Adverse Effects of Gastric Surgery for Peptic Ulceration

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Surgery has played an important role in the management of peptic ulceration for many years. Its popularity stems partly from dissatisfaction with current medical therapy and partly from the excellent clinical results that may be obtained. However, destruction of the normal physiological mechanisms that is inherent in all the surgical procedures used results in adverse effects in a substantial number of patients.

Because of these problems, surgeons have continually sought the ideal operation, one that cures the disease without causing serious adverse effects. Over the years a number of different operations have been described and each has been hailed as the answer to these problems. Unfortunately, subsequent studies, including a few control trials, have later shown that each operation has its own attendant adverse effects, leaving little to choose between them.

In Newcastle we have a long-term interest in this topic, arising from our studies of the long-term metabolic sequelae of vagotomy and gastroenterostomy which demonstrated a significant proportion of patients with serious problems[1]. Since 1968 we have followed up all patients who have undergone gastric surgery on the Professorial Surgical Unit. Follow-up has been documented in a standardised manner, both on entry and at intervals of one, two, five, eight and ten years after operation. The records obtained have been computerised for subsequent analysis.

Adverse Effects After Gastric Surgery

Adverse effects can be divided most conveniently into early, that is less than three months from operation; medium-term, that is less than five years; and long-term, over five years from operation.

Early Adverse Effects

Most of these are related to the surgical procedure and usually occur within the time of the hospital admission. They include wound and intra-abdominal sepsis, anastomotic and wound dehiscence, deep vein thrombosis and pulmonary emboli. There are, however, two adverse effects that are not uncommonly seen after discharge from hospital.

Dysphagia. This tends to occur six to eight weeks after discharge from hospital. It usually becomes apparent when the patient returns to normal eating habits. In our experience, dysphagia is usually self-limiting in nature and resolves spontaneously in the majority of patients. Only in an occasional patient is one or more dilatations needed to relieve symptoms. It is thought that this dysphagia results from oedema or haematoma formation around the lower oesophagus following surgery and it certainly occurs most commonly after proximal gastric vagotomy (HSV), which involves extensive dissection in this area.

Diarrhoea. A short period of diarrhoea is not uncommon after most types of gastric surgery. This generally resolves spontaneously within a few weeks of discharge from hospital and only occasionally is symptomatic treatment required. The diarrhoea is thought to be the result of a change in the small bowel bacterial flora following loss of the normal 'sterilising effects' of gastric acid secretion.

Medium-term Adverse Effects

These include most of the problems commonly associated with gastric surgery and they usually start shortly after the operation.

Recurrent Ulceration. This can occur at any time after gastric surgery but is most common within the first few years. Diagnosis of the condition has been made much easier by the advent of fibre-optic endoscopy, as radiological assessment is extremely difficult and inaccurate because of distortion of the stomach produced by the operation. In our opinion, endoscopy is one of the

primary investigations for anyone who presents with unexplained persistent dyspepsia, anaemia or gastrointestinal haemorrhage following gastric surgery. If an ulcer is found, it is essential that every attempt be made to ascertain why it has recurred, so that effective management can be planned.,

In our own practice, we always obtain a sample of blood for fasting serum gastrin and generally perform a combined pentagastrin-insulin secretion test[2] to assess the relative roles of vagal function and secretory capacity as the cause of the recurrent ulceration. If the serum gastrin is greater than 100 pg/ml a secretin stimulation test (2 units/kg GIH Secretin) is advised to exclude a Zollinger-Ellison syndrome.

The incidence of recurrent ulceration depends on the type of operation performed, being lowest where vagotomy has been combined with an antrectomy, and probably highest after proximal gastric vagotomy, particularly if the latter operation is performed by those inexperienced in its technique.

Abdominal Pain. While recurrent ulceration is relatively uncommon after gastric surgery (in our series, 5 per cent at eight years after vagotomy and pyloroplasty), abdominal pain of unexplained origin is quite common. Two years after vagotomy and pyloroplasty, 22.5 per cent of our patients gave a history of abdominal pain for which, in most cases, no cause was firmly established. What dismays us is a tendency for these symptoms to increase in severity with time, the incidence of severe abdominal pain rising from about 2 per cent at two years to 7.5 per cent at eight years after operation. We had hoped that proximal gastric vagotomy would be better than vagotomy and pyloroplasty in this context. However, on our current experience, some 19.5 per cent of patients still have unexplained abdominal pain following proximal gastric vagotomy, though none so far have severe pain.

