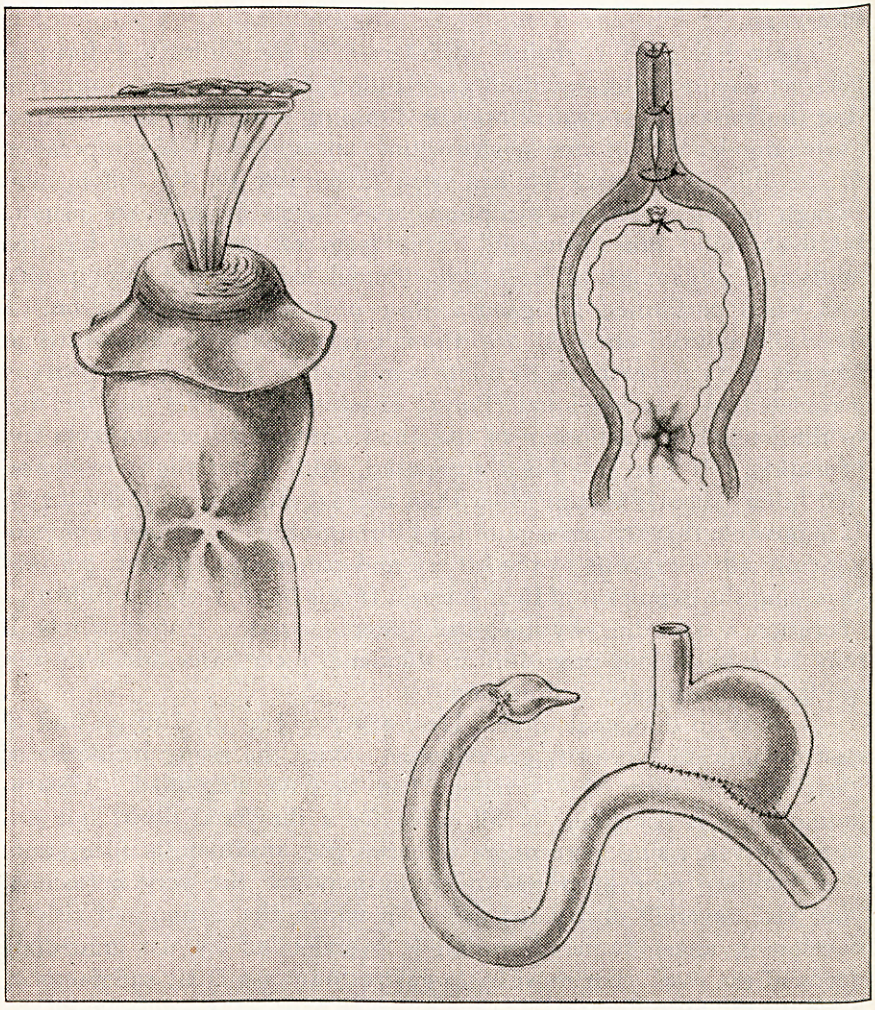

FIG. 14.—The " Bancroft-Plenk " method of exenteration of the pyloric mucosa.

of the duodenum in which the first part is reasonably healthy, I think closure of the first part, leaving the ulcer *in situ*, will give a perfect result. In difficult ulcers of the first part it is sometimes advisable that the pyloric antral sero-muscular coat be left and closed, after the pyloric antral mucosa has been removed. This is an adequate operation, though somewhat vascular and unæsthetic. It is important when doing the operation to leave an adequate blood supply to the antral muscularis, or it may slough. A modification of this method which I have tried recently is to make a transection of the antral seromuscular coat only 2 cm. from the pylorus, the Bancroft-Plenk operation. The

