Current Practice

DISEASE OF THE DIGESTIVE SYSTEM

Postgastrectomy Problems—I

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Partial gastrectomy has an excellent record in the treatment of peptic ulcer; in the well-chosen case its successes far outweigh its disadvantages. Nevertheless between 5% and 15% of patients continue to complain of serious symptoms after operation. As the number of patients subjected to partial gastrectomy in England and Wales in 1961 was approximately 26,000, if 10% of these develop some form of "postgastrectomy syndrome" then this will not be a rare problem. The commonest symptoms of patients who complain after gastrectomy are abdominal pain, vomiting, loss of weight, loss of energy, attacks of faintness, and diarrhoea. These symptoms usually do not occur singly but combine in an infinite variety of complexes that have become known as the "postgastrectomy syndromes." There is a good case for discarding the present concept of these as classical syndromes.

Many syndromes have been described, including those prefixed by the term dumping, afferent loop, and even efferent loop, long loop, and small stomach. To list these syndromes and describe their classical clinical pictures may help to place some of the known facts in an orderly fashion, but it is misleading in that it implies knowledge of the pathogenesis of the patient's symptoms. To categorize a patient as having the "afferent loop syndrome" implies that we know the cause of his symptoms and—often incorrectly—blame the afferent loop for their cause. Even the most closely studied and best understood postgastrectomy disease, the "dumping syndrome," has in its name an implied aetiology which explains only a small part of the syndrome. A further confusion is that the term dumping syndrome implies a different disease in different parts of the world. To some doctors the term is restricted to the feeling of faintness, nervousness, and flushing provoked by hypertonic foods, while others use it to encompass symptoms of vomiting, pain, and diarrhoea, or almost any symptom that occurs after gastric operations. Dumping may be a useful term to retain but only in the former limited sense.

This article will therefore be restricted to an evaluation and investigation of the symptoms of which patients complain, and readers who require descriptions of the variously defined syndromes are referred to the bibliography.

There are two groups of complications which usually present in different ways. Patients with abdominal complications such as pain, vomiting, or diarrhoea usually complain of these symptoms and demand attention from their doctor, while many of those with loss of weight, lack of energy, and the less dramatic symptoms of nutritional deficiency do not readily report for medical attention. However, the frequency of the nutritional complications is sufficiently high to justify a planned, even though infrequent, follow-up programme. The nutritional complications will not be considered in detail in this article.

Abdominal Pain or Discomfort

The patient who complains of continued or recurrent indigestion after an operation designed for the cure of this symptom needs careful investigation.

Once it is established that the pain or discomfort is associated with digestion and is not due to cardiac ischaemia or renal disease, it is then necessary to differentiate between recurrent peptic ulceration, oesophagitis, and biliary or pancreatic disease.

A carefully taken history and physical examination are essential, though these rarely establish the diagnosis with any certainty. Special investigations are therefore essential, and a programme should be designed to permit a thorough evaluation with the minimal discomfort to the patient. A full investigation is important, for it is never safe to assume that the presence of, say, oesophageal reflux or gall stones necessarily means that this is the cause of the patient's symptoms.

Haematological and Biochemical Screening

At the initial consultation a venous blood sample should be examined for haemoglobin and absolute indices. In addition, the level of serum vitamin B₁₂ should be estimated, as B₁₂ deficiency is almost invariably incompatible with recurrent ulceration. Estimations of serum bilirubin, alkaline phosphatase, and amylase are of value as indices of biliary or pancreatic disease. A high serum calcium level will suggest disease of the parathyroids and possibly of the pancreas. If laboratory facilities for plasma pepsinogen estimations are available this is a valuable screening test for recurrent ulceration.

The stools may be tested for occult blood, though I find this test of relatively little value even when repeated on successive days. A positive test can occur in so many conditions—stomal ulceration, erosions, oesophagitis, or carcinoma of the gastric remnant.

Radiography

The next least disturbing investigations are radiological; each patient should be studied with a plain abdominal film, a cholecystogram, and a barium meal. The erect and lateral abdominal films may show biliary or pancreatic calcification or distension of the gastric remnant or small bowel with air or fluid.

A cholecystogram is advisable in all patients. I have seen patients who have had vagotomy and even revision gastrectomy for symptoms that eventually were cured only by cholecystectomy. Gall bladder disease had not been suspected because the gall bladder had been judged normal at the original gastrectomy. It has been suggested that gastrectomy pre-

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disposes to biliary disease. Though this hypothesis lacks proof, the association is sufficiently common always to be considered in differential diagnosis and complete evaluation.

