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Some Paediatric Problems of Australian Aborigines

by

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Australia is generally considered one of the "developed" nations; technically advanced, with high standards of living and health, and with one of the lowest infant mortality rates in the world. This is true for the vast majority of Australians who are of European origin, but not for those of aboriginal descent, the ethnic minority who are now recognized to have serious health problems resembling those typical of poorer communities in other parts of the world.

The exact geographical origins of the Australian aborigines are uncertain. Some evidence suggests that they come from the Asian land mass. Certainly, they had been in "Terra Australia" (the Great South Land) many centuries before the establishment of a British penal colony in what is now New South Wales, in the late past of the Eighteenth Century. The subsequent colonization of the continent with utilization of land for farming , herding cattle, and other activities led to European occupation of aboriginal tribal hunting grounds, and eventually, to substantial disintegration of their traditional tribal customs. Yet, in spite of almost two centuries of European settlement and the firm establishment of modern westernized living in Australia today, the aborigines have had difficulty in adapting the European ways. To a large extent, aborigines have lived only on the fringes of the European society in this country and problems have arisen because of conflict between the desire to preserve at least part of their own ancient culture at the same time as taking advantage of living in an affluent country. At the present time, this is reflected in the

* This article is based on a lecture given by Dr. Gracey to the Dept. of Child Health, University of Indonesia on Thursday, 31st August 1972. Dr. Gracey is the Adolph Basser Research Fellow of the Royal Australasian College of Physicians. This work was assisted by a Grant from the Wellcome Trust, London.
Received 29 th. September 1972.

health problems of the aboriginal people, especially the children.

In Western Australia, for example, aborigines constitute approximately only 2.5% of the population, but they contribute considerably more to the medical problem of the community (Edmonds et al., 1970). Their infant mortality rate, for instance, is probably 5 or 6 times higher (about 100-120/1000 compared with 16-20/1000) than that of the white population (Macdonald, 1971). They account, also, for more than one-quarter of all deaths from the ages of 1 to 4 years despite accounting for only one-fortieth of the total population.

What underlying conditions contribute to these figures? Western Australia is the only State in the nation which keeps hospital records of the race or origin of the patient along with all other hospital statistics: this, therefore, allows us to make a more accurate assessment of the underlying disease patterns here than elsewhere. A survey of our hospital statistics for 1969 showed that diseases such as nutritional anaemia, malnutrition, gastro-intestinal infections, parasitic infestations and chronic respiratory and otitic infections figure prominently in the causes for ill-health in aboriginal children (Table 1) (Williams et al., 1969). This resembles the disease patterns seen in many poorer communities and contrasts sharply with the typical Western pattern of disease seen in white children in Australia where disorders such as asthma, neoplasms, skin diseases, accidents and acute surgical problems are more common causes for admission to hospital.

It has become apparent that substandard nutrition and chronic diarrhoea are among the main causes of the unfavourable morbidity pattern and high mortality rate of young aborigines (Kirke, 1969; Maxwell and Elliott, 1969). In our own hospital, the beds in the "gastroenteritis" ward are occupied largely by children of aboriginal descent who stay in hospital much longer than white children with gastroenteritis (mean stay more than 30 days compared with 6 days).

What accounts for the severity of diarrhoeal disease in these children? In many instances, bacterial infections, for example, by enteropathogenic Escherichia coli, Salmonellae or Shigellae, are responsible. Parasitic infestations, particularly with giardia lamblia, trichuris trichiura and, in patients from the tropics, hookworm, are common.

Lactose intolerance is a common problem in these children (Walker and Harry, 1972) as it is in many of the "developing" countries, including Indonesia. This is a very important entity to diagnose since it will respond promptly to the temporary removal of lactose from the diet. (Figure 1). While lactase deficiency may be a permanent racial characteristic of certain ethnic groups,

such as Chinese (Chung and McGill, 1968), there is good evidence that lactose intolerance in children with gastrointestinal infections is often secondary. Certainly, in Australian aboriginal children,lactose can usually be re-introduced into the diet although the period of recovery may be many months. Our practice is to completely remove lactose from the diet as soon as the diagnosis is established and then to gradually re-introduce the sugar into the diet after a satisfactory clinical recovery has been made; we aim to return our patients to their customary lactose-containing diet, but the time needed for this depends on the load of lactose tolerated as judged by recurrence of watery diarrhoea containing excessive amounts of reducing substances.

