CHRONIC DIARRHOEA CAUSED BY BREAD INTOLERANCE*

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The object of this communication is to draw attention to bread intolerance as a not uncommon cause of persistent diarrhoea in adults. My experience has made it quite clear that this possibility is not taken into consideration by clinicians of the highest standing both inside and outside medical schools. This is exemplified by the histories given below of 4 patients with chronic diarrhoea of over 20 years' duration who had been through many competent hands. One of these patients is myself. These patients had been seen by specialist physicians and gastroenterologists, and had been investigated in teaching hospitals. One had eventually been referred to a psychiatric clinic in Scotland for his complaint, and was seen by me while on holiday in South Africa. During the past 20 years I have encountered many examples of bread intolerance. Numerous forms of treatment had been tried, including various dietary restrictions, but never in my experience had bread restriction been suggested by physicians to any of the patients.

As long ago as the 1930's Sir Arthur Hurst[1,2] propagated the concept which he named 'carbohydrate intestinal dyspepsia'. He stressed a flatulent distension of the colon rather than diarrhoea as the main manifestation, although he did state that the diarrhoea could occasionally be severe. He observed that the onset often dated from an acute intestinal disorder, and attributed the condition to a hypermotility of the small bowel. This allowed insufficient time for the digestion of starch in its cellulose envelope to be completed in the small bowel, with the result that the undigested starch reached the large bowel where bacterial fermentation took place, producing the symptoms described. Sir Arthur Hurst stated categorically, and in my experience wrongly, that blood, even occult blood, is never present in this condition.

I have borne in mind the possibility of carbohydrate intolerance as a cause of diarrhoea since the early 1940's. A trial of the exclusion of carbohydrate was made in all patients with persistent diarrhoea where no cause could be found. Until 1956 I confined this approach to patients in the subacute and subchronic groups described below, whose diarrhoea was usually a sequel to an acute bowel infection. It was only an experience in my own case in 1956 which made me appreciate that patients with diarrhoea of many years' duration and considerable severity, described below under the group labelled 'chronic cases', might also be intolerant to bread. Such patients would usually be incorrectly diagnosed as having chronic colitis, ulcerative colitis, steatorrhoea, nervous diarrhoea, etc.

Hurst advised the exclusion of the following articles from the diet in cases of intestinal carbohydrate dyspepsia: potatoes, carrots, onions, beetroot, artichokes, parsnips, green peas, lentils, rice, cereals, biscuits, and 'the avoidance of bread for lunch and dinner'. Initially I followed this policy and in addition advised avoidance of all carbohydrate including sugar, and the total instead of partial restriction of bread. When improvement followed the patient was allowed to re-introduce one of the restricted items at a time. It eventually seemed to me that, where improvement was obtained, the avoidance of bread alone was all that was necessary, except in the case of one patient in whom sugar proved to be the cause of the diarrhoea. When improvement was well established it usually proved possible to re-introduce limited quantities of bread without ill-effects.

The factor which is responsible for bread intolerance has not been identified. This communication has been entitled 'bread intolerance' and not 'carbohydrate intolerance', thus making no unwarranted assumptions. Possible mechanisms are considered later.

CLINICAL PICTURE OF DIARRHOEA CAUSED BY BREAD INTOLERANCE

Clinical Types

Intolerance to bread may simply cause a flatulent dyspepsia, or it may cause diarrhoea.

1. *Subacute diarrhoea.* In these cases the diarrhoea follows an acute gastro-intestinal infection. Weeks or months later the diarrhoea still persists, though less severely than in the acute phase.

2. *Subchronic diarrhoea.* This refers to a syndrome seen in ex-servicemen in the first decade after World War II. As the years passed such patients were encountered less and less frequently. Most of them had had an acute bacillary or amoebic infection during the war, and subsequently were troubled with occasional bouts of diarrhoea which might take the form of an occasional single urgent stool or numerous stools over several days. They were usually labelled as having an 'irritable colon' or a 'spastic colon'. Some of them responded to bread restriction.

