DISAPPOINTMENTS AFTER GASTRO-ENTEROSTOMY.

BY

ROBERT HUTCHISON, M.D., F.R.C.P.,
PHYSICIAN TO THE LONDON HOSPITAL, ETC.

THE modern operation of gastro-enterostomy when performed for chronic ulcer of the stomach or duodenum is usually so satisfactory in its results that a surgeon is justified in promising the patient complete relief from his digestive troubles if he submits to it. All the greater, therefore, is the disappointment of both when the promised benefits do not follow and when the patient finds that he has not obtained that digestive peace to which he had looked forward. I propose to consider briefly the nature of some of these disappointments and to discuss their cause and how they may be avoided.

Persistence of Pain.

The chief symptom of ulcer is pain, and the most certain immediate result of a gastro-enterostomy is the disappearance of pain. In the great majority of cases the relief from pain is permanent, but in a few it returns after an interval of a few months or longer. The pain may be constant or it may occur in bouts and have the character of "hunger pain." Naturally the patient fears that his old ulcer has come back—although as a matter of fact this rarely if ever happens—and he goes to his doctor, who is apt to put him off with a diagnosis of "adhesions." Now it would be going too far to say that adhesions are never a cause of severe abdominal pain, but they are certainly not often so, and after gastro-enterostomy no adhesions should form. As a matter of fact severe pain coming on some time after the operation should suggest the formation of an ulcer either in the jejunum or at the site of the anastomosis.

Jejunal ulcer seems most apt to form in those cases in which the gastric hyperacidity has persisted, as it sometimes does in spite of the operation. An anastomosis ulcer, on the other hand, is usually, I believe, the result of using unabsorbable sutures when uniting the stomach and jejunum. In either case the ulcer may heal temporarily under medical treatment by rest, diet, and bismuth, but permanent cure by such means is, I should think, rare, and operation is certainly the best remedy. So far as I have seen, it is usually quite satisfactory in its results.

Apart from fresh ulceration, pain recurring after gastro-enterostomy may be due to some condition outside the stomach which has been overlooked at the time of the operation or which has arisen since. Gall stones are a case in point. Most surgeons now make a point of removing the appendix when performing gastro-enterostomy, but if for any reason this has not been done, pain of appendicular origin may arise later and necessitate further operation. I have also seen one or two cases in which stone in the kidney appeared in patients who had undergone gastro-enterostomy. It should be remembered in this connexion that such patients are rather prone to phosphatic stone formation owing to the fact that they have often taken large quantities of alkalis for the relief of pain before the operation, and have had in consequence a condition of phosphaturia.

Pain may also persist if the operation has been unnecessarily performed in a case of acute ulcer or gastralgia. I have met with one example of this in a young woman in whom, when the abdomen was reopened in order to undo the gastro-enterostomy, no sign of a healed ulcer could be discovered.

Lastly, it would seem that an anastomosis between the stomach and jejunum predisposes to pain in the colon, perhaps from too rapid filling of it, perhaps from irritation by food which has been imperfectly digested higher up. At all events, pain of greater or less severity in the lower abdomen, and sometimes attended by looseness of the bowels, is not uncommonly met with in the earlier months after the operation, although it usually disappears later. In not a few instances also I have known mucous colitis appear in patients who had not been the subject of it before the operation, and this also, by reason of the enterospasm which is so often associated with it, may be a cause of pain.

Vomiting.

As a cause of disappointment after gastro-enterostomy vomiting is less common than pain. In the early days of the operation it was all too familiar as the result of the establishment of a "vicious circle," but with the present methods of operating a "vicious circle" is almost never seen. One still meets occasionally, however, with patients who have pretty continuous vomiting, chiefly of bile. Such an occurrence points to some mechanical obstruction in the neighbourhood of the anastomosis, and, if washing out the stomach fails to relieve, surgical measures to deal with the obstacle are indicated.

Functional Disorder.