The temporary nature of lactose intolerance in such situations is of considerable practical importance since it will affect the usage of long-term lactose restriction on a large scale in countries, such as Indonesia, where lactose intolerance is very common (Suharjono et al., 1971). The long-term effects of this treatment on the growth rates and health status of children in such communities are not yet established (Luyken, 1971) and this seems an important area for future prospective investigation.

As in malnourished children elsewhere (Stanfield et al., 1965) morphological damage to the small-intestinal epithelium occurs in aboriginal children with chronic diarrhoea (Harris et al., 1970). This probably results from the combined effects of malnutrition and intestinal infection and infestation. The histological damage contributes to the problem of diarrhoea and malabsorption in these children. Lactose-induced diarrhoea, in particular, is caused in this way since lactase, the lactose-splitting enzyme, is normally present on the luminal surface of the small bowel mucosa (Miller and Crane, 1961) and is, therefore, especially vulnerable to such damage.

We are presently investigating the possible role of bacterial overgrowth in the upper intestine in the pathogenesis of chronic diarrhoea in young aboriginal children. Specimens are collected by duodenal intubation, and then subjected to detailed bacteriological investigation, using several selective media and aerobic and very strict anaerobic techniques. A recently completed study (Gracey and Stone, 1972) involved 18 aboriginal children; 10 who had been given antibiotics prior to investigation and 11 who were studied without receiving prior antibiotic therapy. (Three patients were studied both before and during antibiotic therapy). A group of 11 hospitalized Australian children of European extraction were used for "control" data; specimens were taken from these children during the course of investigation for such diseases

as coeliac disease and cystic fibrosis.

As can be seen from Figure 2, the mean microbial population of the upper small bowel in controls was less than 10^4 per ml.

This figure is in agreement with other studies from elsewhere (Drasar et al., 1969). However, there is a marked and very significant increase in the microbial counts in specimens from each group of aborigines. Interestingly, there is no significant difference whether antibiotics had been given or not.

These results indicate that microbial contamination in the upper intestine occurs commonly in these children. However, its significance is uncertain. The commonly isolated organisms are generally not considered to be enteropathogenic and include, for example, Staphylococcus aureus, candida albicans, nonpathogenic E. coli and streptococcus faecalis. We are now investigating the possible enteropathogenicity of these organisms, using several experimental animal models, and have some preliminary evidence which suggests that they may inhibit the intestinal absorption of water and small watersoluble molecules. Bacterial contamination of the gut certainly has important deleterious effects on intestinal absorption (Gracey, 1971) and this aspect needs further investigation. Lifshitz and his colleagues (1970) in Mexico have recently also found bacterial overgrowth in the upper gut in a series of malnourished infants. Monosaccharide malabsorption was a prominent clinical feature in their patients and there is other experimental evidence which implicates bacterial overgrowth in the gut in the development of this life-threatening disorder. (Gracey et al., 1971). Clearly, this information may have important implications for a large proportion of the world's children; those living in "developing" communities where malnutrition is rife and severe diarrhoeal disease so common.

The nutritional status of Australian aboriginal children is now known to be unsatisfactory (Kirke, 1969; Maxwell and Elliott, 1969). It is manifest mostly by impaired growth and poor weight gain during infancy and childhood and this pattern is punctuated by periods of illness, particularly gastrointestinal and respiratory infections. However, florid marasmus, kwashiorkor and gross vitamin deficiencies as seen in poor communities in South-East Asia, appear to be rare in Australian aboriginal children. Nevertheless, severe nutritional problems occur from time to time. We have recently seen four severely malnourished patients in whom gross depression of plasma betalipoprotein levels occurred (Gracey et al., 1972). Disturbances of plasma betalipoproteins are known to occur in protein-calorie-malnutrition, but the peculiar feature of our patients was the simultaneous presence of thorn-shaped red-blood cells, acanthocytes (Figure 3) and their