We have found that 13 per cent of our patients have persistent heartburn after vagotomy and pyloroplasty and this changes little with time. At present, our incidence of this problem after proximal gastric vagotomy is 10 per cent, which is similar to that reported by other workers.

Eating Habits. We compared the eating habits of those who, two years previously, had vagotomy and pyloroplasty or proximal gastric vagotomy. Fewer patients admitted to a decreased appetite following proximal gastric vagotomy (10 per cent) than vagotomy and pyloroplasty (32 per cent), but there seemed to be a greater tendency to a reduction in the size of meal eaten (54 per cent) after proximal gastric vagotomy than after vagotomy and pyloroplasty (34 per cent). This latter difference may simply reflect the smaller numbers of patients in the former group, but it certainly gives rise to some concern, as we had previously found that reduced food intake, due to this complaint, is associated with long-term weight loss after vagotomy and gastroenterostomy[1].

At present we can see no evidence of any major advantage for the operation of proximal gastric vagotomy as far as post-prandial fullness is concerned. This is supported by the conclusions of Goligher *et al.*[3] on their long-term follow-up, five to eight years after this operation. We have been unable to confirm the improvement in this symptom quoted by Stoddart *et al.*[4] in their control trial comparing proximal gastric vagotomy with vagotomy and pyloroplasty.

Vomiting. While bile vomiting is a recognised complication of gastroenterostomy, whether combined with vagotomy or performed as a result of Polya gastrectomy, it is usually assumed that vagotomy and pyloroplasty is without this problem. Although it is true that bile vomiting is rare after vagotomy and pyloroplasty, in our experience the vomiting of food or gastric fluid is found almost as frequently after vagotomy and pyloroplasty as is bile vomiting after vagotomy and gastroenterostomy. A similar incidence of food vomiting has been found after proximal gastric vagotomy in our series.

Dumping. The incidence of dumping reported by other workers varies considerably. We have therefore been strict in our criteria as to what is called dumping and we have divided this into early and late dumping symptoms.

Early dumping we have defined as the onset of the typical symptoms of faintness, sweating, dizziness or the desire to lie down occurring within a short time of taking a meal. It occurs in some 18 per cent of our patients following vagotomy and pyloroplasty at two years but in only 2 per cent is it severe; at five to eight years, the incidence decreases to 15 per cent but 2.5 per cent of patients have severe symptoms. To our surprise, the incidence of dumping after proximal gastric vagotomy is similar to that found after vagotomy and pyloroplasty (14.5 per cent), although none of our patients complained of severe symptoms. This finding is in sharp contrast to the results of most other workers[3, 4] who have reported that the reduction of early dumping is one of the major advantages of this operation. Unfortunately, if dumping occurs, its management is far from satisfactory and, although many different modifications of diet and medications have been tried, very few patients achieve long-term benefit from these procedures and most have to learn to live with what is a serious disability.

Late dumping we have defined as the presence of symptoms similar to those expected with insulin-induced hypoglycaemia occurring between meals. In fact, this condition has been called 'reactive hypoglycaemia'.

This symptom complex has been found in 11.6 per cent of our patients after vagotomy and pyloroplasty at two years but in only 2 per cent has it been severe. There appears to be little change in the incidence or severity of this symptom with time. So far, very few of our patients have experienced late dumping after proximal gastric vagotomy. There appears to be a marked improvement in this symptom with this latter operation.

Bowel Habits. Diarrhoea is a well-recognised complication of truncal vagotomy but it is often forgotten that gastric operations can affect bowel function in a less dramatic way than by causing diarrhoea. In our study,

both vagotomy and pyloroplasty and proximal gastric vagotomy are followed by an increase in bowel frequency. This occurs in over 30 per cent of patients at two years and in some it may increase to over three times that found before operation. This increased bowel frequency may be accompanied by a small malabsorptive component that, if present for many years, could result in long-term metabolic sequelae.

