JOINT MEETING No. 3

Section of Medicine with Section of Proctology

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DISCUSSION: THE DIAGNOSIS AND INVESTIGATION OF CHRONIC DIARRHŒA (EXCLUDING TROPICAL DIARRHŒA)

Dr. W. Trevor Cooke: Diarrhœa is the passage of an unformed stool and may vary considerably in volume, in some cases up to a litre being passed at one time. It is characterized by an increased water content, increased sodium content and sometimes by an increased content of potassium. The normal stool is passed once or twice a day, is formed and contains 100–200 ml. of water, 2–5 mEq. of sodium and 10–20 mEq. of potassium together with 20–30 grams of solids. With increase of diarrhœa, sodium appears to bear a direct relationship to the water content, as much as 200 mEq. being excreted with 2 litres of fluid per day, though potassium does not bear any close relationship and is not excreted in such large quantities as sodium. The adjective "chronic" may be considered to apply to those diarrhœas not acute and either continual or recurrent. It must be recalled and emphasized, however, that what the doctor might class as chronic diarrhœa is often accepted by the patient as being a normal part of his daily habits and only specific questioning will reveal the presence of chronic diarrhœa and so lead to the elucidation of its cause for the proper treatment of the patient.

The causes of chronic diarrhœa have been classified according to whether they are due to disorders of the stomach, small intestine or colon, or of systemic cause. While this classification is helpful in an empirical way, it offers no intellectual satisfaction as to why these causes should give rise to diarrhœa. It is self-evident that whether or not the given patient has diarrhœa will depend upon the function of the colon alone, and that even some of the most severe disturbances of small intestinal and gastric function may be associated with normal stool consistency. If the discharges from a normal ileum following ileostomy are any guide to the normal daily discharge of ileal contents into the colon, then large volumes of water up to 2 litres or more a day containing sodium in at least isotonic concentration and sometimes being as high as 800 mEq. in the 24 hours are presented to the colon for re-absorption each day (Brooke, 1956). It has generally been accepted that this remarkably efficient re-absorption of sodium and water takes place principally in the ascending colon. There are, however, many clinical observations which suggest that the whole of the colon takes part in this active re-absorption and it is probable that every part of the colon can accommodate to active water and electrolyte re-absorption when necessary. Since sodium and water bear some constant and close relationship to each other, it does not need much upset in the re-absorptive function of the colon to produce a diarrhœic stool.

Causes for diarrhœa may, therefore, be sought in the conditions that bring about in particular,

(1) alterations in the fluids presented to the colon making them either hypertonic or otherwise irritant to the mucosa,

(2) destructive processes in the wall of the colon itself,

(3) alterations in the neuro-muscular and vasomotor tone of the colon brought about by (*a*) constitutional factors, e.g. psychomotor or thyrotoxic, (*b*) local factors, amongst which lesions such as polypi, external adhesions affecting particularly the sigmoid flexure and external pressure of such disorders as uterine and ovarian tumours, may be cited.

I shall deal particularly with the group which results in alterations in the normal ileal contents subsequently causing the colon to produce a diarrhœic stool. I am not proposing to consider the clinical history of a patient with chronic diarrhœa in detail even although it may be diagnostic in some instances.

Methods of Investigation

(1) Inspection of the stool: Inspection of the stool is probably one of the most neglected procedures in clinical medicine yet often it will give clear evidence where the source of the trouble is to be found e.g. in the small intestine, with the bulky pale stools of some patients with steatorrhœa, presence of undigested food. Stools with an aluminium sheen are seen when steatorrhœa is due to causes other than carcinoma of the ampulla of Vater and have no specific diagnostic connotation.

(2) Microscopical examination: This should be practised routinely. The presence of undigested meat fibres, fat globules, fatty acid crystals, pus, red cells, *Giardia lamblia* or

other intestinal protozoa should be looked for. Increased concentration of fatty acid crystals is common in the presence of steatorrhœa and with the history of chronic diarrhœa indicates the need for further chemical analysis of the fæces. However, neither the presence of excess fatty acids nor their absence is any guide as to the presence or absence of steatorrhœa. Muscle fibres can be seen in virtually any chronic diarrhœa but the persistence of fibres when the stools have been standing twenty-four hours suggests pancreatic deficiency. Stress has been laid on the presence of starch granules, but I myself have not gained much information of clinical value from them.

