THE EFFECTS OF GASTRIC OPERATIONS

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WHEN CONSIDERING THE efficacy of an operation for gastric or duodenal ulcer we weigh up the balance of the good effects of the operation in curing the ulcer and the bad effects that give rise to post-operative symptoms. The operations currently used to treat ulcers are partial gastrectomy and vagotomy combined with a drainage procedure. These operations cause gastric hypo- or an-acidity. They remove the antrum, which is part of the stomach responsible for the churning and mixing of food with peptic juice. They destroy the pyloric mechanism that normally regulates the dispensing of boluses of food and peptic juice through into the small bowel (the so-called 'hopper' effect), and finally when a gastrojejunal anastomosis is made they bypass the duodenum. These alterations of upper gastro-intestinal physiology combine to produce the necessary diminution in digestive power of the gastric secretion to prevent the patient developing further peptic ulceration. However, they may bring in their train the liability to a number of undesirable symptoms which mar the good effects of the ulcer cure.

There are three main groups of symptoms which are sometimes dignified by the title of postgastrectomy syndromes. These are:

1. dumping,
2. regurgitation or vomiting, and
3. diarrhoea.

Furthermore (4), there is often a diminution of the capacity to absorb minerals and vitamins which may lead to chronic deficiency states. These bad effects will now be considered in detail.

1. Dumping

Dumping is a term used to describe the unpleasant symptoms that may occur after a meal in a patient who has had a gastric operation that allows rapid emptying of food which is 'dumped' into the small intestine. The symptoms include sweating, a feeling of unpleasant warmth, subjective flushing, nausea, fullness, palpitation, borborygmi and sometimes diarrhoea. To a greater or lesser degree these symptoms may occur after any gastric operation except possibly vagotomy without a drainage

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procedure. The more extensive the gastric operation the greater the likelihood of dumping developing. A subtotal gastrectomy with a gastrojejunal anastomosis appears to be the most likely to produce dumping, and pyloroplasty the least. However, many patients with a total gastrectomy do not experience dumping, while others with vagotomy and pyloroplasty do. There is therefore some inborn predisposition to dumping and it is claimed that the liability to dumping can be predicted before operation by test feeding the patient with 50 gm. of glucose solution through a tube that has been guided through into the upper small bowel19, 13.

There are three important causative factors:

(a) the rapid passage of a meal through into the small bowel as occurs after most gastric drainage operations;

(b) the tonicity of the ingested meal; when a hypertonic solution is in the jejunum rapid equilibration of tonicity takes place and large volumes of fluid accumulated within the lumen of the gut. This is associated with a diminution in splanchnic venous return and a rapid distension of the gut. The gut distension contributes to the symptomatology either by giving rise to afferent autonomic nervous stimulation or by releasing a hormone, possibly serotonin, from the gut wall; and

(c) a natural predisposition to dumping. Such a predisposition is often associated with a past history of allergic states such as hayfever, asthma or eczema or with some psychological instability. It is unusual to find a patient who is physically and psychologically stable suffering from severe dumping symptoms.

Fortunately after gastric operations the severity of dumping symptoms tends to diminish with the passage of time. By the end of the first operative year half the number of patients who initially experience dumping continue to have attacks. Part of this improvement is due to the patients learning to avoid the provoking foods but part also appears to be due to the body's physiological adaptation. The keynote, therefore, of the treatment of patients who complain of dumping immediately after the operation is patience and dietary advice. Drug therapy with insulin, tolbutamide and anti-serotonin drugs has been advocated. In my experience none of these has had significant practical value. No patient on whom I have tried them has ever been sufficiently impressed to want to continue the treatment indefinitely. Surgical treatment has been advocated for the dumping syndrome. Operations have even been suggested to re-create artificial pyloric sphincters. We have been disappointed with the conversion of a Billroth II (Polya) operation to a Billroth I anastomosis; less than 50% of the patients so treated had any significant symptomatic relief35. The conversion of a gastroenterostomy to a pyloroplasty or the interposition of a retroperistaltic jejunal loop between the stomach and duodenum have produced a few dramatically
successful results. In my opinion, revisional surgery should be very much a last resort in the management of patients with the dumping syndrome. Many other symptom complexes that have been dignified by the title of separate syndromes, such as 'small stomach syndrome' or 'intestinal hurry' are almost certainly examples of the variations of the dumping syndrome.