mucosa is dissected away until the circular pyloric sphincter is clearly seen. The mucosal cuff is now ligatured and divided and a very sound closure may be made by stitching over it the thick pyloric muscle (Fig. 14). An alternative that I would recommend to the tyro in duodenal surgery is to transect the antrum, close it and then proceed to do a high gastrectomy. Shortly afterwards it is important to re-operate and remove the antrum, not later than eight weeks afterwards. By this time the antrum will be found to be contracted, the ulcer healed and the adhesions much looser and more easy to deal with. It is most important to remove the antrum for if it be left with its mucosa there is a high risk of anastomotic ulceration. In 1936 I did three gastrectomies conserving the antrum—one developed an anastomotic ulcer within six weeks, the second within a few months, and I lost sight of the third. Ogilvie and many others reported similar results. It is important not to leave them to see how they get on. Most of them will get a stomal ulcer, and once the new stoma has become scarred by ulceration it will be much more prone to recurrent ulceration even after antral resection.

I will not say much more about the technique of gastrectomy for duodenal ulcer. I prefer a gastro-jejunal anastomosis with a valve—retrocolic if it is technically suitable. I would make one warning about the use of silk. It has always been my habit to use catgut for mucosal suturing, but toward the latter part of the war when catgut supplies were difficult, I changed to the use of fine silk for closing half the diameter of the stomach. The result of this has been that this continuous silk suture becomes extruded into the stomach (Fig. 15). This means that there is a small stitch abscess and on four occasions it has led to quite a brisk hæmatemesis. Needless to say I have now reverted to catgut for all " all coats " sutures near the anastomosis.

A much-discussed cause of incomplete success with gastrectomy are the so-called " post-cibal " or " dumping " symptoms. These may appear while the patient is in or shortly after leaving hospital, but usually diminish within a few months so that by the end of a year only about 7 per cent. of cases are incommoded by them. The commonest form in my experience is that shortly after eating there is a feeling of fullness followed by heat, sweating and palpitations, and then fatigue. Now the first thing to realise I think is that these are the exaggeration of a normal reaction to overeating. Those of us who at times have overeaten will remember the feeling of fullness and fatigue, and the stouter trenchermen among us may recollect the feeling of heat, palpitations and mild sweating after our heavier repasts. More than that it must be said that after a period of small feeding, as after a gastric operation, or even after a period of semi-starvation without any operation, a normal meal may produce similar symptoms in a normal person. How much more likely then is the gastrectomy patient with his quarter-stomach likely to suffer. These symptoms are not restricted to gastrectomy patients. Several times in the dyspepsia clinic a patient has been introduced who complains that shortly after

eating he gets fullness, heart palpitation, sweating and faintness. I must confess I usually say " When was the operation ? "—but they have had no operation. In such a case no gross gastro-duodenal abnormality is found, not even excessively rapid discharge of chyme into the jejunum, and they are diagnosed as functional dyspepsias—meaning in this case inexplicable dyspspeia. I think they are usually small eaters who are introspective and exaggerate the normal symptoms they get after taking a big meal.

What is the cause of this form of post-cibal symptoms ? Numerous causes have been postulated. Irritation of the cut end of the vagus has

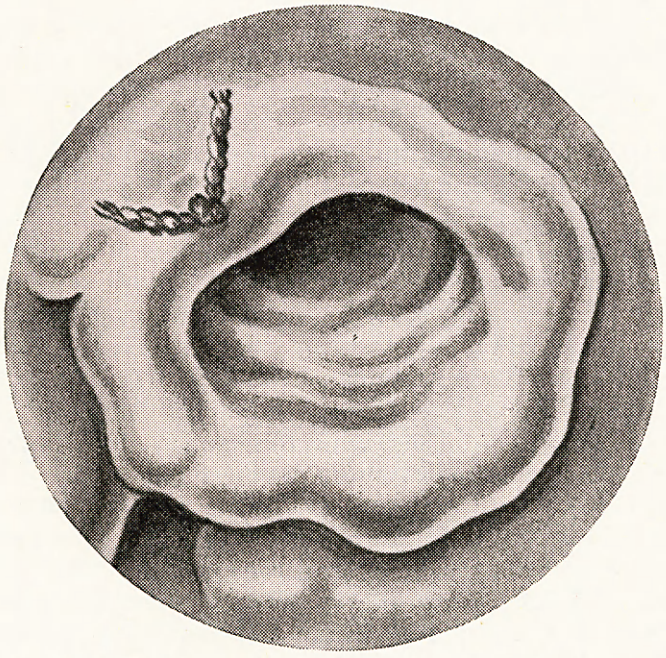

FIG. 15.—Continuous silk suture partially extruded into the stomach a year after gastrectomy. Gastroscopic view.