Chemical Analysis of the Fæces

Analysis of the fæces for fat content has now become essential for accurate diagnosis and the presence of an increased fat content is diagnostic of small intestinal dysfunction but has no further ætiological significance. Determination of the percentage fat (dried weight) in a portion of daily fæces has now largely been abandoned, although percentages over 30 are only rarely to be found in patients who do not have steatorrhœa. Differential analysis of the amount of split and unsplit fat in the fæces is rarely used for it is no longer regarded as valid evidence as to the presence or absence of pancreatic dysfunction. In fact, the only use that I can see for the test is to provide a check on ward routine so that false answers are not obtained through the unwitting administration of liquid paraffin.

Many hospitals have been deterred from fæcal fat analyses on the grounds that careful balance techniques are necessary and not so long ago the administration of a diet containing a known amount of fat usually 50 or 70 grams was considered essential and the resultant daily fat excretion could then be expressed in terms of the amount of daily fat absorption. It has, however, come to be recognized that such expressions give a false impression of scientific accuracy. Further a normal individual on diets containing fat varying between 50 and 120 grams of fat a day rarely excretes more than 6 grams in any one day, and in my experience, the mean varies only slightly on increasing loads of fat rising from 2·8 to 3·5 grams per day. It follows from this that for diagnostic purposes it is permissible to analyse daily fæcal fat excretion in a patient on a normal varied diet and that an average daily excretion for 3–5 days of over 6 grams per day may be taken to indicate small intestinal dysfunction or steatorrhœa. The use of an electric mixer, a good refrigerator and simpler methods of analysis make it possible for even the smallest laboratory to carry out fat estimations with adequate accuracy for the investigation of any case of chronic diarrhœa.

Nitrogen

For diagnostic purposes, there is little point in estimating the daily fæcal nitrogen excretion. Increased nitrogen content of over 2 grams per day has often been taken to indicate pancreatitis. It has, however, been pointed out by a number of workers that almost any diarrhœa will lead to an increased fæcal nitrogen. As far as the diarrhœa associated with steatorrhœa is concerned, the more fat the more nitrogen whether the steatorrhœa be of idiopathic or pancreatic origin.

In the investigation of chronic diarrhœa, a careful radiological examination is important, particularly in those patients in whom the small-intestine lesions have brought about the chronic diarrhœa. It is time-consuming and entails frequent films and much screening but unless some indication can be given to the radiologist as to what exactly is being looked for, such examinations may lead only to waste of the radiologist's and patient's time, to say little of the expense of films involved. For routine purposes, I have been accustomed to ask for a poorly flocculating barium contrast medium, such as Raybar, since areas of dilatation or inflammation as in jejuno-ileitis are more readily delineated, while should flocculating occur then that in itself is good evidence of dysfunction of the small intestine. However, if barium sulphate and water suspensions are used, then flocculation must be accepted as indicating excess mucus in the intestinal lumen and usually indicates the presence of steatorrhœa. It may not always do so, since, for example, some 7% of patients with pernicious anæmia present with chronic diarrhœa and occasionally some have extensive flocculation but no steatorrhœa. Any patient who has a scar on his abdomen and suffers with chronic diarrhœa deserves careful radiological examination of the intestinal tract, so that the normal continuity of the gut can be established and the existence of any blind loop excluded.

The importance of hæmatological investigation must not be overlooked for many disorders of the small intestine are associated with macrocytic anæmia, as has been discussed at a recent meeting of the Society (Witts, 1955).

As aids to diagnosis the use of both glucose tolerance tests and chylomicrographs is disappointing owing to the wide range of results obtained in any particular disorder. Culture examination of the fæces can also provide further evidence of the ætiology of certain types of chronic diarrhœa. The role of the pathogenic staphylococcus is, however, by no means clear, whilst the *Giardia lamblia* is usually rejected as a significant factor even though patients so infected may have their diarrhœa cleared by the administration of mepacrine. The

role of fungi and yeasts still remains to be clarified and little has been reported in this country on the possible association of histoplasmosis and enterocolitis.

