# Section of General Practice

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## Bread and Tears—Naughtiness, Depression and Fits Due to Wheat Sensitivity

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WE would never punish a child for something we knew he could not help doing, but what if we thought the child *could* help it? Just for a moment imagine you were Anna's parents, and think what you would have done—here is her story:

*Case I.*—Anna M., aged 2. She had severe acute tonsillitis and otitis media when she was 11 months old. From then on she had woken at night with two or more crying attacks lasting over a period of some twelve months. Often she seemed awake but confused not knowing anybody and would sob until she went off to sleep again. The more severe attacks would be ushered in with a piercing scream. While in her pram, her mother described her as having a "far away look" at times. She always "behaved badly" and did not play well with other children, being spiteful to them. She was pale and had a poor and unpredictable appetite. Her motions varied; sometimes they were loose and yellowish with a very offensive smell, at other times they were normal. Examination revealed little else beyond a pale child who was thin, and perhaps the absence of fat was most marked in the buttocks. She was placed on a gluten-free diet, which she started on a Friday. She had a bad screaming attack that night, but on Saturday night was "grizzly only". On Sunday night she slept the whole way through the night without waking for the first time for a year. In the next two weeks she had two screaming attacks, each of which had been preceded by an accidental lapse in diet. On one occasion a well-meaning friend had given her a proprietary ice cream and on the other the dog had stolen some bread put out for the rabbits, had taken it on to the lawn where he and Anna had shared it. When she was next seen, she appeared much happier, behaved well, was eating well and had gained weight. The other children had all commented on how much nicer Anna was now. She has remained well, but lapses of diet still adversely affect her.

Study of over 40 similar cases has led me to formulate a syndrome which has been called the pre-cœliac syndrome. Typically a child between 1 and 5 years becomes naughty and difficult a few days after the onset of an acute infectious illness such as measles or gastro-enteritis. He is irritable, negativistic and spiteful, sleep is disturbed and he wakes up many times in the night and often screams; his appetite is poor, he fails to gain weight, his abdomen is often distended and the stools may become bulky, pale and offensive. This condition, if left untreated, usually rights itself after a month or two, but may last for much longer, in which case slight *petit-mal*-like attacks may develop, in addition to worsening of the other symptoms. I have been placing these children on a gluten-free diet at the earliest opportunity, and the symptoms respond dramatically, usually within two or three days. They relapse if a premature return to normal diet is made.

Here are two more examples of the syndrome:

*Case II.*—Peter B., aged 4. At 20 months he had gastro-enteritis after which his mother noticed progressive deterioration in his behaviour, and he became wet and dirty day and night, when previously he had been dry and clean. A month later he started to have "blank turns". I thought that these were *petit-mal* attacks and treated him with sedatives. At this time he had "night terrors" and slept badly. There followed a period of improvement lasting about three months until he had an attack of influenza. After this he became much naughtier and completely uncontrolled, being extremely difficult to examine—in fact "a horrid child". He began to have pale stools and his weight remained stationary. When he was 2¼ he had another attack of diarrhœa and vomiting and deteriorated still more. The attacks were more frequent and no longer influenced by sedatives, notwithstanding a doubled dose, and his behaviour became stranger still. Transferring him from the nursery to his high chair in the dining room nearly always brought on an attack unless his attention was held by someone playing with him. The attack would start by him becoming stock still and staring in front of him, sometimes for as long as a minute; then his head would twitch, there was a fluttering of his eyes and a spasmodic movement chiefly of the upper limbs, and he would often "pant". During this time there was a rush of words and sentences, generally lacking coherence, and often ending with a scream. The attacks were worse following any excitement during the previous twenty-four hours.