been blamed—vagotomy does not cure it. Blood sugar fluctuations, or hyper or hypoglycæmia. The symptoms appear too soon, sometimes before the end of the meal, and blood sugar estimations usually show normal blood sugar levels. In earlier days these symptoms were believed to be due to rapid passage of food from stomach to the jejunum and this was described as dumping stoma by the radiologists. A more modern suggestion is that taking food of high osmotic pressure attracts fluid into the bowel and so distends it. One can only say that many of the most comfortable patients are found on barium meal examination to pass the gastric content almost directly into the jejunum and do not suffer from it. We cannot diagnose the " dumping " syndrome radiologically, and as Dr Cochrane Shanks said in this year's Lettsomian Lectures, the term " dumping stoma " should be dumped.

Traction on the œsophagus or on the vagus ? It is suggested that

cutting the left gastric artery removes a support and thereafter the loaded stomach pulls on the œsophagus or vagus nerve and produces these symptoms. Division of the vagus does not relieve it, and post-cibal symptoms may occur in patients whose gastric remnant is firmly tethered to the diaphragm by adhesions.

Gastric distension? This is not confirmed by X-ray. It seems to me that post-cibal symptoms are more marked in patients whose stoma is equal to the whole width of the stomach. It may be that in such cases there is distension of the afferent jejunal loop due to the addition to the bile and pancreatic juices of the bulk of gastric content which has entered the wrong loop. The smaller stoma such as I use and the very small one recommended by some Edinburgh surgeons may not give its better results so much by slowing the rate of emptying of the stomach as by diminishing the amount of food and gastric juices which enter the afferent loop.

The entry of bile into the stomach, such as normally occurs after gastrectomy, appears to disturb some patients and they regurgitate some of it.

How shall we treat these patients with the dumping syndrome? I have no cure to offer you. I have found no great effect from ephedrine to raise the blood sugar. Fatty and protein meals have been recommended as likely to produce a more stable blood sugar curve.

The most important factor in prevention is to pick cases for surgery who are really anxious to get well. A patient who hopes for an excuse to stop his work can build up a severe psychosis on mild post-cibal symptoms. A patient who has suffered agony from his ulcer for years will minimise even severe post-cibal symptoms.

The patient should be told before operation that he may get fullness and sweating but that it will disappear in a few months, and in the majority of cases that will occur. The patient who has not been warned thinks that the operation has failed, and pays undue attention to the symptoms. The patient who has been warned will not be worried by the onset of the symptoms. When I am confronted with a physiological response which I do not understand, I place my faith in nature's wonderful compensatory mechanism. If it is due to blood sugar alteration—the body mechanism tends to adjust itself, if it is due to distension of the viscus—then distend the viscus adequately and persistently and after a time distension will cease to provoke any reaction. Therefore as soon as the patient is recovered from the operation I advise him to take bulky meals of fatty and protein food, and to eat until he feels bloated, three times a day. By this means many of them increase their capacity, even though for a few weeks they are uncomfortable after meals, and soon a normal meal fails to produce symptoms, though an exceptionally large one may. In addition this helps to prevent too much weight loss, which further distresses the patient. Taking small meals it seems to me prolongs the disability.

