

## NUTRITIONAL DISTURBANCES IN WHITE CHILDREN AS SEEN IN PRIVATE PAEDIATRIC PRACTICE

H. ALTMAN, B.Sc., M.B., B.Ch. (RAND), M.R.C.P. (LOND.), M.R.C.P. (EDIN.), D.C.H., *Paediatrician, Johannesburg, and Part-time Paediatric Assistant to the Transvaal Memorial Hospital and University of the Witwatersrand*

In order to determine the incidence and types of nutritional disturbance seen in White children, I studied the records of all children referred to me in private paediatric practice in Johannesburg during the past 7 years, and included in the survey all children who presented some disturbance of growth or specific nutritional disorder. I must stress at the outset that the majority of these children come from middle class or higher income groups, since the poorer sections of the White population usually receive their paediatric treatment at the Provincial hospitals. However, because a number of poorer families are covered by various medical aid schemes, some children from the lower income groups are included in the series. Hospital patients

have not been included. It will become immediately evident that malnutrition owing to restricted access to food, such as that seen in the Bantu population, was rare in the children studied, and that the nutritional disorders seen were usually due to maternal mismanagement or secondary to other disorders such as malabsorption syndromes.

### *Failure to Thrive*

The commonest complaint relating to the child's nutrition for which he is referred to the paediatrician is undoubtedly failure to thrive. Not infrequently this reported growth failure is purely imaginary, and a result of the mother's anxiety and ignorance. One often sees children



who are obviously robust, and even above the average height and weight for their age, but whose parents are concerned because the former large weekly weight increments have diminished. Nevertheless, poor weight gain, and perhaps associated short stature, is a real and common problem, and in this series there were 143 such infants and children. In the group below 6 months of age there were 63 infants, and in 45 of these the cause of the failure to thrive was undoubtedly underfeeding, in most instances associated with maternal anxiety and inability to manage the child. In the remaining 18 infants there were miscellaneous conditions present which were probably the primary cause for the failure to thrive. Seven of the infants were born prematurely and their physical progress continued steadily, but their weight remained below the average for the full-term infant,<sup>2</sup> whereas most of the other prematurely-born infants in due course attained the average weight for full-term infants. Other conditions responsible for poor growth included congenital heart disease with heart failure, chronic diarrhoea and malabsorption syndromes, congenital anomalies of the gastro-intestinal tract, reticulosis and fungal infections, particularly moniliasis of the mouth.

There were 80 children above 6 months of age who failed to thrive. In 56 of these the obvious feature was the anxiety of the child, invariably associated with parental anxiety and mismanagement. There was no associated organic ill-health in these 56 children as determined clinically by physical examination or laboratory investigation. Among the other 24 children were a group of 7 prematurely-born infants whose growth had remained somewhat tardy. Various illnesses were associated with the poor growth in the remainder, including recurrent respiratory infections, heart disease, eczema, primary tuberculosis, mental retardation, and malabsorption syndromes.

It would thus appear that underfeeding in the younger age group and parental anxiety in the group as a whole were the main causes of the failure of the children to thrive. When organic illness was occasionally associated with the growth failure, it was usually easy to diagnose.

#### *Kwashiorkor*

Although the clinical picture of kwashiorkor<sup>3</sup> is extremely rare in White children of the middle and upper income groups, the condition is certainly not unknown in the lower income groups. The typical findings of hair and skin changes, oedema and hypoproteinaemia were found in 3 children whose diets were identical with that characteristic of Bantu children who develop kwashiorkor. In a further 2 children from middle-class homes, the picture of kwashiorkor had obviously resulted from the use of a hopelessly inadequate diet given to alleviate allergies. These children had infantile eczema, and all milk products had been withdrawn or severely curtailed, but no protein substituted. At 1 year, one child's diet consisted of cereal with 1 ounce of milk daily, soup powders, and glucose water. The second child was being fed on a very dilute soya-bean preparation with no other protein intake. Both children had the typical clinical and biochemical features of protein malnutrition and responded rapidly, with no exacerbation of the eczema, when cow's milk feeding was started. Less severe forms of kwashiorkor were seen in

another 3 children whose diets were obviously inadequate, and in a further 5 whose malnutrition state was probably secondary to chronic diarrhoea or malabsorption.

#### *Malabsorption Syndromes*

Most workers in this field<sup>3-5</sup> have recently tried to differentiate the various aetiological factors in this bizarre and interesting group of patients, and further advances can be expected within the next few years.