Barium contrast radiology is the most useful single investigation in the detection of stomal ulceration. Despite the popular belief that it is difficult to establish a diagnosis on barium meal examination, I have found that in all of ten cases of proved recurrent peptic ulceration there was a large crater demonstrated radiologically.

Some experts have advocated gastroscopy and gastro-photography as more reliable investigations than radiology in the diagnosis of these lesions, though endoscopy is less well tolerated by the patient than radiology. It is true that at gastroscopy it is often possible clearly to see the stomal ring and the jejunum beyond, and to see surface "erosions" at the stoma that are not detected on x-ray. However, it is also possible, even on an apparently satisfactory gastroscopy, to fail to see a large recurrent ulcer, while the examination is often made difficult by the reflux of bile and duodenal fluid. This is an examination for the few experienced experts. Too great an importance should not be given to the presence of stomal erosions. In my experience it is not justifiable to class these as recurrent ulceration and treat accordingly. It is only those cases with unequivocal stomal or jejunal ulcer craters that respond satisfactorily to surgical treatment. It is a commoner error to diagnose recurrent ulcer where none exists than to fail to diagnose it on a good x-ray examination.

In addition to the search for recurrent peptic ulceration on barium meal, it is necessary to look for evidence of hiatus hernia and oesophageal obstruction or reflux. The presence of oesophageal reflux or hernia does nothing more than suggest that the patient might have oesophagitis. The suggestion needs confirmation by the next series of investigations.

Secretion and Provocation Tests

Acid Secretion Tests.—The radiological sign of an ulcer is a constant barium-filled niche or pocket. The finding of such a niche, however "classical" its appearance, can be labelled as a peptic ulcer only if the patient's gastric secretion will permit peptic digestion. Stomal ulceration is often erroneously diagnosed in postgastrectomy patients who are hypochlorhydric or even achlorhydric. To substantiate the diagnosis of stomal ulcer, therefore, it is necessary for the secretion to be able to reduce the gastric pH to below 3.5. This can be tested either by measuring intragastric pH or by aspiration under conditions of maximal stimulation with histamine or gastrin or their analogues. In these tests it is essential to place the pH probe or aspirating tube accurately under fluoroscopic control. In practice some tests are unsatisfactory and a second test is always worthwhile if the diagnosis is in doubt.

Acid Provocation Tests.—It has been claimed that the presence of a stomal ulcer can be confirmed by lowering the pH of the gastric remnant by N/10 HCl infusion via a nasogastric tube and provoking the symptoms of which the patient complains. In my experience this test is unreliable and is more likely to produce the symptoms of oesophagitis than of stomal ulcer (Fig. 1).

FIG. 1.—Acid provocation test using N/10 HCl. For stomal ulcers (A) the test is not reliable; but it is valuable for oesophagitis (B).

The test is, however, of value in confirming that the patient's symptoms may be due to oesophagitis, which may be present even when it is not possible to demonstrate oesophageal reflux on one barium examination.

The oesophagitis provocation test is performed by slowly perfusing N/10 HCl into the lower oesophagus through a nasogastric tube. If this perfusion produces the symptoms of which the patient is complaining, then it is likely that these are due to oesophagitis. The test is also positive in patients with a bile oesophagitis; so the cause of the inflammation can be determined only by means of intra-oesophageal aspiration or pH monitoring. It is always advisable to confirm the final diagnosis of oesophagitis by endoscopy and biopsy. As these are the most unpleasant and hazardous of the investigations of oesophagitis they should be reserved for final confirmation.

Management of Recurrent Dyspepsia

Once having reliably evaluated the cause of the patient's dyspepsia, the problem of treatment remains. Stomal ulceration¹ and pancreatitis² require detailed consideration beyond the scope of this article. The treatment of gall bladder disease presents few problems but that of oesophagitis is difficult. Non-acid oesophagitis may be alleviated by a conversion gastrectomy with an interposed jejunal loop, while oesophagitis with a hiatus hernia may warrant surgical repair of the hernia. The results of surgery are, however, sufficiently unreliable to make it worthwhile first attempting symptomatic control by simple measures of posture, local anaesthetics, or polysiloxane compounds.

Vomiting

Patients complaining of vomiting after partial gastrectomy are usually difficult to evaluate and to treat. A few will have some form of organic obstruction and in them diagnosis and treatment are usually straightforward.

The symptoms that suggest an organic obstruction are unremitting vomiting with loss of weight and dehydration occurring in a patient who had previously been well since his gastrectomy. Upper intestinal dilatation may be detected on physical examination.

