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STEATORRHOEA*

BY

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Steatorrhoea is a descriptive clinical term. It indicates the passage of fluid or semi-fluid, bulky, pale, fatty, and usually offensive stools. It is incorrect to refer to the passage of formed stools as steatorrhoea merely because the stool, on analysis, is found to contain more than the normal amount of fat; indeed, the misuse of this term has led to some confusion. Steatorrhoea is symptomatic of a number of different pathological conditions, and it cannot be regarded as a constant finding in any of them.

Characteristics of the Steatorrhoeic Stool

Volume and Consistency

The normal daily specimen of faeces on our standard diet has a volume of 100–200 ml. In steatorrhoea the volume of the stool may increase to 500–1,000 ml. or more. There is a concomitant increase of dry weight. There are several reasons for this increase in stool volume. In the first place, more fluid may be passed from the ileum, since it is known that water absorption may be delayed or defective in many of these cases; secondly, the time of transit through the colon is often lessened, so that the amount of water absorbed by the large intestine may be reduced. The presence of electrolytes and other absorbable molecules should not interfere seriously with water absorption in the colon, since this organ normally handles such materials. The long-chain fatty acids *per se* do not have any marked effect on the fluid volume of the stool—plenty of fatty acids may be present in formed stools of normal volume. It is possible that some irritant action arises from the presence of excessive amounts of short-chain fatty acids, but no obvious quantitative correlation has been found between these acids in the stools and volume. Tenesmus is not commonly associated with steatorrhoea. It may be tentatively concluded that the increased volume and the fluid consistency of the stools are likely to be due to some increase in the volume of the material passed into the colon from the ileum, coupled with a relatively early emptying of the large bowel. Some slight irritant action may play a part.

Colour of the Stool

The stool is characteristically pale or putty-coloured. Studies of the pigment content indicate that the bile pigment content of the stool may be within normal limits. Strong reduction of bile pigments produces a similar pale putty colour, and the presence of a strong reducing flora due to carbohydrate fermentation is a common feature in steatorrhoea. Administration of antibacterial agents commonly causes darkening of the stools (Anderson *et al.*, 1954). The best explanation at present available, therefore, for the pallor of the steatorrhoeic stool is that it is due to changes in the bile pigments associated with a fermentative intestinal flora.

Types of Fat in the Faeces

The fat in the stools in steatorrhoea may consist of short-chain volatile fatty acids or long-chain fatty acids, soaps, or glycerides, which may be either solid or liquid at room temperature.

Short-chain Fatty Acids.—These are esssentially derived from carbohydrate fermentation. They can be readily removed by steam distillation. They do not contribute to the fatty appearance of the stool, but they may be partly responsible for the offensive odour.

Long-chain Fats.—These are most commonly present as long-chain fatty acids. They may be seen as sheaves of crystals in faecal smears. In certain types of defect and in most severe cases the fat may appear as liquid fat globules, round which clusters of crystals may be collected. In moderately severe cases most of the fatty acids belong to the saturated series, the predominating fatty acids being palmitic and stearic. In more severe cases the quantity of unsaturated fatty acids increases (Weyers and van de Kamer, 1950). Under certain circumstances the fat is present as glycerides instead of fatty acids. This usually indicates faulty pancreatic lipolysis, but extensive hydrolysis of faecal fat may occur in the absence of pancreatic lipase (Cooke *et al.*, 1946).

Quantity of Fat in the Faeces

Short-chain Fatty Acids.—The daily output is normally less than 1 g.; for accurate assessment the stools should be collected into a cold container and refrigerated during storage.

Long-chain Fatty Acids.—These should be assessed on a standard daily dietary fat intake (50–100 g.). We use a method based on that of van de Kamer *et al.* (1949). The total fatty acid is estimated daily and the faecal fat output calculated as a three-day running mean. Normal daily faecal fat output on our standard diet does not exceed 5 g. when averaged over 10 days. The normal level should be estimated for each laboratory for the standard diet used. Typical results under the conditions prevailing in our unit are illustrated in Fig. 1. For comparative purposes it would seem advisable to take some conventional time period for calculation of

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the mean value, so a ten-day period has been used. The daily variation is best expressed as a standard deviation calculated along classical lines.