True coeliac disease<sup>6</sup> due to gluten sensitivity is rare in South Africa. Of the 3 children whom I have treated only one was of South African stock, the remaining 2 having been born in Germany. Their response to a gluten-free diet was excellent and rapid.

Fibrocystic disease of the pancreas<sup>7</sup> was diagnosed slightly more frequently than coeliac disease and I have the clinical records of 6 proved cases. Two have since died of chronic lung pathology and cor pulmonale. A further 2 are subject to recurrent respiratory illnesses and are partially disabled, but the remaining 2 seem to be doing quite well at present on prophylactic antibiotics and pancreatic extract.

Food intolerance is an indubitable cause of malabsorption, although one which has aroused much controversy. Cow's milk allergy,<sup>8</sup> presumably due to protein sensitivity, is commonly diagnosed in paediatric practice in Johannesburg, but the diagnosis is seldom substantiated. I have seen only 5 cases where the diagnosis did not appear to be in doubt. These infants repeatedly developed diarrhoea and vomiting when cow's milk feeding was attempted, but eventually thrive on human breast milk. A state of severe malnutrition may develop before these infants begin to respond to treatment, and intravenous therapy is usually required during the acute phase of the illness. The intolerance to cow's milk may be only temporary, and in 2 of the abovementioned children cow's milk was introduced by giving only a single drop daily and gradually increasing the amount until he was 'desensitized'.

Lack of specific carbohydrate-splitting enzymes from the small bowel has been described recently as a cause of diarrhoeal disorders and malabsorption in infancy.<sup>9-15</sup> Absence of lactase, maltase and sucrase cause a specific disaccharide intolerance, and still more rarely a monosaccharide or polysaccharide intolerance has been postulated. The condition is suspected when there is persistent diarrhoea for which none of the usual causes can be found, and routine treatment is ineffective. Elimination of the offending carbohydrate from the diet leads to an immediate cessation of symptoms. The diagnosis is confirmed by performing a jejunal mucosal biopsy and demonstrating the enzyme defect. I have seen 2 children in whom an absence of lactase was the probable cause of persistent diarrhoea with malabsorption and consequent malnutrition. Jejunal biopsy was not possible, but the children thrive when lactose was eliminated from their diet, and relapsed when lactose was again given. The blood-sugar levels of these children also remained low after oral administration of lactose.

A temporary malabsorption state following an infective diarrhoea is not uncommon, and the resultant steatorrhoea may be severe, causing marked weight loss and a state resembling coeliac disease. Fortunately, the eventual out-



come is usually satisfactory and recovery complete. A drug-induced exudative enteropathy with malabsorption has also been described in this context, neomycin being a known offender.<sup>16</sup>

When the above causes of persistent diarrhoea have been eliminated, one finds that there is still a small group of children in whom the diarrhoea and malabsorption state continue despite all forms of treatment and dietary change. These children develop severe nutritional disturbances, often with low serum proteins and secondary rickets, and about 50% of them die, the other half eventually recovering after a stormy passage, during which time intravenous infusions of blood and plasma are usually required. In some of these infants there is an obvious excessive loss of protein in the stool, and the name 'protein-losing enteropathy' has been coined.<sup>17</sup> The cause of the malabsorption is unknown, but is probably an enzyme defect in the small bowel mucosa. I have seen 8 such infants, 4 of whom died.

Finally, in the miscellany of malabsorption syndromes, there are a number of infants who have recurrent diarrhoea over a period of some months, but between attacks are apparently quite well. A persistent pathogenic organism in the bowel is occasionally responsible, but in the majority no cause is found, and the suggestion of an absorption defect has been made.<sup>18</sup> These children continue for months without thriving, causing much anxiety to the parents and medical attendant. Fortunately, the majority recover eventually.

### Anaemia

Anaemia as a nutritional manifestation does not present a problem in private practice as I see it. Of the 50 cases of anaemia in my records, only 16 cases of iron-deficiency could be attributed to poor intake of iron, and a number of these were associated with recurrent minor illnesses. Megaloblastic anaemia was encountered twice in association with kwashiorkor, and secondary megaloblastic change was seen in the bone marrow during periods of rapid red-cell regeneration following haemolytic crises. The other causes of anaemia were secondary to diseases such as leukaemia, haemophilia, lymphadenoma, and the haemolytic-uraemic syndrome of Gasser.<sup>19,20</sup> It is interesting to note that the iron-deficiency anaemia of prematurity<sup>21</sup> is completely obviated by the prophylactic administration of iron to all prematurely born infants.

### Rickets

Dietary vitamin-D deficiency as a cause of rickets is practically unknown in White children in private practice. Of the 10 cases of rickets I have seen during the past 7 years, 4 were probably due to inadequate vitamin-D intake, 4 were secondary to malabsorption states and 2 the result of renal tubular defects.