A further curious symptom was a dryness of the mouth, the child seeming to have difficulty in eating anything other than moist foods. His appetite was very poor and highly capricious. His play was characterized by over-activity, charging about the nursery, often knocking into furniture and indeed the wall, so much so that anything breakable had to be removed from the room. His speech at this time was most odd; he talked very slowly with an exaggerated drawl so that some words seemed as though they would never stop. His gait was strange, and he was wobbly if made to stand still. An EEG was abnormal by reason of excess high voltage fast rhythm, but there was no epileptic activity seen, the pattern suggesting "a diffuse organic process". I considered the possibility of cœliac disease and I put him on a gluten-free diet.

The response was dramatic—his behaviour improved, and the attacks disappeared. Sedation was discontinued. Three weeks later, he became naughty again, the trouble was traced to some flour-containing blancmange. Removal of this from the diet restored him quickly to normal. He has never really looked back since then, and now is a charming little boy, apparently normal in every way. A more recent EEG showed much less fast activity than in the first record, but was still abnormal by reason of definite alpha activity in the left occipitotemporal areas. In April 1955 he relapsed following the eating of chocolate for a week or so—so he must stick to his gluten-free diet.

*Case III.*—Frances W., aged 2. She had a four months' history of naughtiness, screaming at night, loss of appetite and failure to gain weight following gastro-enteritis. Her stools were pale and foul-smelling and her abdomen was prominent and buttocks small. There was a steady response to a gluten-free diet with sharp relapse of symptoms when returning to normal diet. She had to remain off gluten for four months but then was able to return to normal diet without relapse.

*The diagnosis of pre-cœliac syndrome* is easy. The main symptoms (Table I) were shown in the 3 cases described.

TABLE I.—SYMPTOM ANALYSIS OF PRE-CŒLIAC SYNDROME

| | | % |
|---|---|---|
| 1. | Child becomes naughty, irritable and "difficult" | 90 |
| 2. | Stools are bulky, pale and offensive | 90 |
| 3. | Disturbed sleep and screaming at night | 85 |
| 4. | Poor appetite with tendency to abdominal pain | 80 |
| 5. | Failure to gain weight | 50 |
| 6. | Prominent abdomen and small buttocks | 30 |
| 7. | *Petit-mal*-like attacks | 25 |
| 8. | Skin rashes and skin irritation | 10 |
| 9. | Abdominal pain with nausea | 10 |

Sleep disturbance is a common presenting symptom. Abdominal pain mainly seems to be due to distension of the small bowel with gas. The naughtiness is characterized by being the sort that other children dislike, readily distinguishable from the sort of naughtiness which makes other children heroes of the kindergarten.

There is a very real chance of a child being punished for naughtiness; this may not be the only reason why a child is wrongly punished on account of bread being eaten at an unfortunate time, for adults may get a somewhat similar trouble after infective illnesses, causing them among other things to be highly irritable.

*Case IV.*—Mr. S., aged 41. A week after influenza this man became very irritable and depressed with a headache and some diarrhœa. I regarded him as an early case of "headache/insomnia/depression (H.I.D.) syndrome" (the name I have given to the adult condition). He was placed on a gluten-free diet with a complete remission of symptoms twenty-four hours later. It was then that he confided to me that on the day the condition developed he had punished his son far more severely than the occasion justified, and looking back he was sure that his own mental state was the reason.

"Headache insomnia depression syndrome" in adults is as readily recognizable as is pre-cœliac syndrome in children.

Typically, two to fourteen days after the onset of an acute infection, such as influenza or gastro-enteritis, the patient develops a headache, starts being wakeful at night and becomes progressively depressed. Abdominal distension and diarrhœa may occur and are often associated with pale, offensive stools; pruritus ani may be a prominent feature. A gluten-free diet brings prompt relief of all symptoms.