disappearance when nutritional therapy resulted in clinical recovery and return of plasma betalipoprotein levels towards normal. This combination of transient red-cell abnormality and plasma lipoprotein abnormality has not been reported previously but perhaps it occurs in countries where malnutrition is common and could provide important information about the membrane of the red cell. We have recently completed a necropsy study of aboriginal children with fatal diarrhoeal disease which emphasizes the importance of malnutrition in this group. From July, 1970 to July, 1972, 251 aboriginal children with diarrhoeal disease were treated in the Princess Margaret Hospital for Children, Perth; 13 of these patients died. Clinical evidence of malnutrition, using suggested World Health Organization criteria, was found in six and gross fatty infiltration of the liver was found in eight out of the twelve cases examined. Fatty change in the liver is, of course, one of the most striking features of protein-calorie malnutrition (Halliday, 1967) and while florid kwashiorkor is uncommon in aborigines, these findings probably indicate that many young aboriginal children have a pre-kwashiorkor state.

It probably comes as a surprise to most readers of this Journal to learn that malnutrition, severe diarrhoeal disease and unsatisfactory morbidity and mortality statistics have any place in their wealthy neighbour to the south. We hope it will not remain so for long; there is an increasing social and political awareness of these problems in our midst steps are being taken to overcome them in a systemic, co-ordinated fashion. Perhaps the lessons we learn and the mistakes we make will help overcome similar problems in Indonesia.

TABLE 1 : *Prominent causes for admission of Australian Aboriginal Children to Hospital.*

Disorder	Percentage of total (Aboriginal and non-Aboriginal) Admissions *
Nutritional anaemia	46
Gastrointestinal infestations	31
Nutritional disturbances	27
Pneumonia	16
Gastrointestinal infections	9

* In Western Australia, less than 4% of the population under the age of 16 years are aborigines. These figures therefire, indicate, how much more common these conditions ore in young aborigines.

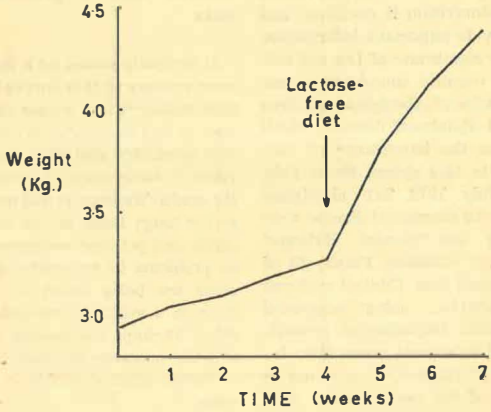

Fig. 1:
Weight chart of an Australian aboriginal infant with secondary lactase deficiency, showing rapid response to a lactose-free diet.

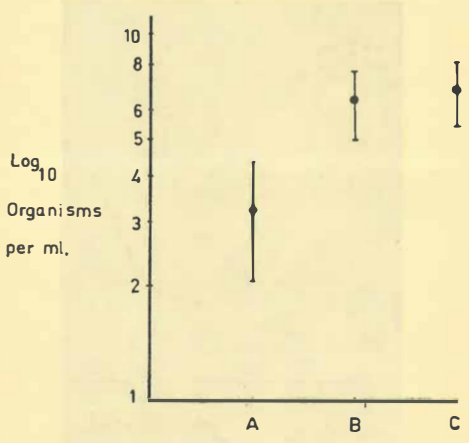

Fig. 2:

Small-intestinal microbial populations (colony counts per ml) in Australian aboriginal and non-aboriginal children. Results indicate means and upper and lower limits of these populations expressed by a standard statistical method.

A = controls (11); B = aboriginals (12 studies); C = aboriginals receiving antibiotic therapy (14 studies). Reproduced with kind permission of the Editor of the Australian and New Zealand Journal of Medicine.

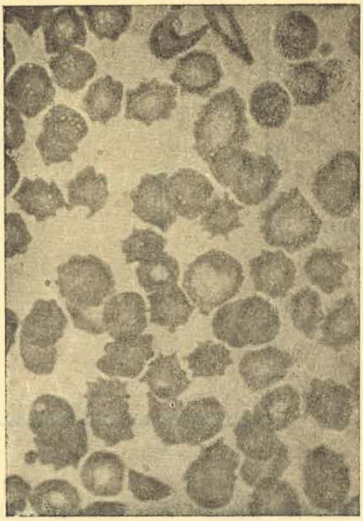

Fig. 3:
Peripheral blood film from a malnourished Australian aboriginal child showing numerous acanthocytes (thorny red blood cells).