Most of the diarrhoea seen after vagotomy is of an episodic type, varying in severity from one episode every month to several episodes per week. The results of our studies, confirmed by Goligher *et al.*[3] show that proximal gastric vagotomy has a major advantage over vagotomy and pyloroplasty in reducing the incidence and severity of post-operative diarrhoea.

Type of Operation and Medium-term Adverse Effects. Most of the above information was collected after proximal gastric vagotomy and vagotomy and pyloroplasty. These are the two most popular operations for duodenal ulcer in Great Britain, but a number of alternative operations are available. The majority were included in the original Leeds/York trial published in 1968[5]. We have therefore compared our own data with that published in this trial (Fig. 1). The comparison shows that, while there are some differences between the operations, none stands out as being considerably better than any other.

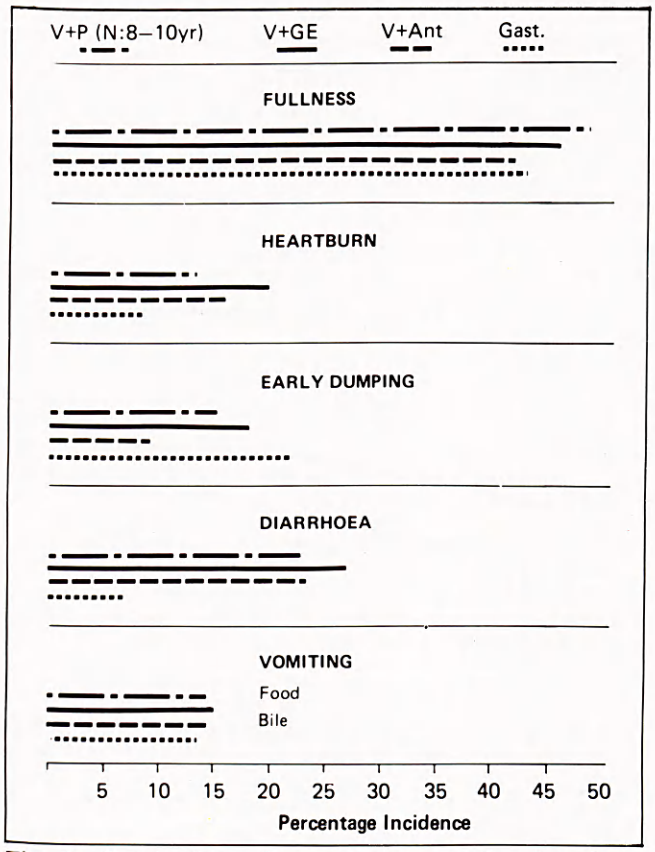

Fig. 1. *Comparison of our vagotomy and pyloroplasty (V + P) series with the results obtained in the Leeds/York trial after vagotomy and gastroenterostomy (V + GE), vagotomy and antrectomy (V + Ant) and Polya gastrectomy (Gast).*

Long-term Effects

In our earlier study, after vagotomy and gastroenterostomy, we found a 7 per cent incidence of tuberculosis which was at least three times the expected incidence for this condition in our area. Following vagotomy and pyloroplasty, we currently have an overall incidence of $1\frac{1}{2}$ per cent of active tuberculosis occurring since the operation.

It has been suggested[6, 7] that gastric surgery is followed by an increased incidence of gastric carcinoma. So far, in our retrospective studies after vagotomy and gastroenterostomy and our prospective studies after vagotomy and pyloroplasty, the overall incidence of gastric carcinoma has been very low and we have no evidence that carcinoma has been increased by either of these operations.

Our major interest has been focused on the metabolic sequelae of gastric operations.

Weight Loss. In our previous study, weight loss following vagotomy and gastroenterostomy appeared to be a significant problem. Our prospective data after vagotomy and pyloroplasty show the same trend (Table 1), although the proportion of patients who are underweight is lower at present. This may well increase in time.