*Case V.*—Mr. L., aged 48. Had just returned from a four months' visit to Damascus where the insanitary cooking conditions predisposed to four or five infective diarrhœa attacks. He was passing bulky, pale stinking motions at frequent intervals, and had considerable excoriation and inflammation of the skin around the anus. In addition he was feeling in "rather poor spirits" and was "edgy and difficult" at the office and prone to headaches. He was treated with a gluten-free diet, which caused a remission of his symptoms, the nervous ones being the first to improve. He stuck to the diet rigidly for the first four weeks but "let up" a bit after that. This back-sliding promptly resulted in a deterioration in the stools with a severe recurrence of his anal skin soreness. These quickly got better again once he kept rigidly to his diet and he has had to remain on it.

*Case VI.*—Mrs. M., aged 44. Developed "blinding headaches" with insomnia, depression and pruritus ani following a severe virus infection associated with meningism—she gave a history of similar symptoms following dysentery in India on many occasions. Her symptoms responded promptly to a gluten-free diet, but extreme sensitivity remained. One chocolate, a piece of "cœliac bread" or a small glass of whisky was sufficient to re-produce the headache lasting for about two days.

*Comparisons.*—Two syndromes have been described both of which appear to be cured by a gluten-free diet. Nevertheless, one cannot conclude that because the gluten-free diet seems to effect a cure, the condition is caused by gluten in bread. Such additional evidence as is available must be examined before drawing such a conclusion.

Does not the very close similarity justify pre-cœliac syndrome being regarded as the human counterpart of canine hysteria? (Tables II and III).

TABLE II.—COMPARISON BETWEEN PRE-CŒLIAC SYNDROME AND CANINE HYSTERIA

| *Canine Hysteria* | *Pre-cœliac Syndrome* |
|---|---|
| Onset often associated with infection. | Onset usually associated with infection. |
| Unfriendly and frightened. | Unfriendly and frightened. |
| Will bite the hand that feeds it. | Spiteful and kicks mother and doctor. |
| Has very disturbed nights. | Has very disturbed nights. |
| Abnormal slow unsteady walking. | Abnormal slow staggering gait. |
| Has dry mouth. | Has dry mouth. |
| Has characteristic fits. | Has similar fits (*see* Table III). |
| Has pale, bulky, foul-smelling stools in later stages of the disease. | Has pale, bulky, foul-smelling stools frequently. |
| Has severe skin irritation and scratches a lot. | Many cases have skin irritation with or without rash. |
| Most cases respond promptly to diet which excludes chemically treated flour. | Most cases respond promptly to gluten-free diet. |

TABLE III.—COMPARISON OF FITS IN CANINE HYSTERIA WITH THOSE IN PRE-CŒLIAC SYNDROME (*See* CASE II.)

| *Canine Hysteria* | *Pre-cœliac Syndrome* |
|---|---|
| Attacks induced by nervous strain or change of environment. | Attacks induced by nervous strain or change of room. |
| Frightened look at beginning of fit. | Frightened look at beginning of fit. |
| Sits in sphinx-like posture at beginning of fit. | Sits stock still and gazes into space at beginning of fit. |
| Jerks head backwards at beginning of fit. | Jerks head backwards at beginning of fit. |
| Runs round cage barking furiously. | Runs round room shouting or screaming. |
| Knocks food and water dishes over and gets cage into filthy mess. | Knocks into walls apparently without seeing them. |
| When running stops, staggers round like drunken person. | When walking, has staggering drunken gait. |
| Often shakes itself at end of fit. | Often shakes himself at end of fit. |
| Looks very miserable at end of fit. | Looks very miserable at end of fit and often cries. |
| Fits stop quickly when chemically treated flour is withdrawn from the diet. | Gluten-free diet stops fits quickly. |
| The longer they have had fits, the longer they take to recover fully on diet free of chemically treated flour. | The longer they have had fits, the longer they take to recover fully on gluten-free diet. |

(The description of canine hysteria fits is from Mellanby, 1946.)

*Discussion.*—Clearly there is a basic similarity of disease pattern between pre-cœliac syndrome, H.I.D. syndrome, cœliac disease and canine hysteria. Not only is the symptomatology similar but also they all respond to the withdrawal of wheat flour from the diet. Whereas cœliac disease remains relatively rare, I contend that pre-cœliac syndrome and H.I.D. syndrome are very common conditions. I have been able to diagnose some 50 or 60 cases in the past two-and-a-half years in a private general practice with two infant welfare clinics per week.