Origin of Faecal Fat

It is generally thought that most of the fat in the normal faeces is of non-dietary origin. Wollaeger *et al.* (1947) have shown that there is a small increment of faecal fat with increasing intake. When the fat in the faeces is increased above the accepted normal level the increment may be due to either dietary or non-dietary fat, or both, as illustrated in Fig. 2. The assessment of the proportion of dietary or non-dietary fat concerned in the increase can be made by using suitably labelled dietary fat, by altering the level of fat in the diet, or by modifying intestinal flora. Unfortunately, the labelled fats available for use in human subjects are far from ideal: with halogens or deuterium the introduction of the label involves a change in properties of the fat, and the stability of certain labels has been questioned; with stable ^{13}C labelled materials, spectrographic methods are required that are not widely available; radioactive ^{14}C labelled fats are not suitable for use in man. However, the effective use of a labelled fat should provide definite evidence of any increase of the dietary residue. Alteration in fat load, especially the reduction of fat intake to minimal levels, should also indicate whether the faecal fat is mainly derived from the dietary fat. There is no doubt that in many cases of steatorrhoea unabsorbed dietary fat contributes significantly to the increase of faecal fat. It is also true, however, that cases have been reported in which the increased faecal fat appears to be entirely derived from non-dietary sources (Weijers and van de Kamer, 1953). It is difficult, at the present time, to define with any precision the relative proportions of dietary and non-dietary fat that go to form faecal fat in any individual case of steatorrhoea; it is likely that this proportion may vary from case to case, and, indeed, it may also vary from time to time in the same case.

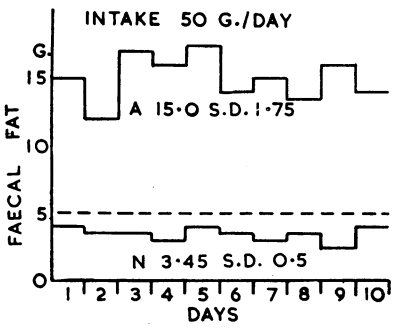

FIG. 1.—Faecal fat content. N=normal levels. A=abnormal levels. Figures are the mean for a ten-day period and the standard deviation.

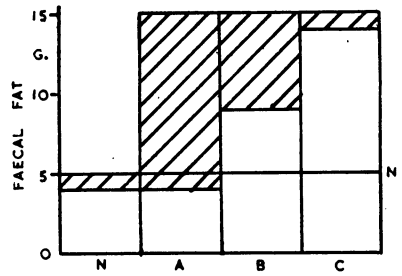

FIG. 2.—Diagram to illustrate possible proportion of dietary and non-dietary components in faecal fat. Hatched area=dietary fat. Clear area=non-dietary fat. N=normal proportions. A, B, and C show three main abnormal types.

For these reasons I would suggest that it is unwise to convert faecal fat figures into any coefficient of fat absorption. Until we are in a position to determine the dietary component independently of the non-dietary, the best way to express the results of faecal-fat analysis is on the factual basis of grammes of faecal fat per diem.

Further consideration must be given to the question of the origin of the non-dietary faecal fat component. It might be derived from excreted fat or from synthesized fat. The excreted fat might pass into the intestinal lumen in the bile; if this is so, an absorptive fault must also be present if this biliary fat passes through the small intestine without being reabsorbed. The fat could also be excreted through the lower part of the intestine. There is little evidence that fat can normally be excreted from the intestine in any significant quantity: whether it might occur under certain abnormal conditions remains to be demonstrated. Synthesis of fat can occur in the intestinal cells, in bacteria, and in other intestinal organisms. These fatty acids can be synthesized from 2-carbon fragments. Under normal circumstances there is no evidence of synthesis of long-chain fats in intestinal cells on a scale that could account for the faecal fat increments observed in steatorrhoea; whether this might occur under conditions of extensive carbohydrate fermentation, or in other abnormal situations, remains to be shown. There seems little doubt that intestinal flora contribute to normal faecal fat and may well make a larger contribution in the steatorrhoeic stool. It has been doubted whether intestinal flora could provide all the non-dietary fat observed in the stools in some cases: this question cannot be answered at the present time. In the absence of any hyperlipaemia, synthesis of fat in the intestinal cells or by intestinal flora offers the most likely explanation for the source of the non-dietary faecal fat increment.