The x-ray signs are, once again, the most informative. On the erect and supine plain films dilatation and fluid levels may be seen in the upper gastrointestinal tract. Contrast studies with barium usually confirm the site of the organic obstruction and may indicate its cause. Recurrent ulceration, external compression of the bowel, or carcinoma of the gastric stump may be seen. Gastric carcinoma should always be suspected, as there is evidence to suggest that partial gastrectomy increases the risk of its development in patients with peptic ulcer. The radiological picture is often confusing, and suspicion of carcinoma should always lead to confirmatory tests by exfoliative cytology or endoscopy. Ulcerogenic endocrine tumours or "retained gastric antrum" should also be suspected, particularly if gastric acid secretion is high.

The treatment of an organic obstruction is almost invariably surgical. Extrinsic obstruction from bands of adhesions respond well to reoperation only when there is unequivocal evidence of proximal dilatation. The patient with vomiting and no definite sign of obstruction who is subjected to reoperation and has only bands of adhesions divided is rarely cured for long of his symptoms. It is likely that his vomiting is not due to the adhesions but rather to one or more of the many factors discussed below.

No Organic Obstruction

When, as in the majority of patients complaining of vomiting after gastrectomy, there is no x-ray evidence of obstruction,

evaluation and treatment are much more difficult. The so-called "afferent loop syndrome" is classically described as post-cibal discomfort or pain, relieved by vomiting bilious fluid, and associated with weight loss and malabsorption. The suggested aetiology is an intermittent obstruction of the afferent loop of a Billroth II type gastrectomy. X-ray studies of patients with copious bile vomiting show that there is no afferent loop obstruction and that symptoms occur when the stomach is distended by the juice ejected rapidly from the afferent loop. In 19 patients with these symptoms abolition of the afferent loop by conversion to a Billroth I gastroduodenostomy has not always cured the symptoms. True afferent loop obstruction does occur but is rare. It is uncommon to find a surgically remediable cause for the symptoms of nausea and vomiting after gastrectomy. In those patients with no radiologically demonstrable obstruction it is important to study the emesis threshold, which varies widely.

Three factors need to be considered: emotion, cardio-oesophageal competence, and gastric sensitivity.

The emotional factors are the most difficult to assess, but they are probably the most important. Some patients give a history of easy vomiting since childhood and were often subject to emotional or travel sickness. Vomiting was often a predominant symptom before gastrectomy, even in the absence of any ulcer stenosis. Other patients experience vomiting later in life as a manifestation of endogenous depression.

Cardio-oesophageal incompetence may be one reason why some patients with nausea vomit easily. Incompetence may have been present before operation; however, there is some evidence that partial gastrectomy interferes with normal competence, possibly by opening out the cardio-oesophageal angle. The incompetence may be assessed by barium examination, but because it can be an intermittent phenomenon it may not easily be detected.

Some patients have a stomach or stomach remnant that is, in some way, more than normally sensitive to stimuli. The stimulus may be distension, but in some cases it appears to be by direct mucosal stimulation. Patients with bilious vomiting after gastrectomy appear to be sensitive to the presence of duodenal juice in the stomach. If their symptoms are provoked by a meal, it is often possible to aspirate bile-stained fluid from their stomach and so relieve their symptoms. The symptoms are brought on again by reintroducing the fluid back into the stomach. This fluid will provoke nausea in another patient with the same symptoms, whereas a post-gastrectomy patient without symptoms of bile vomiting appears to be unaffected if the fluid is introduced into his stomach. It seems likely that the components of this fluid responsible for producing symptoms are bile salts.

Treatment of Non-obstructive Vomiting

A complete appraisal of the patient's nutritional state must be made, with particular reference to any deficiencies of iron, vitamin B₁₂, and folic acid. Correction of deficiencies and improvement in the patient's health and feeling of well-being are sometimes all that is required to control the nausea and

vomiting. When indicated, treatment for depressive neurosis with anti-depressant drugs should be the next line of treatment. In the emotional "easy vomiter" the first line of treatment should be the anti-emetic drugs of the dimenhydrinate or prochlorperazine variety.

FIG. 2.—Vomiting caused by excess regurgitation of bile into the gastric remnant after meals (A). Treatment is by diversion of the bile away from the stomach.

Where this initial treatment is not indicated or fails cardio-oesophageal competence should be assessed by barium meal examination. Regurgitation of bile into the stomach is assessed by tube aspiration and may be confirmed by barium-potato-meal examination. Testing the provocation effect of intragastric bile or taurocholate solution may also be helpful. A small number of patients will then be found in whom gross regurgitation of bile into the gastric remnant appears to be the main cause of their symptoms. These can be effectively treated surgically by operations designed to prevent bile regurgitation, such as jejunal loop interposition or roux-en-Y conversion (Fig. 2).

(This article will be concluded next week, together with a list of references and a bibliography.)

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