3. *Chronic diarrhoea.* Persons who had had diarrhoea for many years, which might date from childhood, or might start in adult life following an acute gastro-intestinal upset. Such patients were usually diagnosed as having nervous diarrhoea, chronic colitis, spastic colon, and even ulcerative colitis. Four examples of this condition are described below. All the patients had had the condition for more than 20 years, and all responded dramatically to the exclusion of bread from their diet.

4. *Flatulent dyspepsia without diarrhoea.* The patient may present with flatulence without diarrhoea and respond well to bread restriction. In some patients with diarrhoea controlled by bread restriction, the development of excessive flatus on reintroducing a little bread in the diet was a warning that further indulgence in bread would result in a recurrence of the diarrhoea.

Symptoms

The general condition of the patients is usually good and, unlike patients with ulcerative colitis, they rarely lose weight in spite of a long history of diarrhoea. They are not anaemic, the erythrocyte sedimentation rate (ESR) is normal, and they do not develop a pyrexia.

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Patients may be subject to an occasional single urgent stool a few times a month, they may experience bouts of diarrhoea lasting a few days with weeks of freedom between bouts, or they may suffer prolonged periods of severe diarrhoea lasting weeks or months. In these exacerbations blood may be seen occasionally in the stools. Between the bouts of diarrhoea the stools may be formed, although in some cases there may not have been a formed stool for many years.

Outwardly the patients usually appear quite healthy; however, this appearance belies the very considerable mental suffering and the social inconvenience of living with the ever-present hazard of a sudden urgent stool at the most inconvenient times. One of the features of this disorder is the appearance of a sudden urgent call to stool when up to a moment or two previously the patient has been feeling well with 'all quiet on the Abdominal Front'.

The following example highlights the urgency of this condition. One of the patients with chronic diarrhoea described how he was walking down a street in the West End of London one day, feeling perfectly well. Suddenly he felt a slight looseness and weakness in the abdomen. After walking on a hundred yards or so the urge to stool became so intense that it was irresistible. He saw an open door of what looked like an apartment house between two shops. He peered in, saw a staircase, dashed up it and encountered two astounded persons on the stairs. He cried out urgently for the whereabouts of the toilet. It was pointed out to him. He darted in, but too late! He could not get his trousers down in time.

Numerous investigations are usually performed without finding a cause for the diarrhoea. Antibiotics are tried, and residue-free diets, milk-free diets, anti-amoebic treatment, and tranquillizers are prescribed. The doctor begins to dread the appearance of the patient in his consulting room. The patient, too, becomes desperate and introspective and develops an anxiety state. He watches every symptom in the hope that some new detail may help the physician to help him, and reports them in detail to the harassed doctor. Nothing makes a doctor more irritable than being presented with a lot of minute details about a patient he cannot help. He makes it quite apparent to the patient that he is a neurotic and a nuisance, and in desperation may refer him to a psychiatrist, who, in turn, fails to be of help.

Physical Examination

During exacerbations there may be considerable abdominal distension and general abdominal tenderness. The abdomen may be so tender to touch that the patient may not be able to wear a belt. One patient stated that during bad phases he wanted to walk flexed. Walking erect induced the sensation of adhesions being pulled on.

The stool is usually porridgy in consistency, but may be watery. During severe exacerbations blood may appear in the diarrhoeic stool.

Microscopy of the stool is negative, except for undigested food residues. The presence of undigested starch granules in particular may be commented upon.

Sigmoidoscopy may be normal or show a shiny mucous membrane covered with excess mucus; occasionally hyperaemia may be present. The passing of the sigmoidoscope during an exacerbation may induce bleeding from the mucous membrane.

CASE HISTORIES OF SOME CHRONIC PATIENTS

Case 1

The author — 21 years of diarrhoea following an acute gastro-intestinal upset at the age of 23.