Aetiology

General Relationship of Steatorrhoea to Faulty Intestinal Absorption

The characteristics of the steatorrhoeic stool could be explained by the presence of large dietary residues in the material entering the colon, which are passed out in the faeces or give rise to increased bacterial growth, with further consequent effects. If small-intestinal absorption is normal, practically no assimilable dietary materials pass into the large bowel or into the faeces, and bacterial growth in the colon tends to be restricted by the adverse nutritional conditions. Increased non-pathogenic bacterial activity in the intestinal lumen is thus largely dependent upon some inadequacy of intestinal absorption. Steatorrhoea is likely to be closely associated with faulty absorption with regard to increase of both dietary and non-dietary components in the faeces. The only other situation that might result in similar changes is excretion of food materials into the lower part of the bowel, for which there is no satisfactory evidence at the present time.

Classification of Absorptive Defects

For the purposes of this discussion, defects of absorption may be conveniently classified into pancreatogenous, hepatogenous, and enterogenous groups.

Pancreatogenous Group

Pancreatic juice contains enzymes, water, and alkali, all of which play an important part in the preparative phase of absorption (Frazer, 1952a). The pancreatogenous type of absorptive defect is most commonly associated with deficiency of one or more pancreatic enzymes, although inadequacy of pancreatic digestion may arise from other causes. Thus, incoordination of the entry of pancreatic juice and chyme into the intestinal lumen may account for the absorptive defect sometimes observed after gastro-enterostomy (Brain, 1953). Pancreatic enzyme deficiency may result from pathological change in the pancreas, such as fibrocystic disease, chronic pancreatitis, and pancreatic lithiasis. It may also be due to more generalized disorders, such as hypoproteinaemia due to malnutrition (Thompson and Trowell, 1952). Generalized pancreatic enzyme deficiency is the commonest fault associated with steatorrhoea, but deficiency of a specific enzyme can also occur. Marked depression of pancreatic enzymes results in delayed absorption of carbohydrates, proteins, and fats, which may pass further down the intestine than normal. Some of these dietary materials may be passed in the stools; the improved supply of food materials results in increased bacterial growth and fermentation. Steatorrhoea therefore results.

HEPATOGENOUS GROUP

The liver elaborates bile, which plays an important part in the preparative phase of absorption and other aspects of gastro-intestinal function. The full significance of the bile is not yet understood, but bile salts are considered to be the most important constituent from the absorptive point of view. Bile may be excluded from the intestine by obstructive lesions of the biliary tract. Under these circumstances there is considerable interference with absorption. The stools are loaded with fat, but they are usually well formed and may be of a putty-like appearance and consistency. We have recently had the opportunity of studying a child who was diagnosed as suffering from the coeliac syndrome. On further examination it was found that this child had a marked deficiency of bile salts: this defect appeared to have been present from birth (Ross *et al.*, 1955). The hepatogenous group of absorptive defects are only occasionally associated with steatorrhoea.

ENTEROGENOUS GROUP

When the various food materials are prepared for absorption they enter the small-intestinal cells and are passed through into the portal blood or the chyle. A number of factors are concerned in this transport of absorbed food materials through the intestinal wall. If the preparative phase is normal, interference with absorption may be due to a number of causes—access to the intestinal cells may be prevented, some fault may be present in the intestinal cell itself, intestinal motility may be defective, or the pathways leading from the intestine may be blocked. It is not at present possible to distinguish with certainty between these various possibilities, several of which may coexist. We may, however, split the enterogenous group into two parts—according to the association with, or absence of, gross irreversible pathological changes in the intestine.

(A) *Conditions Associated with Gross Irreversible Pathological Changes in the Intestinal Wall.*—In this group we may include regional ileitis, Whipple's syndrome, tuberculous enteritis, scleroderma, and atrophy of the small-intestinal wall in long-standing cases of malnutrition from any cause. Any of these conditions—most of which are rare—may be associated with steatorrhoea.