Biliary regurgitation, which is described as a burning fluid, troubles some. Some are more worried than incommoded by it, and go to

great efforts to try to vomit it. Such patients may be made well by simply explaining that it is a normal result of turning bile into the stomach and the fluid is not an abnormal fluid, nor need the patient try to bring it up. Sometimes, however, it is difficult to deal with. One of the worst cases I had was in a total gastrectomy, end-in-side, done in 1941. Now since that date all my total gastrectomies have been on the Roux principle and I have noticed that they do not get

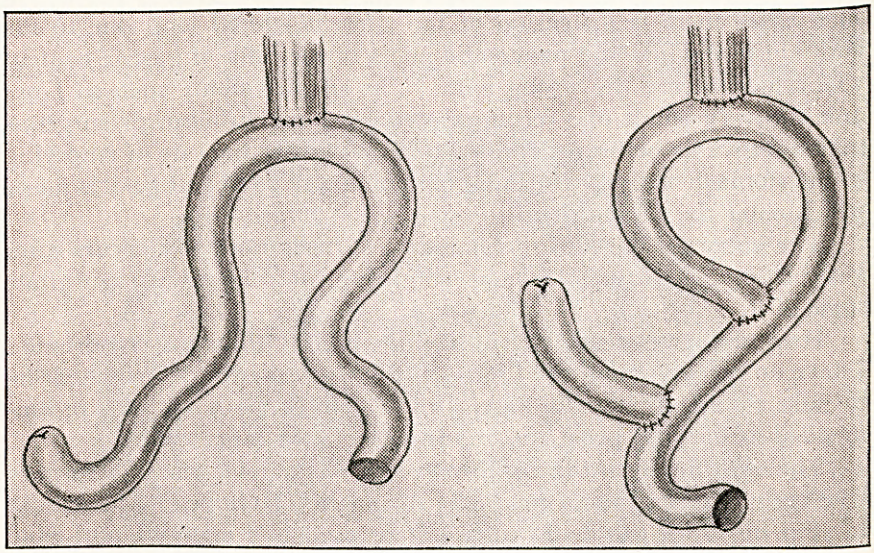

FIG. 16.—Simple method of conversion of an end-in-side total gastrectomy to a modified Roux operation, with great relief of symptoms of regurgitation.

post-cibal symptoms. Therefore one year ago I operated on the lady and converted her anastomosis into a Roux type (Fig. 16). From that moment she has lost all symptoms, she has gained weight, and is remarkably better. Now can we do the same with a partial gastrectomy? I would be afraid to do so for fear of recurrent ulceration and the most I have dared to do in three cases is a vagotomy combined with short circuiting of the loops, and in one a Roux anastomosis with indefinite results (all four cases being somewhat mixed types).

It is said that post-cibal or dumping symptoms are less noticeable after the Billroth I operation, and less after retrocolic short loop operations than after antecolic long loop operations. There is some evidence in favour of this statement though there is no doubt that post-cibal or so-called dumping symptoms may follow the Billroth I operation. However, in certain cases, where regurgitation symptoms warrant it, and where the symptoms have been severe for more than a year, it may be worth while to convert a Polya into a Billroth I operation. This is not so easy, nor a small operation, and should not be undertaken without much thought. In three cases where it was done, one patient was enabled to return to his work and was definitely improved. The other two were not really better.