In conclusion, the prerogative of an opening speaker allows a few dogmatic comments on some of the traditional causes of diarrhœa. The diagnosis of lienteric diarrhœa or diarrhœa associated with achlorhydria should now be discarded. There is no satisfactory evidence that achlorhydria is prone to give diarrhœa or to cause rapid gastric emptying. Many of the patients to whom this diagnosis is attached will prove to have steatorrhœa and in others the basis will be psychological. Gastro-intestinal hurry is much favoured as a cause for diarrhœa. The evidence, however, on which this is based is poor. The difficulties in measuring the transit time through the small intestine are great, for visualization of the rate of passage of barium does not guarantee that food travels at the same rate. The diarrhœas following gastrectomy that I have studied showed the rate of barium transit through the small intestine to be longer rather than shorter. The third diagnosis which may be queried is that of tabes mesenterica or mesenteric lymphadenopathy; in my experience neither of these two conditions gives rise to chronic diarrhœa unless the intestinal wall itself is involved in the disease processes.

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Mr. W. M. Capper:

Diarrhœa of gastric origin.—Gastrogenous diarrhœa in general is usually distinguished by the fact that it occurs very soon after a meal and often in the early morning hours. It tends to affect patients in the mid-life or beyond, and evacuations rarely exceed six in a day: indeed, they are usually less. Stools are soft and mushy, not watery; there is no blood, pus, or mucus, and the patients may say that they cover the pan. Motions are usually unaccompanied by pain and may be intermittent in character, i.e. they are frequent for two or three days and then are better perhaps for five or more days, when the diarrhœa will again recur. Attacks may be associated with the intake of specific food such as hot sweet nutrient liquids, or possibly by fried or highly spiced foods. The stools may contain recognizable food particles from the last meal. Gastrogenous diarrhœa may be divided as to causation into two main groups: those cases which follow surgery on the stomach, and those that occur entirely apart from any surgery at all.

(1) *Diarrhœa following Gastric Surgery*

(*a*) *Following total gastrectomy.*—Welbourn (1956) found diarrhœa present in 3 out of 9 patients surviving one year or more. Re Mine and Priestley (1952) followed up 13 patients who had survived five or more years, and found that diarrhœa was "infrequent". Of my own cases, it has not been troublesome in more than 1 out of 6 surviving longer than two years. It has the classical features of gastrogenous diarrhœa and barium meal usually shows intestinal hurry.

(*b*) *Following partial gastrectomy.*—This occurs in 3 main groups—(i) Diarrhœa may occur on the third to fifth day after operation when solid food first starts to be taken. It is noticeably worse in cases when operation has been carried out for pyloric stenosis, especially if adequate pre-operative lavage has not been performed. It usually consists of anything up to ten stools a day and must be closely observed in case the features of a severe enterocolitis supervene. It has been stated that it will respond to hydrochloric acid by mouth or to antibiotics, but I have not found either of these measures to be effective. It usually subsides without special treatment beyond frequent small doses of I-so-gel.

(ii) Severe and sometimes fatal enterocolitis may appear about the second post-operative day. In 35 cases out of 1,700 partial gastrectomies reported by Dawson-Edwards and Morrissey (1955) 5 were fatal. Profound shock and incessant diarrhœa are the main diagnostic features and resuscitative treatment must be instituted immediately. This condition has assumed importance recently since it has been realized it is an important factor in any series of deaths following partial gastrectomy.

(iii) In a few cases, a low-grade diarrhœa starts after partial gastrectomy and persists. Frank steatorrhœa with bulky and offensive stools is rare, but many patients have an excess of fat in their fæces. Welbourn (1953) investigated 248 cases of partial gastrectomy, most of which were a 70% Polya, and found that 110 (i.e. 44%) noticed that their bowels were more regular, and 9 of these (4%) developed diarrhœa for the first time. B. N. Brooke (1954) found 9 out of 167 cases (i.e. 5%) followed a Polya operation. He found no cases out of 48 who had a Billroth I operation. Bohmansson (1926) found that there were intestinal disturbances in 24% of cases after the Polya operation, and 6% after the Billroth operation. Of my own cases, 3% of Polya cases complained of loose stools which tend to follow meals, and are sometimes explosive in character. They may, or may not, be associ-