(B) *Conditions not Associated with Gross Irreversible Pathological Changes in the Intestinal Wall.*—The cases with enterogenous steatorrhoea that fall into this group have certain common features. All these patients exhibit depressed absorptive capacity of the upper small intestine, as shown by delay and depression of the absorption curves for glucose, xylose, fat, and many other substances. Radiographic examination of the upper intestine reveals increased mucus secretion, which causes clumping of a simple barium sulphate suspension; if a non-flocculable opaque medium is used the upper intestine is found to have an abnormal mucosal pattern, and it is often dilated (Frazer, 1952b).

1. *Gluten-induced Enteropathy.*—The coeliac syndrome has been conclusively shown in Holland and England to be due to the presence of wheat gluten in the child's diet (Dicke, 1950; Anderson *et al.*, 1952; Dicke *et al.*, 1953). In a series of 30 children diagnosed clinically as suffering from the coeliac syndrome, all of whom had steatorrhoea, 28 responded to a gluten-free dietary regime. On this treatment these children passed normal stools containing a normal level of fat (Fig. 3); they also showed significant improvement in weight-for-age ratio, in absorptive capacity, and in the radiographic appearance of the small intestine. The reintroduction of gluten into the diet caused deterioration: one of the children tested had been on a wheat-gluten-free diet for four years. Of the two children who did not respond to the gluten-free diet, one was the child with congenital deficiency of bile salts referred to above; the other has not yet been fully characterized (Ross *et al.*, 1955). The precise cause of the response to gluten in coeliac children is still under investigation. Gluten-induced enteropathy is also found in adult patients (Anderson *et al.*, 1954).

2. *Tropical Sprue.*—The gastro-intestinal situation in this condition is similar to that observed in gluten-induced enteropathy. The main differences between the two conditions are that tropical sprue has a peculiar geographical distribution, it occurs in adults rather than children, it is unrelated to dietary gluten, and it is closely associated with folic acid deficiency. The main areas in which sprue is endemic are the Far East, India, and the Caribbean; it is rare in Africa. Even in the endemic areas, however, there are striking local differences: sprue occurs in Hong Kong, but not in Singapore; in Puerto Rico, but not in Jamaica. There is increasing evidence that some dietary factor may be involved and that this may be related to oxidative rancidity of unsaturated fatty acids. This question is being intensively studied by us at the present time. Folic acid deficiency may play an important part in the development of the syndrome: for a number of reasons, however, it seems unlikely that it is a simple dietary deficiency. Some of the patients with tropical sprue of the type we have been studying develop a more chronic form of the syndrome in which folic acid deficiency is no longer a dominant feature. The steatorrhoea in these patients persists even after their removal to a temperate climate. It is possible that some change in the intestinal flora is responsible for the maintenance of the syndrome in these patients. This is supported by the fact that they dramatically recover when treated with antibacterial therapy (Anderson *et al.*, 1954) (see Fig. 4). It is possible that this form of the syndrome provides the link with classical chronic sprue.

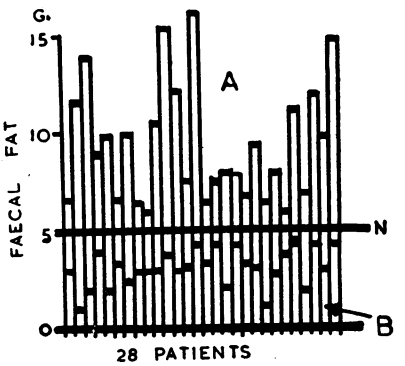

FIG. 3.—Mean levels of faecal fat in 28 consecutive cases of coeliac disease. A=on normal diet. B=on wheat-gluten-free diet. N=normal limit of faecal fat.

3. *Non-tropical Sprue.* — Adult patients diagnosed as suffering from non-tropical sprue usually include a significant number of cases of gluten-induced enteropathy. If these are excluded there remains a small group of patients with steatorrhoea, the aetiology of which remains obscure.