Table 1. Changes in weight (related to standard weights) in patients after vagotomy and gastroenterostomy (V + GE) and vagotomy and pyloroplasty (V + P).

		SW or above	4-6 kg under SW	7-13 kg under SW	>13 kg under SW
V + GE (15-20 yr)	♂ 106	53%	15%	23%	9%
	♀ 16	36%	4%	32%	28%
V + P (8-10 yr)	♂ 161	68%	9%	17%	6%
	♀ 34	47%	18%	18%	18%

Anaemia. This was found in 30 per cent of men 15 to 20 years after vagotomy and gastroenterostomy. When we set up our prospective study there were at least two aims; one was to see whether anaemia would be as common following vagotomy and pyloroplasty, and the second was whether one could predict its occurrence before it became severe.

Figure 2 illustrates the mean haemoglobin levels of the men in our study. Each point on this graph represents the mean haemoglobin level of all men reviewed at each follow-up interval. Patients at a previous follow-up found to be anaemic were excluded, to avoid the bias that they might introduce into the results.

It will be seen that by five years there has been a significant fall in the mean haemoglobin level. Further-

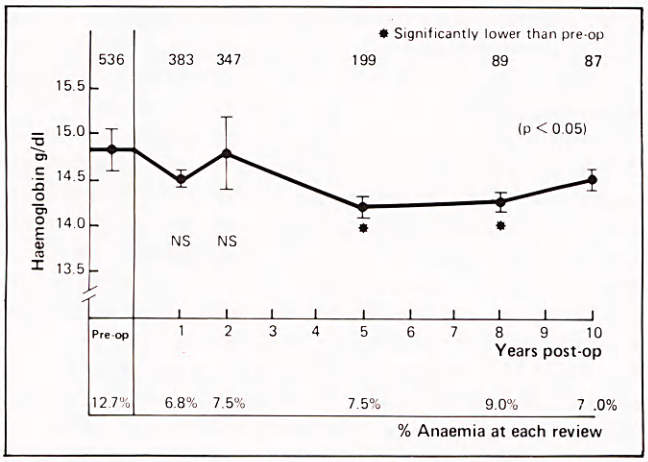

Fig. 2. *Mean ± S.E. haemoglobin levels before and 1, 2, 5, 8 and 10 years after operation. Percentage of patients anaemic at each follow-up are recorded below.*

more, at each follow-up point, between 7 per cent and 10 per cent of patients were found to be anaemic. To determine whether one could predict those who would become anaemic, we plotted the trends in haemoglobin levels (Fig. 3) of the patients who became anaemic at 5, 8 and 10 years after operation. It will be apparent from this graph that prediction from the haemoglobin was impossible.

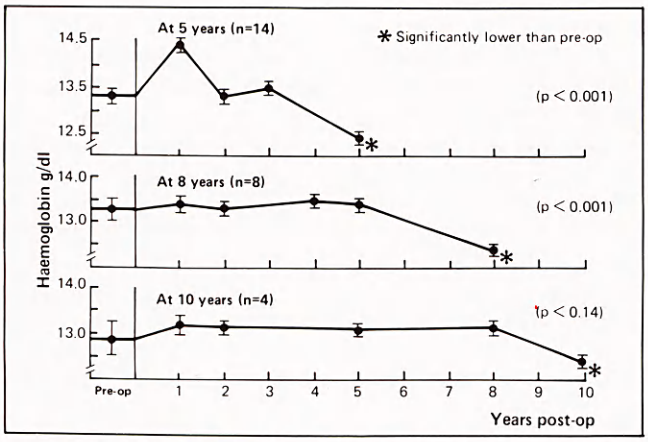

Fig. 3. *Mean ± S.E. haemoglobin levels pre-operation and at follow-up in patients who became anaemic.*

Serum Iron. At each follow-up attendance, blood was taken for serum iron estimation. There was no significant trend in serum iron level with time. It would seem unlikely, therefore, that iron measurement could be used to predict the patients who would become anaemic.

Serum Protein and Calcium (Fig. 4). Analyses of total protein and serum calcium were made at each review. Immediately after operation there was a significant rise in the total protein, which persisted thereafter. In view of our data on weight loss, it is unlikely that this reflects improved nutrition. More probably it represents a reduction in protein loss with healing of the ulcer.