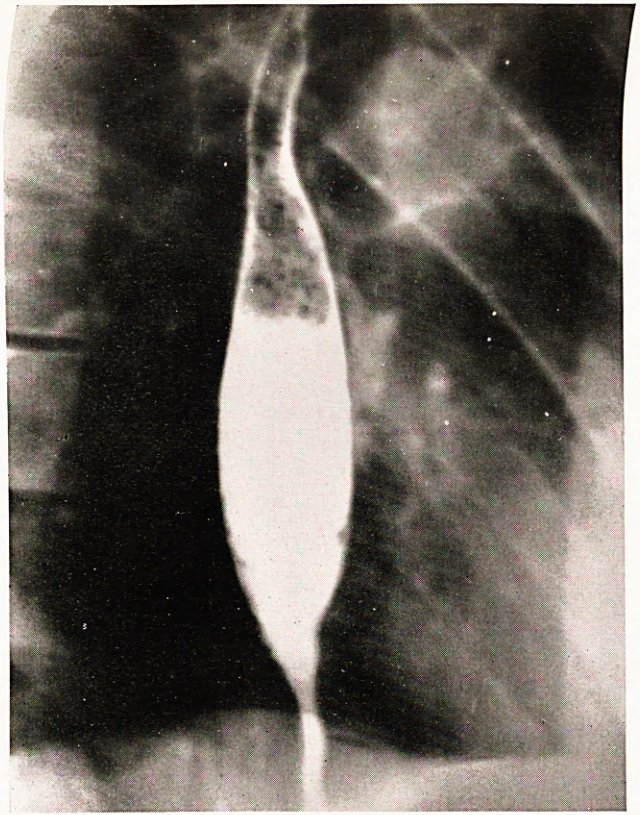

FIG. 17.—X-ray of barium swallow three weeks after vagotomy, in a patient complaining of mild dysphagia.

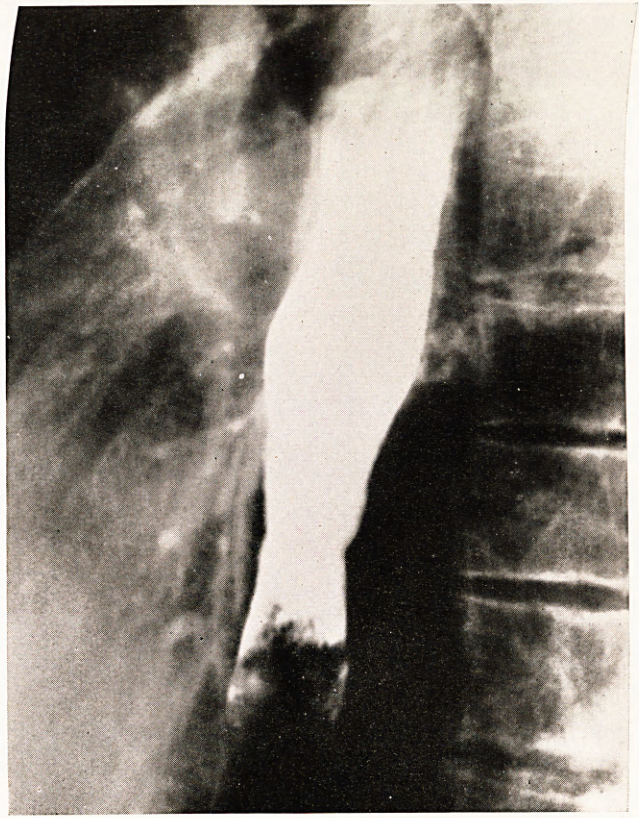

FIG. 18.—Filling defect due to food debris in lower œsophagus. Associated with mild neuromuscular dysfunction.

Results of Gastrectomy

There is no doubt that on the whole partial gastrectomy is a very satisfactory operation for duodenal ulcer, provided that the cases are wisely selected. A five year follow-up of ninety-one cases in 1947 showed these results.

Follow-up of Gastrectomy for Duodenal Ulcer only
1941-43
(Examined September 1947)

Operation: Rather long loop antecolic Hofmeister-Finsterer type

Cases traced, 91	Cases
Completely symptomless, normal occupation	57
Complete absence of ulcer symptoms, occasional sweating or flushing, or nausea, admitted on close questioning	19
Complete absence of ulcer symptoms. (Sweating, faintness or nausea, insufficient to prevent work, but worry patient at times or frequently)	7
	83
Recurrent ulceration	2
Poor result. (No ulcer symptoms but no better than before owing to: (1) cyclothymia; (2) functional vomiting; (3) post-prandial fullness; (4) depression and post-cibal fullness)	4
Since died of: (1) bronchial neoplasm; (2) asthma and bronchitis	2

You will notice that two cases developed stomal ulceration. It has been suggested that an extremely high gastrectomy—leaving only three or four inches of greater curve will eliminate stomal ulceration. I am much against such drastic resections for simple lesions. We are operating on people who may normally hope for another twenty to fifty years of life. We know that a moderate two-third or three-quarter gastrectomy may confidently anticipate no shortening of this period, but the very late effects of a quasi total gastrectomy are unknown. I would prefer therefore to perform a reasonably high gastrectomy—dividing only the lower two vasa brevia, and be prepared to perform vagotomy on two out of every ninety who relapse.