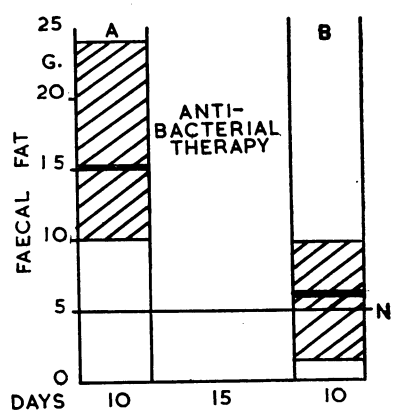

FIG. 4.—Effect of antibacterial therapy on faecal fat level in seven cases of "chronic" tropical sprue (see text). A=before therapy. B=after therapy. The heavy line shows mean value; the shaded area indicates range of values observed.

4. *Anastomotic Syndrome.* — The possible effect of gastro-enterostomy on the co-ordinated digestion of food in the upper intestinal lumen has already been mentioned. There is, however, another type of surgical case in which steatorrhoea may be the outstanding feature. Gastrocolic or enterocolic fistula and various types of intestinal loop may be associated with steatorrhoea. The precise cause of this is not yet clear, but it is possible that it may be related in some way to the intestinal flora.

Differential Diagnosis

Steatorrhoea may be associated with a number of conditions. Differential diagnosis is important, since different types of treatment are required. The first step is to separate the pancreatogenous, hepatogenous, and enterogenous groups. This is best done by intestinal intubation, as indicated in the Table printed overleaf.

When the pancreatogenous and hepatogenous conditions have been excluded the enterogenous group requires fractionation. Such conditions as Whipple's syndrome, scleroderma, and tuberculous enteritis are rare, and the diagnosis may be reached as a result of associated changes. Regional ileitis can be distinguished from the sprue group by the fact

Investigation of Defects of Intestinal Absorption

Investigation	Expected Results		Enterogenous Group
	Pancreatogenous Group	Hepatogenous Group	
Balance studies:			
Faecal fat	Markedly increased*	Markedly increased*	Increased*
„ nitrogen	Increased	—	May be increased
Short-chain fatty acids	May be increased	—	„ „ „
Intubation studies:			
Pancreatic enzymes	Deficient*	Normal range	Normal range*
Bile salts	Normal range	Deficient*	„ „
Absorption curves:			
Glucose	Normal	Normal	Depressed
Chylomicrograph	Flat	Flat	Flattened
Radiography:			
Flocculable media:			
Clumping	Absent	Absent	Present
Mucosal pattern	Feathery	Feathery	Not visible
Non-flocculable media:			
Pattern	„	„	Ladder or coin type
Diameter	Normal	Normal	Dilated

Note.—The results recorded in this Table are the expected findings; some variations will be found, however, in individual patients. The observations marked with an asterisk are the key points in diagnosis.

that the upper intestinal absorptive capacity is usually normal in this condition, and radiographic studies show abnormalities in the lower part of the small intestine. The anastomotic syndromes usually have a surgical history—the fistulous opening may be extremely difficult to demonstrate.

With regard to the three classical varieties of the sprue syndrome, gluten-induced enteropathy is indicated by marked improvement on a gluten-free regime, but it is proved only by demonstrable deterioration when wheat gluten is reintroduced into the diet. In adults it may be necessary to persevere with a gluten-free diet for at least six months before accepting a negative response. It is to be hoped that a quicker method of diagnosis of this condition will be made available soon. The rapid development of the sprue syndrome in a previously healthy adult in an endemic sprue area usually leads to the diagnosis of tropical sprue. Again, a more specific diagnostic test would be advantageous. The haematological changes associated with folic acid deficiency may not appear for some time—indeed, they may not occur at all. Those cases of non-tropical sprue that do not respond to a gluten-free diet must be classified as enterogenous steatorrhoea of unknown aetiology: "non-tropical sprue" would seem to be as good a term as any for this small residual group.

Treatment

There are two main lines of treatment: (1) to cut down intestinal bacterial activity in general and carbohydrate fermentation in particular, and (2) to correct the intestinal absorptive fault, if possible.

General Dietary Regime.—The first of these objectives has formed the basis of treatment for many years. The diet that is usually recommended has a low residue, high protein, and restricted carbohydrate. Some authorities have also advised cutting down fats. There has been fairly general agreement in recent years, however, that a fat intake of 50 g. a day, or even more, is quite well tolerated by most of these patients. The diets recommended by Fairley (1930) for the treatment of tropical sprue form a good basis for the general treatment of the whole group. The proportions of protein/fat/carbohydrate are 1/0.3/1; as improvement occurs, usually after three to four weeks on the high-protein diet, these proportions are stepped up to 1/0.36/2.

