

Current Practice

DISEASE OF THE DIGESTIVE SYSTEM

Postgastrectomy Problems—II

J. ALEXANDER WILLIAMS,* CH.M., F.R.C.S.

(Last week Mr. Williams considered the cause and management of some symptoms arising after partial gastrectomy. The article continues with an account of other common complaints.)

Loss of Weight

Many patients lose weight or fail to gain weight after gastrectomy. Those who are undernourished at the time of the operation seem to be at the greatest risk.

Some degree of malabsorption of food is common after gastrectomy. It is only a malabsorption of fats that is important. Carbohydrate digestion and absorption as judged by xylose and glucose absorption tests are almost always normal. Although intestinal intubation studies have suggested that protein digestion is impaired by gastrectomy, it is unknown for protein deficiency to occur as the result of gastrectomy provided that oral intake is normal and there is no excess loss. Malabsorption of fat is the easiest form of malabsorption to detect, as the residuum is passed relatively unaltered and can be measured in the faeces. The normal person on an average intake excretes in the faeces no more than five grammes of fat per day. After partial gastrectomy the average daily output of fat in the faeces is from seven grammes (with a Billroth I anastomosis) to ten grammes (with a Billroth II anastomosis).

It is the diminished intake that is the most important cause of weight loss after gastrectomy. The causes of the diminished intake are: lack of appetite, early satiation after a small quantity of food, and fear of provoking postcibal symptoms. The analysis of these three factors is the initial step in the evaluation and treatment of weight loss.

Appetite.—This is determined by a number of apparently unrelated factors, including low blood sugar, empty stomach, and feeling of mental and physical wellbeing. The patient may have a poor appetite because he takes too much tobacco or alcohol or because he is deficient in minerals or vitamins. Iron deficiency is particularly likely to impair the appetite, so to a lesser extent do deficiencies of vitamin B₁₂ and folic acid. In the correct evaluation of the postgastrectomy patient all these factors must be assessed. Treatment may not be easy, particularly of those patients who poison themselves with nicotine or alcohol. For those who suffer from, but do not deserve, a poor appetite even after the correction of deficiencies some form of gastric stimulation before meals may be advised. The French gastroenterologist will advocate a glass of wine, the American chilled orange juice, while the British adviser may prefer sherry.

Early satiation will depend on the size of the gastric remnant, its speed of emptying, and its ability to distend. The more radical the gastric resection, the greater the likelihood of loss of weight. This is a generalization that needs qualifica-

tion and it is unwise to suggest treating weight loss due to a feeling of fullness after meals by reoperation to increase the size of the gastric remnant.

The ability of the gastric remnant to dilate without symptoms may be related to the health of the gastric mucosa. There is a good correlation between the presence on biopsy of atrophic or inflammatory gastritis and the symptom of fullness after meals. General nutritional measures to improve appetite may also increase the capacity to take food. Nevertheless, the simplest way to overcome early satiation is to take small, frequent meals. This is the oldest and simplest of the pieces of advice offered to patients after gastrectomy, but it is still the best.

Fear of Discomfort.—Some of the most severe malnutrition after gastrectomy occurs in patients in whom oesophagitis and regurgitation were provoked by taking food. The patients sought relief from these distressing symptoms in the only way they found effective—by starvation. Treatment is that of the responsible symptom.

Severe malabsorption is relatively uncommon after gastrectomy and always suggests additional disease. This may be pancreatic insufficiency, jejunocolic fistula, regional enteritis, gluten enteropathy, or the intestinal blind loop syndrome.

Diarrhoea

Increased frequency of bowel action is common after gastrectomy, and to many patients cured of both dyspepsia and constipation this is an added bonus of the operation. Many patients complaining of loss of weight, faintness, or vomiting also complain of diarrhoea, though the diarrhoea is not usually disabling.

Some of the important causes of increased frequency and fluid consistency of stool after gastric resection are: increased small bowel transit time resulting from the loss of the gastric emptying regulation once the antrum and pylorus are removed; the osmotic cathartic effect of large quantities of hypertonic foodstuff arriving in the jejunum and attracting fluid into the lumen; the production of large bowel irritants within the gut. Probably the most important factor in this mechanism is the breakdown of bile salts by bacteria. In addition to depriving the digestive tract of an effective emulsifying agent, this breakdown of bile salts liberates powerful colonic irritants which cause diarrhoea. The impaired enzymatic digestion of foods provides more fuel for fermentation and bacterial breakdown in the gut.

As all these factors are present to some extent in all patients after gastrectomy, it is not surprising that mild upsets of bowel habit are common. The practical policy of management is to treat all with simple empirical methods and to reserve sophisticated investigations for those with severe and unremitting symptoms.

* Consultant Surgeon, United Birmingham Hospitals, External Scientific Staff, Medical Research Council.

Diet.—The oldest gimmick known to gastroenterologists is to inquire if any particular food causes diarrhoea and advise its avoidance. Milk and milk products are the commonest provoking agents. The avoidance of all milk products for a trial of two weeks and then observation of the provocative effect of a glass of raw milk should indicate those patients who are sensitive to milk.