I had always considered myself a very fit person with no previous illness of significance. One morning in January 1935, while a 23-year-old medical student, I was working in the Physiology Laboratory on a research project during the university vacation. I suddenly felt ill, vomited and fainted. After coming round, urgent diarrhoea was experienced, and for about 10 days there were a dozen or more watery stools a day. I happened to be spending a holiday in the home of a specialist physician at the time and was treated conservatively. No medicaments were given and I was fed mainly on barley water, lemon juice and tea without milk. The acute attack subsided, but formed stools were not experienced again for 21 years. They remained usually porridgy, with occasional exacerbations associated with watery stools. With severe exacerbations during the war years, blood was sometimes seen.

After six months a gastro-enterologist was consulted in Cape Town. He diagnosed a 'lienteric diarrhoea' and advised dilute hydrochloric acid with meals. This produced no relief and after three months it was discontinued.

In December 1935 I went to a Karroo farm for the university vacation and there the condition became much worse. (In the light of the final diagnosis, this exacerbation may have been due to the coarse home-made farm bread.) I returned to Cape Town and was investigated at the Medical School of the University of Cape Town. A stool was examined and cysts of *Entamoeba histolytica* were reported. A course of anti-amoebic treatment with emetine and carbarsone was given, but without benefit. All kinds of foodstuffs were avoided, particularly 'roughage' and milk, without any success.

In June 1937, while engaged in research at the Guy's Hospital Endocrine Clinic, an exacerbation occurred. Sir Arthur Hurst was consulted and I was hospitalized. Stool examinations showed only Charcot-Leyden crystals. This was accepted as suggestive of previous amoebic infection. A barium meal and follow-through examination showed intestinal hurry, but there was no other significant finding. Another course of anti-amoebic treatment was given, including emetine, carbarsone and yatren retention enemas — again without benefit.

I continued to have one or two porridgy stools a day, which caused no real discomfort. About one day a week I would have a sudden very urgent explosive stool associated with abdominal colic. These attacks were very distressing because they could occur at most inconvenient times, and it was essential that a toilet be reached within a couple of minutes. Once the stool was passed I usually felt quite well within a few minutes, except for a somewhat 'loose' feeling in the abdomen. However, on two occasions, once in 1941 and once in 1947, I fainted during such attacks. After an attack (or prophylactically if I thought one imminent), I would take a large dose of colloidal kaolin and half a teaspoonful of chlorodyne. This would make me feel 'firmer' and more 'settled' in the abdomen. There was always a somewhat sore feeling about the abdomen.

I joined the South African Medical Corps in 1940. The symptoms continued and I never moved without a supply of colloidal kaolin and chlorodyne. In 1941, for the first time, bad attacks were occasionally associated with streaks of blood. In 1944 in Natal the diarrhoeic attacks became very severe. Stools were examined and cysts of *Entamoeba histolytica* were reported. A third course of anti-amoebic treatment with emetine, bismuth iodide, emetine by injection, yatren retention enemas and carbarsone was undergone. This treatment again produced no significant improvement, and so matters continued until 1955. I always kept a supply of colloidal kaolin and chlorodyne strategically stored in my home, my office, my mother's house, and my motor car.

In the summer of 1956 I thought I was putting on a little excess weight around the abdomen, and I resolved to counter this 'middle-aged spread' by avoiding carbohydrates. Formed

stool immediately developed — for the first time in over 20 years. Eventually one slice of bread a day could be taken without untoward effect, and latterly two slices of bread a day have been tolerated. There has been no relapse in the past five years, except when I have overindulged in bread and cake.

In retrospect it seems amazing that I had not tried carbohydrate restriction on myself until 1956, because I had been conscious of it as a possible factor in the causation of diarrhoea since 1940. Sir Arthur Hurst also did not consider it in my case. I think the reason is that we both accepted that I had chronic amoebic colitis.

Case 2

Diarrhoea of more than 30 years' standing — no history of preceding acute bowel infection.

A well-to-do bachelor, director of a chain of furniture stores in Aberdeen, Scotland, came to South Africa on holiday in January 1957, aged 52. He stayed with his brother-in-law, who is a doctor. He was finding his diarrhoea so troublesome that he endeavoured to persuade his brother-in-law to accompany him to the USA in search of a cure. Before agreeing to this proposition the doctor referred him to me.