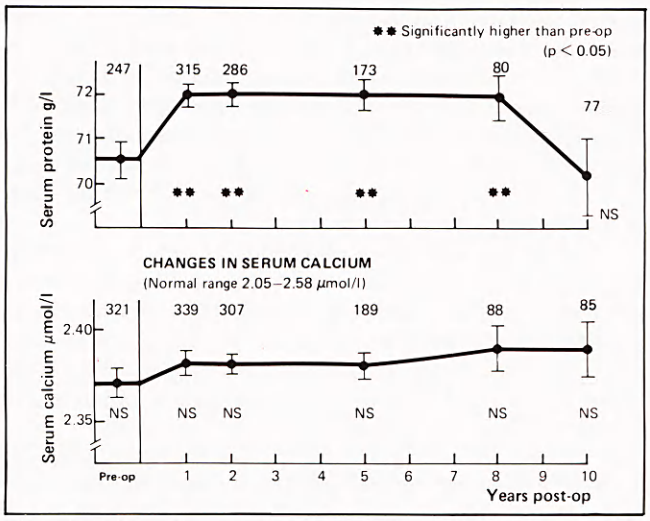

Fig. 4. *Plots of mean total protein and serum calcium measured before operation and at each follow-up. Normal range 60-80 g/litre.*

Serum calcium also rises slightly following operation but this is not significant and there is no obvious trend in calcium thereafter. Certainly there is no evidence of progressive hypocalcaemia which might be expected if metabolic bone disease was developing.

Alkaline Phosphates (Fig. 5). After operation, there is a slow but progressive rise in this enzyme with time. This cannot be explained by ageing of the population, as there is still a significant difference when pre-operative patients are compared with age-matched post-operative patients.

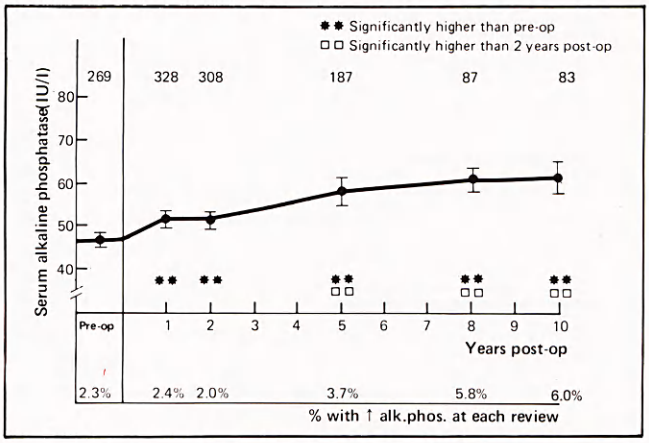

Fig. 5. *Mean ± S.E. serum alkaline phosphatase before and after vagotomy and pyloroplasty. Normal level 20-90 iu/litre.*

We have no explanation for this finding. However, the isoenzyme was principally intestinal in type in a few patients with high levels, so it seems possible that bacteriological colonisation of the gut following the operation could explain this trend.

Discussion

This study has demonstrated that gastric surgery is associated with a number of adverse effects that can occur at any time following operation. While proximal gastric vagotomy has a somewhat lower incidence of dumping and diarrhoea, the differences found in our study are not as striking as has been suggested by others[4, 8, 9] and in view of the higher incidence of recurrent ulceration following this operation reported by other groups[10, 11, 12], its general adoption as the routine procedure for surgical management of duodenal ulceration should still be regarded with caution. As far as vagotomy and pyloroplasty is concerned, we have shown that adverse effects and long-term metabolic sequelae appear to be as common after this operation as after other previously described operations. Clearly we have no 'ideal' operation to recommend for the surgical treatment of duodenal ulcer. It is incumbent upon us, therefore, to be extremely careful in selecting patients for operations and to recognise that surgery inevitably means taking on the possibility of these long- and short-term adverse effects. Obviously, this must be considered as a factor when the relative merits of medical or surgical long-term therapy are considered.

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