2. Vomiting and regurgitation

Many patients after gastric operations experience occasional vomiting or regurgitation of gastric contents. Although these symptoms appear to be worse after the more major forms of gastric resection with gastrojejunal anastomosis, the results of the controlled trial at York and Leeds\(^\text{15}\) suggest that these complaints are just as common in patients who have had a gastroenterostomy. A study of recent reports in the world literature, however\(^\text{7}\), suggests that they are probably less common after pyloroplasty. The factors giving rise to these symptoms are:

(a) any form of stenosis or obstruction of the gastric outlet stoma or, more rarely, obstruction in the small bowel; and,

(b) regurgitation of bile and pancreatic secretion into the gastric remnant. The latter is of particular importance, and is also thought to damage the remaining gastric mucosa. It is likely that the bile salts in particular may damage the protective mucous barrier which normally protects the gastric mucosal cells from peptic drainage. There is evidence to suggest that the copious bile vomiting that occasionally occurs after gastrojejunal anastomosis (the so-called 'afferent loop syndrome') is caused by free regurgitation of duodenal juices into the stomach rather than by obstruction to the afferent loop\(^\text{32}\).

Further effects of regurgitation into the stomach may therefore be gastric atrophy and gastritis. The gastritis is probably an important factor in causing the anorexia which in its turn is an important cause of weight loss after gastric operations.

Medical therapy has little to offer in the management or control of regurgitation and vomiting although sleeping propped up in bed may help the patient with nocturnal regurgitation and oesophagitis. The association of malnutrition and anaemia will often make the symptoms very much worse and any step taken to correct these deficiencies may help the suffering patient.

Surgical treatment is occasionally indicated in patients who are severely handicapped. This may be directed towards preventing gastric regurgitation into the oesophagus or duodenal regurgitation into the gastric remnant. If there is no associated hiatus hernia it is more profitable to direct the surgical treatment towards preventing reflux into the stomach. This may be effected by an isoperistaltic jejunal interposition between the
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stomach and the duodenum or the creation of a roux-en-Y with an efferent loop of at least 15 cm. in length to prevent regurgitation back into the stomach. The results of these forms of surgical treatment are good in well selected cases but are obviously not indicated in patients with only minor degrees of regurgitation or anorexia.

3. Diarrhoea

Diarrhoea after gastric operations takes one of two forms. It may be continuous with frequent loose or watery stools every day or episodic in which a normally regular bowel habit is punctuated by short attacks of water diarrhoea often associated with urgency.

*Continuous diarrhoea* is a less common problem and appears to occur with equal frequency after gastric resections or after vagotomy with drainage. In its milder forms this diarrhoea does not necessarily warrant investigation or treatment, particularly if the patient's general nutrition is maintained. In patients with the severer forms of continuous diarrhoea a full investigation is indicated. Mild degrees of steatorrhoea with a daily faecal fat output of up to 10 g. are quite common after partial gastrectomy and is not usually associated with troublesome diarrhoea. Severe degrees of steatorrhoea, however, with daily faecal fat output of 20 or 30 g. has always, in our experience, been associated with some underlying abnormality. We have investigated many patients with continuous diarrhoea after gastric resection and have detected at least two patients with a gluten enteropathy (adult coeliac disease) and two patients with chronic pancreatitis and pancreatic exocrine insufficiency. In these patients the defects were almost certainly present before operation and their effects merely accentuated by the gastric operation. We have also investigated such patients and found gross colonization of the upper gastro-intestinal tract with colonic type organisms and have assumed that these patients have a variation of the blind loop syndrome and when a gastro-jejunal anastomosis has been present have claimed the blind duodenal loop as the source of persistent infection. Long-term anti-biotic therapy has been of some value in controlling the symptoms in these patients but has not been dramatically or permanently successful. In one patient's conversion to a Billroth I anastomosis, an abolition of the 'blind loop' abolished neither the colonization nor the symptoms.

A relatively rare but important cause of acute profuse diarrhoea is the development of a gastro-colic fistula due to stomal ulceration. In the three patients that I have treated with this complication, profuse debilitating diarrhoea was the major presenting feature.