The gastrectomy mortality is also to be considered, and so I append my mortality since the air raids of 1941.

Mortality of Gastrectomy for Benign Ulcer (excluding Operations done Solely to arrest Massive Hæmorrhage and Gastro-colic Fistula)
June 1941 *to December* 1950

Ulcer	Cases	Death	Mortality
Duodenal	338	4	1·2 per cent.
Gastric	502	9	1·9 ,,
Anastomotic	64	1	1·5 ,,

Now what of the alternative operation, vagotomy? When this operation became popular I felt it would be wise to perform the vagus resection pure and simple rather than combine it with gastro-jejunostomy, because gastro-jejunostomy cures duodenal ulcer and so

it would confuse the results. However, some patients were found to have such a degree of duodenal narrowing that I felt that further narrowing due to ulcer healing would lead to pyloric stenosis, and so in these narrowed cases I decided to do a simple Heineke-Mikulicz pyloroplasty. I do not think that pyloroplasty can be claimed as a very successful cure for duodenal ulcer, and so any beneficial results from this combination could be properly attributable to the vagotomy. In a few cases, however, there was so much duodenal shortening and deformity that one was compelled to perform a gastro-jejunostomy.

The result of this was that all the cases which had the combination of vagotomy with pyloroplasty did very well and had practically no retention symptoms. Of the pure vagotomies, however, one-third developed persistent retention symptoms, with foul belching, cramps, fullness after meals, etc., and radiological evidence of six to twenty-four hours gastric retention. It is difficult to say why these patients had such a foul smell to their eructation. I have pointed out that the belch of pure pyloric stenosis due to duodenal ulcer may be inflammable, but it is not really malodorous as a rule. The belch in these cases resembles more the gas expelled by the patient with a carcinoma of the œsophagus or stomach causing obstruction. I suspect that abnormal sulphurous products are produced as a result of the combination of stasis plus hypo- or anacidity, and to avoid the smell we must eliminate either the stasis or the hypoacidity. Now we do not want to correct the trouble by restoring the acidity, for that may reactivate the ulcer, and the obvious course is to try and obviate the stasis. Therefore I re-operated on these cases with stasis, usually about a year after vagotomy. I expected to find that their ulcers would have healed leading to further narrowing of the duodenum, but in fact found that the duodenum was healed and quite wide. In view of the good results in the pyloroplasty cases I used a simple pyloroplasty on a few of them. Somewhat to my surprise this worked excellently, although some of them took up to three months to get a normal gastric emptying time.

Some have suggested that vagotomy be combined with either pylorectomy—thus removing the pyloric antrum with its hormonal phase of gastric secretion. This introduces the possibility of post gastrectomy as well as post vagotomy symptoms and will give a mortality slightly higher than that of partial gastrectomy. Others have suggested a high partial gastrectomy plus vagotomy for all cases of duodenal ulcer. This again introduces the post-operative syndromes and the operative morbidity and mortality of both operations. Furthermore, as the recurrent ulcer rate after high gastrectomy is only 3 per cent., in 97 per cent. of cases the vagotomy will have been unnecessary. It is much better to do the one operation and reserve the second for the small minority who really require it.

What of gastro-jejunostomy as an additional procedure to vagotomy? An obvious disadvantage is that we do not yet know if vagotomy is adequate protection against jejunal ulceration. Certainly stomal ulcers appear in some cases. My chief criticism of this combination is that

gastro-jejunostomy increases the chance of severe post-operative small intestinal ileus. Perhaps swallowed air passes through the stoma and into the paretic bowel, faster than it can be aspirated from the stomach through the indwelling gastric tube. This condition may be extremely serious and is a cause of occasional post-operative mortality.