More baffling than either of the forms of disappointment already considered are those cases in which the symptoms point not to an organic (or surgical) lesion, but to a profound functional disorder of the alimentary tract. The operation may have relieved the patient of his devil of pain, but it has let in seven other devils which are almost as disagreeable tenants. The complaints of such patients are various: there is a "heavy" feeling or "distension" in the epigastrium, as if the stomach was "too big"; or again there is a sense of "emptiness," as if the food dropped "straight down"; distressing nausea, like "constant sea-sickness," is frequent; flatulence and regurgitation are also met with; and along with any of these symptoms there may be a feeling of great weakness and prostration, failure to gain weight, profound mental depression with nervous feelings and phobias of various sorts. No wonder that, when many of these symptoms concur, the patient is disappointed with the results of the operation. What is the cause of these untoward manifestations? It is not always easy to say. A test meal usually reveals diminished acidity, such as is common after gastro-enterostomy, and the *x* rays generally show that the stomach is acting well; sometimes, indeed, the stomach is emptying too rapidly, but in one or two cases within my experience it has been found that owing to dropping of the stomach the stoma is placed too high and stagnation in the lower part of the organ has resulted. This is specially apt to happen if the pylorus has been occluded at the time of the operation, and the delayed emptying, combined with a lessened acidity, is enough to account for much digestive discomfort.

One feature which is common to almost all the cases of functional disorder after gastro-enterostomy is diminished gastric tone. It is probable that this has been present in the majority even before the operation; but in some it may date from the operation itself. It is not sufficiently realized that the operation of gastro-enterostomy profoundly affects the gastric functions, both secretory and motor, and that it is not the simple drainage procedure sometimes pictured. Given an atonic stomach which also empties itself too fast, we get in an exaggerated degree that sensation of sinking and emptiness which is so characteristic a symptom of ordinary gastric atony.

The effects of too rapid emptying of the stomach are aggravated by a consequent overfilling of the intestine which is often accompanied by some ileal stasis, as revealed by the *x* rays. In two of my cases stagnation had been carried so far that part of the jejunum beyond the anastomosis had become dilated so as to form, as it were, an accessory stomach, as if to compensate for the practical abolition of the true stomach as an organ of digestion.

Glycosuria.

An interesting symptom of which I have met with three examples amongst the cases now under consideration, is glycosuria. How this is produced it is difficult to say. It has been small in amount and apparently not much influenced by diet. In at least one case it disappeared after the anastomosis had been undone.

TREATMENT.

What can be done for patients who exhibit these symptoms? Not very much, unfortunately, but considerable relief is experienced from the wearing of an efficient abdominal support with a diet consisting of small dry meals and rest in the recumbent position after food. Abdominal massage has a good effect upon the ileal stasis. Drugs are not of much help. In at least four cases the discomfort was so great that I was obliged to advise that

the anastomosis should be closed. One is naturally reluctant to recommend such a step, but so far as I have seen the results have always justified it. The constant nausea, which was a prominent symptom in all of them, was got rid of, and, although none of the patients was restored to perfect health, they were all greatly relieved and brought back to a condition of comparative efficiency.

The moral to be drawn from these disappointments is that cases for gastro-enterostomy should be selected with greater care than is always exercised, and the opinion of a physician as to the advisability of the operation should be obtained as well as that of a surgeon. Even with the greatest care mistakes in diagnosis will occur, but the surgeon should have the moral courage to close the abdomen and proceed no further if at the time of the operation no definite organic lesion of the stomach or duodenum can be demonstrated. In some of the cases I have seen there was strong reason to suspect that the operation had been performed for atonic dilatation or gastroparesis only; the results are always bad. Even when an organic lesion is present the coexistence of decided atony of the stomach is a contraindication to operation unless careful and prolonged medical treatment has failed to relieve.

The Lumleian Lectures ON CEREBRO-SPINAL FEVER.

DELIVERED BEFORE THE ROYAL COLLEGE OF
PHYSICIANS OF LONDON,

BY

SIR HUMPHRY ROLLESTON, K.C.B., M.D., F.R.C.P.,

EMERITUS PHYSICIAN, ST. GEORGE'S HOSPITAL; PRESIDENT,
ROYAL SOCIETY OF MEDICINE.

LECTURE II.

(Abstract.)

In his second lecture Sir Humphry Rolleston gave a clinical picture of the disease. The incubation period has been variously stated to be from one day up to as long an interval as thirty days, most writers inclining to a period of four or five days, and very few regarding it as long as ten days. These estimates have been based on the interval between known contact with a case or a recognized carrier and the appearance of symptoms, but a more accurate basis for deciding the incubation period would be provided by determining the average duration of the period that meningococci are present in the nasopharynx before symptoms of systemic infection appear. Fildes and Baker examined with a negative result 26 men who subsequently contracted the disease, concluding that carriers seldom develop the disease, and that the period of time between the acquisition of the meningococcus and the onset of the disease must usually be short. Out of these 26 cases, 5 were examined within a week of the onset, 8 during the second week, and 4 in the third week before the onset.