The patient stated that he had had chronic diarrhoea for at least 30 years. He remembered seeing a specialist for the condition at the age of 22. As far as he could remember he did not have diarrhoea in childhood. He did remember having been subject to attacks of biliousness as a child, and also remembered being forbidden liquorice as a little boy, but he could not say he was subject to diarrhoea until he was 22. He stated that he might have attacks of diarrhoea lasting 2 or 3 days a week. On such days he might pass 5 or 6 porridgy stools. In all the years he had never had more than a week's freedom from diarrhoea. He saw many specialists in the United Kingdom and on the Continent without avail.

Nothing had ever been found, and his diarrhoea was regarded as nervous; he was under the care of a psychiatrist for his condition in Aberdeen, and he thought that his diarrhoea tended to be worse when he had worries.

When I saw him in January 1957 he looked well-covered and quite fit. Clinical examination was negative. He stated that he had had stool examinations, sigmoidoscopies, barium meals and barium enemas on numerous occasions. I told him that I did not propose to repeat the investigations. Since they had all been negative I would recommend a trial of a diet low in starch. I saw him 10 days later, when he said that he was much improved. His stools were less frequent, they were not so urgent, and they were mostly formed. The projected trip to the USA was cancelled, and he went back to Scotland. A letter was subsequently received from the physician in charge of the psychiatric clinic which he attended in Aberdeen, asking rather poignantly for details of the treatment that had so improved his condition.

Five months later I happened to be in Newcastle-on-Tyne. I phoned the patient in Aberdeen and he stated that his improvement persisted provided he did not indulge in dietary indiscretions. There was some tendency to relapse during periods of mental stress. In a recent letter (August 1961) he stated that his improvement continued to be maintained.

Case 3

Miss V.R., a nursing sister, aged 43 — diarrhoea since childhood.

She was first admitted to Groote Schuur Hospital under another physician in January 1956, in the throes of a severe bout of diarrhoea. She stated that she had been subject to these bouts since childhood, and that the attacks had become more frequent and more prolonged as the years went by. The hospital records stated that she was having up to 30 stools a day when admitted. Bright-red blood was present in the stools and she was vomiting 2 or 3 times a day. Her temperature on admission was 100°F. She was not anaemic, her haemoglobin level being 14·5 G. per 100 ml., and her ESR was only 2. There was generalized abdominal tenderness along the entire course of the colon.

Stool examinations performed on numerous occasions were always negative.

Sigmoidoscopy. Notes stated: 'Mucosa red and redundant. More than hyperaemia. One cannot see the blood vessels. Proctitis'.

She was looked upon as having a form of ulcerative colitis, and was treated with penicillin and streptomycin parenterally, and sulphathalidine orally. She was also given tinct. opii, 'largactil', vitamins, and liver by injection, and a low-fat, low-roughage diet. Her pyrexia settled, but her notes stated that the diarrhoea did not improve much on this regime, and on discharge she was given a course of 'diodoquin' (diiodohydroxyquinoline) in case there was an amoebic basis to her condition.

She was readmitted to Groote Schuur Hospital on 1 February 1961. She stated that since her discharge from hospital 5 years previously she never had a formed stool, and would average 6 or 7 stools a day. She would have to get up at least twice a night to have an unformed motion.

Numerous *stool examinations* were again negative. *Sigmoidoscopy* showed a hyperaemic, granular mucous membrane. *Biopsy* was negative. The Widal test, and agglutination tests for brucellosis were negative. The *liver function tests* were normal. Her *Wassermann reaction* was negative. A *barium-meal examination* showed a normal jejunal and ileal pattern with no bowel dilatation. At one hour the head of the meal reached the distal transverse colon. The X-ray features were 'not those of a malabsorption syndrome, but this does not exclude steatorrhoea of pancreatic origin'. A *cholecystogram* and *intravenous pyelogram* were normal.