Vagotomy has other effects apart from gastric distension. As a result of previous animal experiment and occasional operations on man, many of the physiological changes which would follow vagotomy were well known before the operation became relatively common. Furthermore, we can now prognosticate with reasonable accuracy changes which may occur at a later stage; for example, if acid levels in the stomach remain depressed in some of the vagotomised cases, we can anticipate that some of them will develop iron deficiency anæmias in the years to come. Let us hope that even worse surprises are not in store for us.

In 1906 Cannon showed that vagotomy produced paralysis of the unstriated muscle of the œsophagus, with cardiospasm, or at any rate a failure of relaxation of the muscle at the cardia. These effects were usually transitory, there being a certain return of activity within a few days. Now it is a common clinical observation that dysphagia with radiological evidence of cardiospasm may occur in some patients (3+per cent.) shortly after vagotomy (Fig. 17). This usually subsides with simple measures within a few days. The question arises as to whether late effects will occur in any of these cases. I know of no case of persisting cardiospasm after vagotomy but one case gave me some basis for fear.

A man aged 64 had an abdominal vagotomy and gastro-enterostomy in February 1947, having suffered two perforations and subsequent stenosis of a duodenal ulcer. Following this he was extremely well until two years and seven months later, when he was admitted with a five days' history of being unable to swallow fluids or solids. A barium swallow showed an irregular filling defect in the lower œsophagus (Fig. 18). I œsophagoscoped him and removed some inspissated meat, not a very great quantity, from his lowest œsophagus. There was no tumour or stenosis and so I presumed that the impaction of such a small amount of soft meat must have been basically due to a neuromuscular defect. He has since been perfectly well and shows no evidence of cardiospasm. We must keep an open mind as to the possibility that there may be permanent neuromuscular effects on the gullet.

Before finishing I would like to mention one other effect I have noticed when operating on cases of vagotomised patients who have suffered from prolonged obstruction. That is, that there is a great development of gastric adhesion, the anterior wall of the stomach being firmly bound to the liver, anterior parietes and the diaphragm.

In my opinion three factors are responsible for this.

(1) Peritoneal trauma of the operation. This is an essential ingredient because adhesions do not form on the posterior wall of the stomach and they did not occur in one transthoracic vagotomy we re-explored.

(2) Atony with immobility of the stomach obviously favours adhesion formation.

(3) Gastritis due to retention producing congestion of the gastric wall may produce an adhesive quality in the peritoneum, and combined with the other factors produce adhesion.

Now I have only re-operated on cases which had had pure vagotomy and one vagotomy plus gastro-jejunostomy. It is possible that a coincident pyloroplasty may diminish these troublesome adhesions, but I can say nothing on this point for I have not had to re-operate on any cases of coincident vagotomy and pyloroplasty. It is a serious drawback to the operation.

My best way of ending may be to give some results of vagotomy. These are cases at St James' Hospital and have just been worked out by a careful follow-up by the Dan Mason Research Unit.

Vagotomy

180 *Cases*

166 (91 per cent followed up)

D.U.	155
Pyloric	2
G.J.U. post gastrectomy	9

Pre-operative Complications

Hæmorrhage	54
Perforation	36
Stenosis	15

Ages

15-20	3
21-40	80
41-60	60
61-	14

Length of Follow-up

Under 1 year	54
1-2 years	63
2-3½ years	49

? Recurrence of Ulceration

1. Recurrent pain	22
2. X-ray evidence	3
3. Operative evidence	2
4. Bleeding	3
5. Perforation	1

Vagotomy Results

Vagotomy alone

56 cases.

28 per cent. underwent further operation for retention symptoms.

Vagotomy + Pyloroplasty or Vagotomy + Gastro-jejunostomy

116 cases.

102 (88 per cent.) satisfactory.

All Operations

166 cases.

6 (3·6 per cent.) proved recurrence.