ated with the dumping syndrome or biliary vomiting. Glazebrook and Welbourn (1952) found that barium meal in these cases shows unusually rapid intestinal passage and kymography reveals excessive peristaltic activity. Very rarely X-rays may actually show a reduction of activity with clumping of the barium, although there is complaint of diarrhœa (Glazebrook, 1952; MacPhee, 1953). It seems definite that symptoms are commoner following a Polya anastomosis and there is usually increased fat in the stools. The probable cause is excessive bowel mobility following the entry of food into the jejunum. The symptoms can usually be relieved by the exhibition of ganglion-blocking drugs such as propantheline bromide and hexamethonium.

(*c*) *Following gastro-enterostomy.*—Diarrhœa may follow this operation exactly as in partial gastrectomy, but is unusual.

(*d*) *Following vagotomy.*—This operation when it is done as a solitary procedure gives rise to a combination of achlorhydria and pylorospasm. The infected pent-up gastric contents pass into the jejunum from time to time. They are highly irritating and give rise to diarrhœa which may be quite severe in character. The condition usually ceases when the stomach is drained by a pyloroplasty or gastrojejunostomy.

(*e*) *Gastro-jejuno-colic fistula.*—This must always be borne in mind as a possible cause of diarrhœa in any patient who has had a gastro-enterostomy. Indeed, in such a case it is usually right to assume that such a fistula is present until there is absolute proof that it is not so. Classically, the story is that after an interval of months or years, following a gastrojejunostomy, there is a brief period when typical symptoms of anastomotic ulcer are present. The patient then says that the persistent indigestion has suddenly ceased, and is replaced by incessant diarrhœa and belching of foul gas. Clinically this is followed by progressive cachexia, hypoproteinæmia, and a deficiency state with steady decline. The stools are sprue-like and there may be fæcal vomiting. In certain cases, depending on the size of the fistula, there may be no symptoms relating to the bowel. Lowdon (1953) found diarrhœa as the main symptom in 41 out of 46 cases. The severity varied from 4 to 20 or 30 stools a day. In some cases there was incontinence of a watery fluid. Fæcal vomiting occurred in 25, and wasting in 36, out of the 46 cases. It may be difficult to prove the diagnosis. In a barium meal the medium often does not pass into the bowel via the fistula. A barium enema revealed the lesion in 31 out of 32 cases (Lowdon, 1953). If the fistula is small, an enema of aqueous methyl carmine with an in-dwelling stomach tube may show dye in the gastric washings. It is also worth while to have an in-dwelling stomach tube when the barium enema is given, to see whether there is any barium in the washings. Uncertainty in the diagnosis must not be allowed to delay laparotomy, as these patients may decline rapidly and their condition become precarious. Adequate and efficient surgery becomes then a life-saving measure.

(2) *Gastrogenous Diarrhœa not associated with Surgery*

Various writers have described diarrhœa as secondary to achlorhydria but it certainly is not common. In my experience a patient with alcoholic gastritis usually has one or two loose stools in the morning and then is not generally bothered for the rest of the day. Bockus (1946) says that diarrhœa occurs in 10% of cases of achlorhydria. Amongst the causes, he quotes pernicious anæmia, hypochromic microcytic anæmia in females and carcinoma of the stomach. The attacks in susceptible patients are often precipitated by eating large amounts of roughage or indulgence in alcohol. One of my colleagues, Mr. Gordon Paul, has recently had a case of pyloric cancer, where the main symptoms were loss of weight and diarrhœa usually occurring just after food. At operation the pylorus was rigid and stenosed but the passage of infected food material through the pylorus was possible, thus causing jejunal irritation and the postprandial diarrhœa.

It must be emphasized that gastrogenous diarrhœa does not present undue difficulty in diagnosis. The history as given by the patient is usually directed towards the gastric condition, with diarrhœa as a secondary complaint. In cases following gastric surgery, of course, the patient usually volunteers the information that the diarrhœa started after operation and had not occurred before.

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