In view of the failure of previous methods of therapy in this patient, it was decided to try a carbohydrate-free diet. Within a few days diarrhoea had ceased and the patient had semi-formed stools. She was gradually allowed to resume all carbohydrates except bread, and was discharged from hospital feeling well. On 16 March she was readmitted to the gynaecological wards for a hysterectomy and right salpingo-oophorectomy for a chronic pelvic infection.

In May 1961 she was seen at the medical outpatient department. There was still no recurrence of her diarrhoea, and no cramps or other abdominal discomfort. She was having two comfortable semi-formed stools daily on a diet that excluded only bread.

Case 4

Mrs. D.M.L., aged 57 — diarrhoea of about 23 years' duration, insidious onset.

She stated that the diarrhoea started insidiously in December 1938. She would experience several watery stools a day, and occasionally there were severe exacerbations for which she was given sulphasuxidine. She was advised not to eat curry or spicy foods, but this produced no improvement. Later she was advised to avoid fruit, but this also proved of no avail. About 18 months before being seen she began to correlate certain dyspeptic symptoms, such as a feeling of pressure in the chest after food, with the eating of bread. She decided to restrict her bread to one slice at breakfast and noticed a marked improvement, not only in the dyspeptic symptoms, but also in the diarrhoea. For the first time in *22 years* the stools became semi-formed, and were reduced to 2 daily.

If she eats bread or cake the diarrhoea recurs, but she finds that potatoes in the amounts she is accustomed to eat do not induce loose stools.

Case 5

Diarrhoea due to sugar intolerance — 10 years' duration.

I have come across one example of diarrhoea due to sugar intolerance in an adult. Mr. R.B., a White male aged 49, on the administrative staff of the Cape Provincial Hospital Services, developed a coronary thrombosis in 1958. He had suffered from bouts of abdominal cramps and diarrhoea which started insidiously about 10 years previously. He would have 3 or 4 watery stools 'mixed with faeces in small lumps', but blood never appeared in the stools. The condition had been labelled 'spastic colon'. In September 1958 he developed a posterior myocardial infarct. He estimates he was in the habit of taking 20 - 30 teaspoonsful of sugar a day with his tea

and other dishes. In an effort to lose weight he eliminated bread and sugar from his diet, and discovered that his diarrhoea and cramps ceased on this regime. Eventually he established that it was the elimination of sugar and not bread that was responsible for the improvement. As soon as he resumes the use of sugar in fairly large amounts the diarrhoea recurs. This is the first case I have encountered of diarrhoea due to sugar.

DISCUSSION

Apart from acute exacerbations, patients with this condition usually look quite well, but most of them suffer severe mental stress because of sudden urgent calls to stool at most awkward times. Such episodes can be very inconvenient, a psychological diagnosis very unhelpful, and cure by the simple procedure of restricting bread after decades of suffering very gratifying.

It is interesting to observe that in spite of the fact that Sir Arthur Hurst drew attention to the entity he named intestinal carbohydrate dyspepsia 30 years ago, and in spite of the publicity given to gluten-induced enteropathy in the past decade,[3] none of the patients I have encountered had been advised by their physicians to try the effect of avoiding carbohydrate or wheaten products.

What is the Factor Responsible for Bread Intolerance?

It is true that most of the patients could control their diarrhoea adequately by bread restriction alone. This does not mean that the erring factor is confined to bread. The diarrhoea may be due to a wider intolerance to carbohydrate, and success from bread restriction alone might well be due to the fact that this is the form in which most people take the main portion of their starch. Hurst's suggestion that the primary trouble was a condition of *hypermotility* of the small bowel resulting in inadequate time for the digestion of starch, seems to describe one feature of the condition, but not its cause.

An *allergy* would also seem to be unlikely because the patients whose diarrhoea followed an acute bowel infection showed no suggestion of such allergy before. Moreover, they can usually tolerate small, but not large quantities of bread eventually. This suggests an inability to digest the usual ration, rather than an allergy which would be expected to manifest itself even with small quantities of bread.