The general opinion, as voiced by von Lingelsheim, Gordon, and Andrewes, is that meningococci are constantly present in the throat at the onset of the disease, and that every case arises in a carrier. Fildes and Baker's observations, as far as they go, are certainly compatible with this view that the incubation period is very short, and need not be more than a day or two, but their failure to find any meningococci in the throats of persons who afterwards contracted the disease is exceptional, for other observers with much smaller series of cases have usually been able to point to some examples of known carriers contracting the disease; thus, among Martin Flack's 185 carriers 4 developed the disease during isolation.

ONSET.

The usual form is much like that of ordinary "influenza," and is sudden, with headache, fever, and vomiting, the symptoms becoming progressively worse, so that in twenty-four hours meningitis is suggested. Sometimes after this form of onset the symptoms rather suddenly advance, so that in a short time—an hour or two—the patient is comatose or delirious; in young children, and occasionally

in adults, the onset may be marked by convulsions. On the other hand, after the onset there may be a fallacious improvement for a day or two, as if the blood invasion were yielding to the forces of natural immunity, and then meningitic symptoms appear. The catarrhal onset lasting some days is more gradual.

The acute or fulminating onset is the least frequent; it may take various forms. It may be maniacal, imitating delirium tremens, attended by convulsions suggesting epilepsy, by hallucinations or syncope, or the patient may be unconscious when first seen. In patients found unconscious, or in the apoplectic mode of onset, the diagnosis is obviously very difficult; in some instances the patient has fallen out of bed or out of his hammock, and the question of fractured base has arisen. This apoplectiform onset may be rapidly followed by death, and thus accounts for cases of sudden death unexplained until examination after death shows the presence of the meningococci in the central nervous system.

With severe haemic infection the manifestations are those of grave toxæmia, approaching the typhoid state, often at the outset with a subnormal temperature, which subsequently rises rapidly, quick pulse and respirations, vomiting, and a haemorrhagic rash. Sudden cardiac collapse and fatal syncope at the onset of meningitis are specially described by Sainton. The initial shock may be considerable, and when accompanied by vomiting, abdominal pain, and diarrhoea, may suggest an acute abdominal lesion such as appendicitis or perforation, and laparotomy has naturally been performed. One such case in the navy was operated upon for a supposed perforated gastric ulcer and found to have a normal abdomen; at the suggestion of the anaesthetist, lumbar puncture was at once performed, and turbid fluid containing meningococci obtained.

CLINICAL FORMS.

Cerebro-spinal fever, like acute poliomyelitis, has two distinct phases: (a) The general or systemic infection; and (b) the localization in the meninges. The evidence of systemic infection is usually prominent in the initial stages, and the process is then commonly extremely acute, but it may continue for weeks, and in rather rare instances chronic septicaemia follows meningitis.

A most important factor influencing the course and type of the disease is the serum treatment. Absence of or inefficient serum treatment is responsible for fatal results either early in the disease or after a chronic illness due to adhesions or chronic septicaemia. The different types of cerebro-spinal fever show transitional forms, and description of the forms is apt to be artificial and confusing, as a single case may appear to belong to two. Further, the clinical picture of the disease is often abruptly changed by unexpected alterations, such as improvement or the onset of grave symptoms. The following forms of cerebro-spinal fever may be briefly mentioned without a detailed description, and to avoid repetition the symptoms will be mainly dealt with subsequently: (1) Fulminating; (2) ordinary acute; (3) abortive; (4) chronic: (a) septicaemic; (b) encysted or loculated meningitis.

The posterior basic meningitis of infants is chronic encysted meningococcal meningitis in young infants, and its special features appear to depend on the early age of the patients rather than on any peculiarity in the infecting organism. More than twenty years ago Still showed that the disease was due to a micro organism almost identical with the meningococcus, and of late years it has become recognized that there is no constant bacteriological distinction between the meningococcus of posterior basic meningitis and that of cerebro-spinal fever in later life. But Andrewes states that serologically the meningococci may be of a different strain from those of the epidemic disease in adults, and M. H. Gordon states that the meningococci in posterior basic meningitis do not differ serologically from those in adult cases, except that some, which are more difficult to identify, are usually atypical specimens of his Group IV. Among the cases called posterior basic meningitis half occur under six months of age, three-quarters within the first year, and practically all under the age of two years, but there is no essential difference between the condition of closed meningococcal infection of the cerebral ventricles in infants and in adults. It may be well to mention the main clinical features differentiating the clinical form called posterior basic