Specific Measures

Certain specific measures are effective in steatorrhoea, and they are directed against the main aetiological factors in each case.

Pancreatic Enzyme Deficiency.—In patients with defective pancreatic enzymes some improvement is generally achieved by the administration of pancreatin. The oral administration of pancreatin differs in many respects from the co-ordinated flow of pancreatic juice into the duodenum, so that it is hardly surprising that completely effective replacement is not achieved. One of the best ways of administering pancreatin, especially in children, is to mix the powder in milk, taking care that the milk is not so hot that it damages the enzymes (Ross, 1955). Emulsifying agents may sometimes be helpful in connexion with the absorption of fats and fat-soluble vitamins.

Bile Salt Deficiency.—In the child with deficient bile salts referred to above, the oral administration of bile salts had a dramatic effect on the fat in the stools (Fig. 5). However, on a long-term basis it may be simpler to restrict the fat intake in such cases.

Patients With Gross Pathological Lesions of the Intestine.—Resection of the affected part of the small intestine may be successful in selected cases of regional ileitis. Other cases require general dietary treatment.

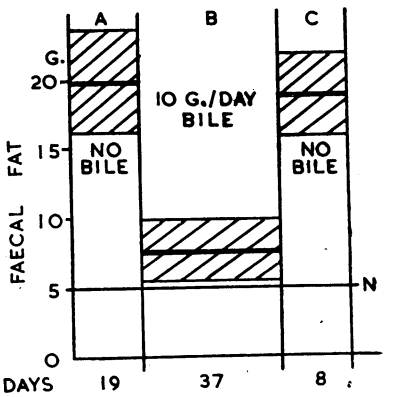

FIG. 5.—Effect of bile on faecal fat level in child with bile salt deficiency. Periods A and C—no bile; period B—oral administration of 10 g. of bile ("desibyl") a day. Heavy line shows mean value for the period; hatched area indicates standard deviation.

Gluten-induced Enteropathy.—These patients must be placed on a wheat-gluten-free diet. This involves the exclusion of a wide range of foods, since wheat flour is widely used in infant foods, pudding mixtures, sauces, soups, confectionery, and custard powders, as well as in bread, cakes, and biscuits. The patient may have any foods that do not contain wheat or rye gluten—including wheat starch. It is important to ensure that the calorie intake is adequate and to keep the protein intake on the high side. In children a response may be expected within six to eight weeks, but in adults the gluten-free diet may have to be continued for six months or more. If vitamin or other deficiencies are present they should be appropriately treated.

Tropical Sprue.—It is probably wise to exclude all fried food from the patient's diet and in severe cases to cut down fat intake. The patient should be treated with folic acid parenterally—5 mg. a day should be adequate. If he does not rapidly respond to treatment he should be removed from the endemic area—preferably to a more temperate climate. Patients that develop the more chronic form which no longer responds to folic acid should be given antibacterial therapy. In the small series here studied various combinations have been used: a five-day course of succinylsulphathiazole 6 g. a day, followed by five days of chloramphenicol, 1.5 g. a day, and then five days of chlortetracycline, 1.5 g. a day, has proved most effective. The final choice of antibacterial agents has not yet been decided; there are a number of objections to the use of chloramphenicol and chlortetracycline. This matter is still under investigation.

Non-tropical Sprue.—Vitamin or other deficiencies should be treated by appropriate replacement therapy. Antibacterial therapy may be dangerous to these patients, especially if the syndrome is one of long standing. It is usually unwise in such cases to attempt to alter the intestinal flora, except by dietary means. Cortisone may be helpful. The beneficial effect is maintained only so long as the therapy is continued.

Anastomotic Syndromes.—These are usually relieved by appropriate surgical treatment. Antibacterial therapy may be useful pre-operatively.

Summary

" Idiopathic steatorrhoea " can now be classified into the pancreatogenous, hepatogenous, and enterogenous groups. The last named can be further differentiated into gluten-induced enteropathy, tropical sprue, or one of the anastomotic syndromes.