### Obesity

The final nutritional disturbance commonly encountered in private paediatric practice is obesity.<sup>22-25</sup> This important problem of adult medicine commonly starts in the latter part of childhood, i.e. in the pre-pubertal or early adolescent period. Of the 54 cases analysed, 36 were girls and 18 boys. In 41 of the children, i.e. about 75% of the series, it was obvious that there were severe emotional problems in the children, often associated with a poor school performance well below the potential for the known intelligence. Parental anxiety and matrimonial difficulties were frequently encountered. Obesity was a problem in one or other parent in only 10 of the children, and only 1 child, a boy of 6 years, had an endocrine disorder, a suprasellar cyst having been found on routine radiological examination of the skull.

### SUMMARY

All cases of nutritional disturbances in White children seen in a private paediatric practice in Johannesburg during the past 7 years were analysed.

Primary nutritional disturbance owing to inadequate food intake is almost unknown in this population. The problem of failure to thrive is the main concern in infancy, and the problem is usually the result of mismanagement by an anxious mother. More specific nutritional disturbances are usually the result of organic pathology such as malabsorption syndromes rather than of inadequate food intake.

The main nutritional problem in the older paediatric age-group is that of obesity. Again, emotional factors seem to be commonly associated.

### REFERENCES

1. Drillien, C. M. (1961): *Arch. Dis. Childh.*, **36**, 1.
2. Brock, J. F. and Autret, M. (1952): *Kwashiorkor in Africa*. Geneva: World Health Organization.
3. Brock, J. F. (1961): *Recent Advances in Human Nutrition*. London: J. & A. Churchill.
4. Di Sant'Agnes, P. A. and Jones, W. O. (1962): *J. Amer. Med. Assoc.*, **180**, 308.
5. Heiner, D. C. and Lahey, M. E. (1962): *Pediat. Clin. N. Amer.*, **9**, 975.
6. Sheldon, W. (1959): *Pediatrics*, **23**, 132.
7. Anderson, D. H. (1938): *Amer. J. Dis. Childh.*, **56**, 344.
8. Kunstadter, R. H. M. and Schultz, A. (1953): *Ann. Allergy*, **11**, 426.
9. Weijers, H. A. and Van de Kamer, J. H. (1962): *Acta paediat. (Uppsala)*, **51**, 371.
10. Levin, B., Oberholzer, V. G., Snodgrass, G. J., Stimmler, L. and Wilmers, M. J. (1963): *Arch. Dis. Childh.*, **38**, 220.
11. Cornblath, M., Rosenthal, I., Reischer, S., Wybregt, S. and Crane, R. K. (1963): *New Engl. J. Med.*, **296**, 1271.
12. Carson, N. A. J. and Neely, R. A. (1963): *Arch. Dis. Childh.*, **38**, 574.
13. Lifshitz, F. and Holman, G. H. (1964): *J. Pediat.*, **64**, 34.
14. Holzel, A., Schwarz, V. and Sutcliffe, K. W. (1959): *Lancet*, **1**, 1126.
15. Anderson, C. M., Kerry, K. R. and Townley, R. R. W. (1965): *Arch. Dis. Childh.*, **40**, 1.
16. Jacobson, E. D., Chodos, R. B. and Faloon, W. W. (1960): *Amer. J. Med.*, **28**, 524.
17. Lahey, M. E. (1962): *Pediat. Clin. N. Amer.*, **9**, 689.
18. Ingomar, C. J., Mullertz, S. and Terslev, E. (1964): *Arch. Dis. Childh.*, **39**, 79.
19. Gasser, C., Gautier, E., Stech, A., Siebenmann, R. E. and Oechstin, R. (1958): *Schweiz. med. Wschr.*, **85**, 905.
20. Shumway, C. N. and Terplan, K. L. (1964): *Pediat. Clin. N. Amer.*, **11**, 577.
21. Schulman, I. and Smith, C. H. (1953): *Amer. J. Dis. Childh.*, **86**, 354.
22. Eberlein, W. R., Bongiovanni, A. M. and Jones, T. T. (1957): *Pediat. Clin. N. Amer.*, **4**, 949.
23. Lloyd, J. K., Wolff, O. H. and Whelen, W. S. (1961): *Brit. Med. J.*, **2**, 145.
24. Wallace, W. W. (1964): *Pediatrics*, **34**, 303.
25. Bruschi, H. (1958): *Pediat. Clin. N. Amer.*, **5**, 613.