REFERENCES

1. CHUNG, M.H. and Mc GILL, D.B.: Lactase deficiency in Orientals. Gastroenterology 54: 225 (1968).

2. DRASAR, B.S.; SHINER, M. and McLEOD, G.M.: Studies on the intestinal flora. I The bacterial flora of the gastrointestinal tract in healthy and achlorhydric person. Gastroenterology 56 : 71 (1969).

3. EDMONDS, R.; ROBERTS, R.W. and SCHLAFRIG, G.: The mortality of young children in Western Australia: The Aborigines' contribution; Aust. Paediat. J. 6: 76 (1970).

4. GRACEY, M.; BURKE, V.; OSHIN, A.; BARKER, J. and GLASGOW, E.F.: Bacteria, bile salts and intestinal monosaccharide malabsorption. Gut 12: 683 (1971).

5. GRACEY, M. and STONE, D.E.: Small-intestinal microflora in Australian aboriginal children with chronic diarrhoea. Austr. N.Z.J. Med. 3 : 215 (1972).

6. GRACEY, M.; WILSON, R.G. and PETERSEN, M.: Transient acanthocytosis in hypobetalipoproteinaemia. Austr. N.Z.J. Med (in the press).

7. GRACEY, M.: Intestinal absorption in the "contaminated small-bowel syndrome". Gut 12: 403 (1971).

8. HALLIDAY, D.R. : Chemical composition of the whole body and individual tissues of two Jamaican children whose death resulted primarily from malnutrition. Clin. Sci. 33: 365 (1967).

9. HARRIS, M.J.; DUFFY, B.J. and BEVERIDGE, J.: Studies of the small bowel of a group of New South Wales aboriginal children. Med. J. Aust. 1: 356 (1970).

10. KIRKE, D.K.: Growth rates of aboriginal children in Central Australia. Med. J. Aust. 2: 1005 (1969).

11. LUYKEN, R.: Studies on milk intolerance. A review of literature for Latin America. Paediat. Indon. 11 : 233 (1971).

12. LIFSHITZ, F.; COELLO-RAMIREZ, P. and GUTIERRES- TOPETE, G.: Monosaccharide intolerance and hypoglicemia in infants with diarrhoea. I Clinical course of 23 infants. J. Pediat. 77 : 595 (1970).

13. MILLER, D. and CRANE, R.K.: The digestive function of the epithelium of small intestine. II Localization of disaccharide hydrolysis in the isolated brush border portion of intestinal epithelial cells. Biochem. Biophys. Acta. (Aust.) 52: 293 (1961).

14. MACDONALD, W.B. : Mortality rates and morbidity patterns in Aboriginal children. A New Era (Perth) 2: 4 (1971).

15. MAXWELL, G.M. and ELLIOTT, R.B.: The nutritional state of Australian aboriginal children. Amer. J.clin. Nutrit. 22: 716 (1969).

16. SCHWARTZ, R. and DEAN, R.F.A.: The serum lipids in kwashiorkor. I Neutral fat, phospholipids and cholesterol. J. Trop. Pediat. 3: 23 (1957).

17. SUHARJONO; SUNOTO; ASWITHA BUDIARSO and SUTEDJO: Lactose Malabsorption in "healthy" Indonesian pre-school children. Paediat. Indon. 11: 251 (1971).

18. STANFIELD, J.P.; HUTT, M.S.R. and TUNNICLIFFE. R.: Intestinal biopsy in Kwashiorkor. Lancet ii : 519 (1965).

19. WALKER, A.C. and HARRY, J.G.: A survey of diarrhoeal disease in malnourished aboriginal children. Med. J. Aust. 1: 904 (1972).

20. WILLIAMS, E.J.B.; FORBES, D.A. and MACDONALD, W.B.,: Morbidity patterns
dren in Western Australia in 1969 (unpublished data).