Steatorrhoea is taken to mean the passage of loose, bulky, pale, fatty, offensive stools. The steatorrhoeic stool has an increased water content, increased dry weight, normal amount of pigment, which is paler than normal, increased amount of long-chain fatty acids, and increased short-chain volatile fatty acids. The long-chain fatty acids may be of dietary or non-dietary origin, or both. The non-dietary fat may be excreted, or it may be synthesized in the intestine. Increased activity of intestinal organisms may play an important part in the stool changes. Steatorrhoea is, usually, secondary to defective intestinal absorption.

The dietary regime is planned to reduce bacterial activity in the intestine. Specific measures in appropriate cases include replacement of pancreatic enzymes or bile salts, a gluten-free diet, folic acid therapy in early sprue, antibacterial therapy in more chronic cases, and corrective surgery. Cortisone is helpful in some cases of idiopathic steatorrhoea.

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A three-and-a-half year study of the physical and mental health of children in Florence is being carried out jointly by Harvard University and the University of Florence. A third interim report was published in November, 1954, at Florence, and describes three projects. The first was to study the influence of a mid-morning half-hour rest, and the second that of a mid-morning snack. Variations of physiological constants were noticed in the course of the morning, but were not affected by a mid-morning rest, while children who had a snack did not have the fall of blood pressure and pulse which appeared in the control children. A decoding test showed that a rest and snack avoided the loss of concentration noticeable at 12.30, when neither was available. The third project was a comparison between classes attending three State elementary schools, one for boys, one co-educational, and one founded by Professor Codignola to provide a less formal education designed to develop initiative. The pupils of the last, though from bad environments and with lower intelligence levels initially, were plainly superior to those of the other two in both intelligence and personality development in the higher classes.

STEATORRHOEA

A REVIEW OF 40 PATIENTS, WITH PARTICULAR REFERENCE TO DIAGNOSIS

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It is generally agreed that the amount of fat in the faeces should not be greater than 10% of the amount of fat ingested each day (Cooke *et al.*, 1946). Steatorrhoea is the term used when this amount is exceeded.

It is convenient to classify steatorrhoea into two main groups : (1) *Primary steatorrhoea,* in which no aetiological factor for the steatorrhoea can be found during life and in which no abnormal underlying disease is found in those cases that come to laparotomy or necropsy. This includes idiopathic steatorrhoea and tropical sprue in adults and coeliac disease in children. Many authors, including Thaysen (1932) and Cooke *et al.* (1953), consider that these three conditions are all manifestations of the same disease process, the aetiology of which is unknown. (2) *Secondary steatorrhoea,* in which a definite lesion that might well cause faulty fat absorption is diagnosed during life, and can always be found at laparotomy or necropsy. This group can be readily subdivided into two : (*a*) cases in which the clinical picture is suggestive of idiopathic steatorrhoea—for example, pancreatitis, post-operative blindloop syndrome ; and (*b*) cases with steatorrhoea, but with some other clinically obvious mechanism of disease process—for example, biliary cirrhosis or following pancreatectomy. This subdivision is important, as a differential diagnosis must be made between idiopathic steatorrhoea and cases in group 2*a*.

It was decided to study all the adult cases of steatorrhoea admitted to the Central Middlesex Hospital during the years 1949–54 inclusive, in order to find out the relative frequency of primary and secondary steatorrhoea and to review the diagnostic features. Previously most authors have tended to consider one particular group of steatorrhoea rather than all cases (Thaysen, 1932 ; Stefanini, 1948 ; di Sant'Agnese, 1953 ; Cooke *et al.*, 1953).

We have reviewed the notes of 40 patients admitted during the past six years : the majority of them have been seen personally at some time by one or other of us (Table I). It will be seen that 12 cases have been classified with reasonable certainty as idiopathic steatorrhoea, one case as tropical sprue, and 23 as secondary steatorrhoea ; in 4 patients there was insufficient evidence to allow a definite classification into one or the other type.

Idiopathic Steatorrhoea

In establishing that a case of steatorrhoea is in fact idiopathic there are certain characteristic symptoms, signs, and *x*-ray and laboratory findings the assessment of which is necessary before making a final diagnosis.

Weight loss, lassitude, and abdominal discomfort or even pain are frequent symptoms. In contrast to this the weight