I believe that these syndromes are the result of a sensitivity to a digestion product of wheat flour, at a time when patients are specially liable to develop this, i.e. when they have an infection that can cause cell damage. Some may say these changes I have described are the result of direct toxicity by the pathogen following the illness. I do not think this is true because I have cases where the preceding cell damage has been caused by trauma

(concussion) and not by any infective process at all. Belief in my explanation of these cases demands acceptance of two fundamental concepts:

(1) That damage to nerve tissue can be followed by sensitization to certain common allergens.

(2) That such sensitization once caused can be temporary only provided the allergen is withdrawn early.

Allergists have described cases such as mine in the past, but unfortunately scant attention has been paid to their observations by the rest of the profession (Rowe, 1944; Walker, 1954).

It is quite true that the majority of cases get better anyway, but they often take quite a long time to do it—weeks, or even months elapse before cure results. All are very glad when it is over, and anyone who can cut short this miserable convalescent period is sure of gratitude. Quite apart from this, permanent damage to the brain cells may be left after some of the longer illnesses, so that treatment must be started before the "point of no return" is reached.

Falconer and his colleagues (1955) found such damaged areas at operation in some of their cases suffering from temporal-lobe epilepsy and many of their cases had a preceding history of infection followed by personality changes similar to my cases. May not some cases of temporal-lobe epilepsy be end-results of these syndromes when the damage has been severe and mainly cerebral, just as cases of cœliac disease may be end-products when the damage has been mainly intestinal? Should we not re-examine our cases of *petit-mal* and behaviour and sleep disorders in children, also cases of reactive depression? Cases of recurrent headaches and insomnia might also be reviewed; in them you may find one or other of these syndromes, and have the satisfaction of being able to put them right. Whatever the significance of my findings may be, it is certain that at least we have in the gluten-free diet a fine symptomatic treatment.

Finally, I must face the most difficult problem of all. Bread and other flour products have formed the staple diet of the inhabitants of Europe and America for centuries. Is it possible that this common article of diet has been acting in the way I have suggested without anybody recognizing it, or is there some factor which has made these disorders much more common in recent years? Certainly nervous disorders in general seem to be increasing considerably in most strata of society at the present time. Ever since Mellanby (1946) showed a link between canine hysteria and agenized wheat-flour many people have been wondering whether the chemical treatment of flour has been adversely affecting the health of the population. Nevertheless, I do *not* think that agene alone is responsible for these syndromes, nor incidentally do I accept the current view that it is the sole cause of canine hysteria.

I have observed in many of my cases that the severest reactions have been precipitated by *stale* wheat flour. This suggests that the basic toxic allergen may be found in the staling process of flour itself and flour products. We know that vitamin E is progressively destroyed during the staling process. When flour is treated by agene or chlorine dioxide, vitamin E is destroyed more quickly and completely (Horder *et al.*, 1954). Let our chemical research workers find the cause of its destruction, and our offending allergen may be revealed. At the moment this remains but an idea, but further research will find the truth, and when it is found many another door may open.

## REFERENCES

FALCONER, M. A., HILL, D., MEYER, A., MITCHELL, W., and POND, D. A. (1955) *Lancet*, i, 827.
HALLIDAY, J. L. (1953) *Lancet*, i, 742.
HORDER, Lord, DODDS, E. C., and MORAN, T. (1954) Bread. London; p. 97.
MELLANBY, E. (1946) *Brit. med. J.*, ii, 885.
ROWE, A. H. (1944) Elimination Diets and the Patient's Allergies. London; p. 131.
SHELDON, G. C., and YORKE, A. (1953) *Lancet*, i, 577.
WALKER, V. B. (1954) *Acta allerg.*, 7, 415.