It must always be remembered that some diarrhoea is often a feature of the Zollinger-Ellison syndrome, and association between peptic ulcer and diarrhoea should lead to this diagnosis being considered.
**Episodic diarrhoea** is a common problem, particularly after operations associated with a vagotomy, although many have claimed that diarrhoea after vagotomy and drainage is due to the drainage more than to the vagotomy. There is no doubt that the episodic type of diarrhoea is very much more common after vagotomy and drainage than after the simple drainage procedures alone, such as gastroenterostomy or after subtotal gastric resection. Patients usually have a normal regular bowel habit interspersed with unheralded attacks of urgent watery diarrhoea. This typical progression of events suggests that patients with a vagotomy might be more susceptible than others to the intermittent development of gastro-intestinal infection. However, so far there has been no widespread acceptance of this view or convincing proof of the episodic fluctuation in the upper gastrointestinal flora in association with attacks of diarrhoea. Reliable evidence is beginning to appear that suggests that selective vagotomy may reduce the incidence of episodic diarrhoea below that experienced with truncal vagotomy. In our study of patients with episodic diarrhoea after truncal vagotomy it appears that the symptoms become much less troublesome with the passage of time. Because of this continual improvement in the tendency towards episodic diarrhoea after vagotomy the initial treatment should be essentially symptomatic, the patients benefiting from dietary advice, particularly if directed towards the avoidance of milk products and wet, sweet foods. Symptomatic treatment with codeine and related compounds is of little value when the attacks of diarrhoea are unheralded. However, such simple treatment may tide patients over the early difficult months and should always be tried in preference to the more radical forms of reconstructive surgical treatment that have been advocated. Some authors have recommended interposition of reversed small bowel loops in an attempt to control postvagotomy diarrhoea. Although undoubtedly successful in some patients, radical surgical treatment should rarely be recommended.

4. **Deficiency states**

It has long been recognized that many patients after gastric operations may develop deficiencies of iron, Vitamin B₁₂, folic acid and other minerals and vitamins essential for bone development. It is also well recognized that such deficiencies develop insidiously and may not present as an obvious clinical syndrome until 5, 10 or more years after the original gastric operation. Because of the insidious onset of these deficiency states they may escape detection until the patient is severely disabled. Because of this it is important that the possibility should be recognized by surgeons, physicians and medical practitioners responsible for the care of these patients after their gastric operation.

**Iron deficiency**: minor degrees of iron deficiency are extremely common, particularly in women before the menopause, even in the absence of any gastric surgery, so that any estimate of the incidence of this state after
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gastric operation should always be compared with that in a control population. There is, however, good evidence that chronic iron deficiency is very common after major gastric resection\(^8, 24\). More recent studies have shown that after vagotomy and drainage procedures there is a significant fall in the patient’s haemoglobin levels. Furthermore, it appears likely that this fall in haemoglobin may occur less frequently when pyloroplasty is the drainage procedure than when gastrojejunostomy or antrectomy is employed\(^30\). In the conclusion of their study Pulvertaft and Cox said: ‘It is now clear that haematological deficiencies follow vagotomy with drainage procedures more commonly than was once supposed. There is good evidence that the problem is less serious after vagotomy than after gastrectomy. However, the severity and extent of the problem remains to be elucidated by investigations with patients with long intervals after vagotomy with differing drainage operations.’\(^30\)

The cause of the iron deficiency is probably not due to a diminution in the oral intake of iron but is due to a combination of impaired absorption and minimal increase in iron losses following gastric surgery.

All patients with chronic iron deficiency following gastric operations respond well to simple oral treatment with iron compounds. It has been my experience that parenteral iron therapy is only rarely required and that once the anaemia has been corrected the patients are readily maintained on a regular small oral supplement; one tablet of a simple iron preparation taken each night is almost always sufficient to maintain these patients in a normal haematological state.

_Vitamin B\(_{12}\)_ deficiency: Examination of unselected patients at least five years after partial gastrectomy for peptic ulcer shows that 15\% to 60\% have low serum levels of Vitamin B\(_{12}\). Two per cent to 6\% have megaloblastic or transitional megaloblastic anaemia\(^9, 20\). The cause of impaired Vitamin B\(_{12}\)_ deficiency is impaired absorption of the vitamin due to decreased production of intrinsic factor associated with gastric mucosal atrophy\(^2, 23, 10, 21\). Post-gastrectomy Vitamin B\(_{12}\)_ deficiency may be followed by all the complications associated with Vitamin B\(_{12}\)_ deficiency from whatever cause: peripheral neuropathy and subacute combined degeneration of the cord have both been reported\(^28, 1, 4, 14, 33\).

After vagotomy and simple drainage procedures the incidence of gastric atrophy, intrinsic factor deficiency is low and detectable deficiencies of Vitamin B\(_{12}\) are rarely reported.

Vitamin B\(_{12}\)_ deficiency appears, therefore, to be the important cause of disability after major gastric resection and should always be borne in mind in the post-operative supervision of these patients. After vagotomy and simple drainage procedures the incidence of Vitamin B\(_{12}\)_ deficiency is sufficiently uncommon for this to be disregarded in the regular supervision of these patients.
Although there is some evidence to suggest that oral supplementary $B_{12}$ therapy might well be sufficient to keep patients haematologically normal after gastric resection it is more usual to rely on the detection of Vitamin $B_{12}$ deficiency on routine post-operative screening tests and to treat the patients with established deficiency by regular monthly injections of Vitamin $B_{12}$.