Absorbents.—The safest medicaments are the methylcellulose group of water-absorbing compounds. If they produce symptomatic control they can be used indefinitely.

Sedatives.—Sedatives of the codeine or diphenoxylate type can be used and many patients are so controlled permanently. Because of the dangers of habituation they should be used with caution.

Antibiotics.—A short course of broad spectrum antibiotics will often control an attack of diarrhoea. The agents effective against the gram-negative organisms are the most useful, presumably because they abolish or alter the flora of the upper gastrointestinal tract, and in particular control those organisms that cause breakdown of bile salts.

Neomycin, which is not normally significantly absorbed from the alimentary tract, is the safest for prolonged administration, though even this drug may cause mucosal damage. Combinations of tetracycline and nystatin may also be used. Prolonged therapy rarely produces prolonged relief, but intermittent antibiotic therapy may help to control symptoms.

The investigation and treatment of severe or intractable diarrhoea is best accomplished in a specialized gastroenterological unit. Gastrojejuno-colic fistulae may be due to recurrent ulceration. This is investigated by barium enema and barium meal examinations as well as acid secretion studies to evaluate the cause of the ulceration.

Pancreatic insufficiency is investigated by pancreatic function studies with duodenal intubation and hormonal stimulation of the pancreas.

Gluten enteropathy may be demonstrated by peroral jejunal biopsy. The intestinal blind loop syndrome is studied by intestinal aspiration and culture and vitamin B₁₂ absorption tests.

Faintness

The feeling of faintness after meals is the predominant symptom of the so-called "dumping syndrome." The faintness occurs within thirty minutes of taking food and is usually accompanied by one or more of the following symptoms: flushing, sweating, palpitation, anorexia, nausea, and sometimes colic and diarrhoea. This combination of symptoms may occur after any gastric resection, even after pyloroplasty, but is usually worse if there is a small gastric remnant that empties rapidly. After partial gastrectomy almost all patients experience some of these symptoms at some time in the early months after operation, but in most the symptoms are transient. Prolonged complaint of annoying or disabling symptoms occur in less than 5% of most reported series.

The term "dumping" was first used by Mix,³ in 1922, who believed that the symptoms were produced by food being dumped straight into the small bowel. The physiological implications of this syndrome are so fascinating that an increasing amount of research has been devoted to its study. This work has all confirmed Mix's original concept, but it is only in the last five or six years that there has been much significant advance.

There are three essential features of the phenomenon of dumping. The small bowel cannot handle the glucose load of a meal "dumped" into it straight through the stomach. This results in the osmotic retention of large volumes of fluid in the gut with a consequent fall in blood volume, usually greater than 10%.

Symptoms are produced by distension of the small bowel and may occur even if the blood volume is maintained at normal levels by rapid transfusion. The humoral role of serotonin has been postulated but its aetiological significance is not proved.

These symptoms tend to occur in patients who can be shown preoperatively to have a predilection to dumping. These same patients have a more than average incidence of allergic diseases such as hay-fever or asthma. They are also said to have a less than normal mental and emotional stability.

Certain additional observations about these patients help us to keep this problem in perspective. Patients with dumping often develop these symptoms after several symptom-free years. The symptoms are not always constant and they may have weeks or even months of freedom at a time. Dumping is rarely seen in a patient who is otherwise fit and mentally stable.

It will be seen from these observations that any purely mechanistic theory of aetiology cannot be the whole explanation, and a purely mechanistic approach is unlikely to succeed.

Special investigations in these patients are of little practical value in determining the type of treatment and should be employed only if their purpose is to advance knowledge. There is no practical value in determining whether a patient has hyper- or hypoglycaemia during an attack, nor what type of glucose tolerance curve they have, nor yet whether the barium meal empties rapidly from the stomach. Exactly comparable results to these tests would be obtained from many asymptomatic postgastrectomy patients.

Treatment is, once more, empirical on a progressive scale, the first treatment for the least severe and earliest cases.

Reassurance.—Most patients who, soon after operation, experience faintness after meals require nothing more than an explanation and reassurance. They may be reassured that their symptoms are likely to become less and will probably disappear in the coming months.

Correction of Deficiencies.—It is essential in the management of any postgastrectomy problem to consider the possible role of mineral and vitamin deficiencies. Iron deficiency is the commonest abnormality found after gastrectomy and may predispose to "dumping" symptoms. Vitamin B₁₂ and folic acid deficiencies should also be sought and corrected.

Diet.—In addition to reassurance and iron therapy, simple dietary measures may be advised. These are sufficient to control most mild symptoms. Rather than eating, as many do, one main meal each day, the patient should be advised to take three small meals, and, unless obesity is a problem, to take a small snack between each meal. Large carbohydrate meals should be avoided and the caloric intake should be made up with proteins and fats. Restriction of fluid intake at mealtimes also helps to delay emptying of the gastric remnant and relieves symptoms.