Is Gluten Intolerance a Factor?

It seems improbable that the diarrhoea in these cases is due to gluten intolerance. Gluten is found in flour derived from wheat, barley, rye and oats, and has been shown to be responsible for the steatorrhoea of adult coeliac disease. Thus gluten has a wide distribution in foodstuffs and the mere restriction of bread has not been reported to produce significant benefit. In fact Avery Jones and Gummer[4] stated that even tasting gluten-containing food and spitting it out can cause a relapse. The patients described in this communication demonstrated no such intense sensitivity. These authors also stated that adults take several months to recover on a gluten-free diet. The patients with diarrhoea referred to in this paper responded to bread restriction in a few days.

Enzyme Deficiency

The facts seem to be best explained by the assumption that bread intolerance may be a manifestation of an enzyme deficiency. As a rule this seems to date from an acute gastro-intestinal upset, but occasionally no such history can be elicited and some cases may be constitutional.

The enzymes concerned in starch digestion are amylase or maltase. The imperfectly digested starch would reach the large bowel, there being acted on by intestinal bacteria causing a fermentative diarrhoea.

Recent work has provided evidence in infants of diarrhoea due to carbohydrate intolerance resulting from a deficiency of various enzymes. I can find no reference to any similar work in adults.

Holzel *et al.*[5] in 1959 described infantile diarrhoea due to a deficiency of intestinal lactase in two siblings. This produced a failure of absorption of lactose. Bacteria cause a fermentative breakdown of the lactose in the large bowel, resulting in diarrhoea.

Weyers *et al.*, in 1960[6] and 1961,[7] described diarrhoea due to enzyme deficiencies in 3 children. In two cases it was due to an invertase deficiency so that sucrose could not be broken down. In the other case maltase was deficient so that starch could not be properly broken down to glucose. Because of the excessive intestinal fermentation, the stools contain abnormally large quantities of lactic acid. Unfortunately facilities were not available for determining the lactic acid in the stools in my patients.

SUMMARY

1. Attention is drawn to bread intolerance as a not uncommon, and widely unrecognized, cause of diarrhoea in adults.

2. As a rule the condition seems to be a sequel to an acute bowel infection. No such history may be elicited from some patients and their diarrhoea may be due to a constitutional defect.

3. Intolerance to bread may cause a flatulent dyspepsia, or it may cause diarrhoea.

4. Subacute, subchronic and chronic forms of diarrhoea are described. Four cases are discussed in which diarrhoea was controlled after 20 or more years' duration.

5. Because most cases could be controlled by bread restriction alone, the condition is referred to as bread intolerance, thus making no unwarranted assumption as to the causal factor or mechanism. It is suggested that the condition may be due to a deficiency of a starch-splitting enzyme. Adequate results may be achieved by excluding bread alone, probably because that is the form in which most people take the largest portion of their carbohydrate.

6. Reasons are given why these patients are thought not to have an allergy or a gluten sensitivity.

7. A case of diarrhoea due to intolerance to sugar in an adult is also described.

I wish to thank Dr. S. Bank for introducing me to case 4.

REFERENCES

1. Hurst, A. F. and Knott, F. A. (1931): Quart. J. Med., **24**, 171.
2. Hurst, A. F. (1932): *British Encyclopaedia of Medical Practice*, vol. 4, p. 371. London: Butterworth.
3. Dicke, W. K. (1950): *Coeliakie*. M.D. thesis, Utrecht.
4. Jones, A. F. and Gummer, J. W. P. (1960): *Clinical Gastroenterology*. Oxford: Blackwell Scientific Publications.
5. Holzel, A., Schwarz, V. and Sutcliff, K. W. (1959): Lancet, **1**, 1126.
6. Weyers, H. A., van de Kamer, J. H., Mossel, D. A. A. and Dicke, W. K. (1960): *Ibid.*, **2**, 296.
7. Weyers, H. A., van de Kamer, J. H., Dicke, W. K. and Ijsseling, J. (1961): Acta paediat. (Uppsala), **50**, 55.