**Folic acid**: The problem of folic acid deficiency appears to be similar to that of Vitamin $B_{12}$ deficiency in that it is a common state after major gastric resection but is uncommon after vagotomy and simple drainage. Estimations of serum folate levels indicate that, many years after gastrectomy, folic acid deficiency may be present in from 6% to 12% of patients. Like iron and Vitamin $B_{12}$ deficiency, folic acid deficiencies take several years to become manifest. Deller found the incidence of low serum folic acid activity to be 4.2% in 24 patients examined less than six years after operation and 14.5% in 76 patients examined more than six years after operation. Studies of patients after vagotomy and gastrojejunostomy have tended to indicate that folic acid deficiency is not a serious problem.

Although there may be some indication for treating post-gastrectomy patients with folic acid in addition to iron and $B_{12}$ supplementary therapy, there appears to be no evidence in favour of prescribing regular folic acid therapy in patients after vagotomy and simple drainage.

**Metabolic bone disease**

The detection and assessment of metabolic bone disease after gastric operations presents much greater difficulty than does the assessment of haematological abnormalities. It is largely for these reasons that there are apparent differences of opinion, some authors considering that after gastric resection metabolic bone disease is a serious problem, others considering it to be relatively uncommon.

The two major metabolic bone diseases that have been studied are osteomalacia and osteoporosis.

**Osteomalacia** is due to a deficiency of Vitamin D. Even after gastric resections the incidence of unquestioned osteomalacia is only 2%. However, when this condition occurs it may lead to serious and permanent disability. It is important, therefore, to recognize its possible occurrence after gastric resection. It appears to be due to a combination of factors, chiefly the poor oral intake of fat-containing foods and possibly an impaired facility of absorption usually associated with the general malabsorption of fats.

Although frank osteomalacia has been reported after simple gastro-enterostomy, osteomalacia has not been reported in long-term follow-up studies of the patients after vagotomy and pyloroplasty or vagotomy and gastroenterostomy.
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In established cases of osteomalacia after gastric resection, permanent Vitamin D therapy is indicated and may usually be given orally. It is more difficult, however, to make out a case for regular supplements of Vitamin D in patients with no established disease, particularly in view of the potential dangers to the kidney of long-term Vitamin D therapy.

Osteoporosis is more difficult to detect and to quantitate than is osteomalacia. Morgan et al.26 investigated the problem by studying the radiological thickness of the cortex and the metacarpal bones and found that 'thin' bones were commoner in patients after partial gastrectomy than in patients of the same age with unoperated ulcers but they did not find any difference between the normals and the patients after vagotomy and simple drainage. Furthermore, they found that these changes of osteoporosis, which are quite common in normal people with increasing years, occur at a younger age in patients who have had a partial gastrectomy. There was no detectable variation from normal in patients who had a vagotomy and simple drainage. It is not clear whether this thinning of the cortices of the bone after gastric operation is due to deficiencies of calcium or protein or to any other factor. It appears that bones 'age' earlier in patients after partial gastrectomy.

REFERENCES

J. ALEXANDER WILLIAMS


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**PROCEEDINGS OF THE COUNCIL IN DECEMBER**

At a meeting of the Council on 10th December 1970, with Sir Thomas Holmes Sellors, President, in the Chair, the deaths of Mr. Eric L. Farquharson of Edinburgh (Member of Council and Past Member of the Court of Examiners) and of Professor Martin A. Rushton (Past Dean of Faculty of Dental Surgery) were reported with deep regret.

The following were elected members of the Court of Patrons:

Mr. Philip Sydney Henman (Past Chairman, Transport Development Group Limited);

Mr. Ernest John Partridge (President, Confederation of British Industry).

Sir Henry Osmond-Clarke, K.C.V.O., C.B.E., F.R.C.S., was appointed the Bradshaw Lecturer for 1971.

Professor J. G. Robson, F.F.A.R.C.S., was invited to be the Joseph Clover Lecturer for 1972.

Dr. Lester Cahn, F.D.S.R.C.S., was appointed Menzies Campbell Lecturer for 1968–71.

The award of the Evelyn Sprawson Prize (Faculty of Dental Surgery) to Paul Derek Robinson, L.D.S.R.C.S., of Guy's Hospital Dental School was reported.

One Licence in Dental Surgery was granted.

One Diploma of Membership was granted.

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