Advice to lie down or rest after a meal may be useful in the immediate postoperative period, but is impracticable once the patient returns to work.

Drug Therapy.—The appreciation of the aetiological role of impaired intestinal handling of glucose and the observation⁴ that in patients with "dumping" insulin given before meals prevents symptoms have led to a most significant new therapeutic approach with oral hypoglycaemic drugs. Tolbutamide 500 mg. before each main meal will often control symptoms, and should be a first line of treatment in the patient with severe symptoms. Some success has been claimed with anti-serotonin drugs, and Sullivan⁵ advocated 4 mg. of cyproheptadine before meals in addition to or instead of tolbutamide.

Operative Treatment.—The principle behind attempted surgical treatment of patients with "dumping" is the attempt to delay the rate of emptying of the gastric remnant by narrowing the stoma, rerouting the food through the duodenum (by conversion to gastroduodenostomy), or the creation of an

artificial "sphincter" by placing a reversed jejunal loop between stomach and duodenum. There is, however, rarely if ever any need to operate on patients with "dumping."

Conclusions

The patient who complains of pain, nausea, vomiting, weight loss, diarrhoea, or faintness after partial gastrectomy presents a problem that is rarely easy to evaluate. All symptoms tend to be buried beneath a mound of ineffective antacids.

The essential principle of the approach to the problem is to take a careful history from the patient with no preconceived ideas or with no empty mental pigeonholes labelled "——— syndrome" waiting to receive the problem as soon as the patient admits to the first classical symptom. Then evaluate the symptoms, looking for objective evidence of organic disease. The symptoms of pain and vomiting always require a comprehensive series of investigations, whereas loss of weight, diarrhoea, or faintness are best managed initially by simple empirical methods.

In some patients with recurrent dyspepsia after gastrectomy no definite diagnosis will be made or no permanent cure found. Some of these will be psychologically unstable and others will appear to become so as their symptoms remain undiagnosed and unrelieved. This residual group of unhappy complaining patients taxes the sympathy and resources of anyone who has to deal with many postgastrectomy patients. From several years' experience of reviewing many and reoperating on some of these unfortunate patients, I believe that they frequently need sympathy and continued interest but only rarely benefit from reoperation.

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ANY QUESTIONS?

We publish below a selection of questions and answers of general interest.

Corticosteroids in Pregnancy

Q.—What are the particular hazards in pregnancy and childbirth in primiparae who have been taking corticosteroids continuously for some years for intractable asthma? What may be done to minimize the hazards, and should the baby be given corticosteroids after birth?

A.—The dangers of long-term corticosteroid therapy are due mainly to adrenal suppression, but since pregnancy itself stimulates the adrenals to double the normal secretion of cortisol this effect is minimized.¹ There is usually no need to increase the dose of steroid during pregnancy, but the possibility of steroid deficiency arising during any acute illness remains.

Labour is regarded as any major surgical operation would be, and hydrocortisone is given parenterally throughout this period of stress. Postpartum haemorrhage may produce a more dramatic collapse than in the normal patient, and must be treated with intravenous hydrocortisone as well as blood volume replacement.

Though corticosteroids do cross the placenta, they do not usually produce any noticeable effect on the baby. There is no

reason to give steroids to the baby unless signs of deficiency are manifest.

Patients receiving corticosteroids should be confined in hospital.

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Animal Hosts to Rabies

Q.—Do lemurs contract rabies, and are there any other of the rarer infections which they may convey to humans by biting?

A.—The host specificity of rabies virus is low and the range of susceptible species includes man, domesticated mammals, and many wild ones, including foxes, wolves, mongoose, skunks, rodents, and certain kinds of bat. Monkeys can be infected experimentally, and the first natural case was recently recorded¹ in a rhesus monkey (*Macaca mulatta*) which had been imported into Britain from India. Lemurs are primitive primates of the suborder *Prosimii*. Though I know of no report of rabies in these animals, the possibility cannot be ruled out in view of the wide host-range of the virus, and it should not be ignored because

of the extreme danger attending the bite of an infected animal.

Monkeys may also, while showing trivial or no mouth lesions, harbour herpes B virus (*Herpesvirus simiae*), which, if transmitted to man, causes a serious and usually fatal condition. B-virus infection has been detected in many Old World monkeys,² and, though lemurs have not been examined, the risk of infection should be considered after any bite inflicted by these animals.

Local sepsis or septicaemia resulting from infection by mouth flora are also possible, and it might be remembered that pulmonary tuberculosis, with the production of infective sputum, can occur in captive primates. Extreme caution should be the watchword where primates are being kept. Every effort should be made to avoid contact with the animals or material derived from them, in particular from the mouth, and bites should be regarded most seriously as matters for immediate and thorough surgical attention.

During 1966 only one licence was issued, under the Animals (Restriction of Importation) Act, 1964, for the importation of a lemur—a *Lemur catti* from a French zoo. On the other hand, licences covering more than 20,000 Old World monkeys, required mainly for scientific purposes, were granted.³

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