- 3 Early DF, Nicholas M. Two decades of change: Glenside Hospital population surveys 1960-80. *Br Med J* 1981;282:1446-9.
- 4 Watts FN, Bennett DH, eds. *Theory and practice of psychiatric rehabilitation*. Chichester: John Wiley and Sons, 1984.
- 5 Social Services Committee. Second report. *Community care with special reference to adult mentally ill and mentally handicapped people*. London: HMSO, 1985. (Chairman: Renée Short.)
- 6 Anonymous. Community care [Editorial]. *Br Med J* 1985;290:806.
- 7 Department of Health and Social Security. *Better services for the mentally ill*. London: HMSO, 1975. (Cmd 6233.)
- 8 Wing JK, ed. *Long-term community care: experience in a London borough*. Psychological Medicine monograph suppl 2. Cambridge: Cambridge University Press, 1982.
- 9 Wing JK, Morris B, eds. *Handbook of psychiatric rehabilitation practice*. Oxford: Oxford University Press, 1981.
- 10 Shepherd G. *Institutional care and rehabilitation*. Harlow: Longman, 1984.
- 11 Bewley TH, Bland M, Mechen D, Walch E. "New chronic" patients. *Br Med J* 1981;283:1161-4.
- 12 Royal College of Psychiatrists. *Psychiatric rehabilitation in the 1980s. Report of the working party on rehabilitation of the social and community psychiatric section*. London: Royal College of Psychiatrists, 1980.
- 13 Pryce IG. A study of long-attending psychiatric day-patients and the services provided for them. *Psychol Med* 1983;13:875-85.
- 14 Vaughn P. The disordered development of day care in psychiatry. *Health Trends* 1983;15:91-4.
- 15 Wilkinson G. Day care for patients with psychiatric disorders. *Br Med J* 1984;288:1710-1.
- 16 Carter J. *Day services for adults: somewhere to go*. London: Allen and Unwin, 1981.
- 17 Wing JK, Olsen R, eds. *Community care of the mentally disabled*. Oxford: Oxford University Press, 1979.
- 18 MIND (National Association for Mental Health). *New directions for psychiatric day services: a report of a MIND conference held in 1980*. London: MIND, 1981.
- 19 Shepherd G, Richardson A. Organisation and interaction in psychiatric day centres. *Psychol Med* 1979;9:573-9.
- 20 Milne D. A comparative evaluation of two psychiatric day hospitals. *Br J Psychiatry* 1984;145:533-7.
- 21 Linn MW, Caffey EM, Klett CJ, Hogarty GE, Lamb HR. Day treatment and psychotropic drugs in the aftercare of schizophrenic patients. *Arch Gen Psychiatry* 1979;36:1055-66.
- 22 Wykes T, Wing JK. A ward in a house: accommodation for "new" long-stay patients. *Acta Psychiatr Scand* 1982;65:315-30.
- 23 Garety PA, Morris I. A new unit for long-stay psychiatric patients: organisation, attitudes, and quality of care. *Psychol Med* 1984;14:183-92.
- 24 Leopoldt H, Hurn RJ. Thorncliffe House. *Health and Social Services Journal* 1975;1671-2.
- 25 Leopoldt H, McStay P. The psychiatric group home. *Nursing Times* 1980;76:829-32, 866-8.

Paediatrics Among Ethnic Minorities

Afro-Caribbean and African families

JOHN BLACK

West Indian families

The term Afro-Caribbean is now commonly used to indicate people from the West Indies who are of African origin. The population of the West Indies, however, includes many other races, of which those of Indian origin are the most numerous; but most families in Britain from the West Indies are of African origin.

Afro-Caribbeans and Africans have in common three important genetically determined conditions: sickle cell disease, haemoglobin C (or more commonly the combined condition Hb SC disease), and glucose 6 phosphate dehydrogenase (G6PD) deficiency. Their family structures and social problems are, however, quite different.

Immigration from the West Indies occurred mainly during the 1950s and 1960s owing to a combination of economic depression in the Caribbean and vacancies in lower paid jobs in Britain. About 60% of the immigrants came from Jamaica, the remainder from Trinidad, Barbados, and the smaller islands. The main areas of settlement were London, Manchester, Leeds, Nottingham, Bristol, Northampton, and Reading.^{1,2} In Reading most families came from Barbados,² while High Wycombe has a mainly Vincentian settlement.² In some cases the younger children remained at home for months or years before joining their parents in Britain. Many families retain strong ties with their homeland, and the older people regard themselves primarily as Jamaicans, Barbadians, etc, whereas those born in Britain consider themselves to be British but are conscious of their roots in the West Indies. The question "Where do you come from?" should be rephrased as, "Where does your family come from?"

SOCIAL STRUCTURE AND FAMILY LIFE

Traditionally, Afro-Caribbean society is matriarchal, with the father playing an inconspicuous part. Children are looked after by

the women of the extended family. When this tradition was brought to Britain several difficulties arose. Unmarried mothers had to work and, in the absence of other female members of the family, were forced to send their infants and preschool children to baby minders, who were often unregistered and gave the children little stimulation or affection. On starting school the children suffered disadvantages

FIG 1—Blood film of patient with sickle cell disease (by courtesy of Dr J C Sharp, Department of Haematology, King's College Hospital, London).

in language, and, later, in verbal and non-verbal reasoning and reading skills³; these disadvantages tended to persist throughout their school career and afterwards, when they started to look for jobs. Similar problems arose with the children of couples who, out of economic necessity, were both working. The present generation of Afro-Caribbean children entering primary school, however, have no language problems if they have attended a nursery school and are also more likely to have a stable home and to be looked after by their parents in the preschool years; their progress in primary school is now indistinguishable from that of other groups.

RELIGION

Christianity plays an important part in the life of the Afro-Caribbean community in the United Kingdom. The Afro-Caribbeans have tended to set up their own congregations and have their own clergy. In London and the other larger cities formal Christianity and churchgoing seem to be less popular among adolescents and young adults.

The Rastafarian movement, called after Ras Tafari, who in 1930 became Haile Selassie, emperor of Ethiopia, and died in 1975, has

develop symptoms of sickle cell anaemia after the age of 3-4 months and should be carefully followed up from birth at a special clinic, and their parents should be fully informed about the care of their child. The sickle cell test does not yield positive results until the age of 3-4 months, and a positive test does not distinguish between sickle cell trait and sickle cell anaemia (table I); all patients, therefore, showing positive results should be investigated initially by a competent haematologist.

Genetic advice may be required for adolescents with sickle cell

TABLE 1—Distinction between sickle cell trait and sickle cell anaemia

	Symptoms	Anaemia	Jaundice	Splenomegaly	Peripheral film	Sickle cell test
Sickle cell trait	None	None	None	None	Normal	Positive
Sickle cell anaemia	Usually severe	Severe (Hb 7-10 g/dl in a stable state)	Usually	Yes (up to 8-10 years)	Sickle cells and target cells	Positive

attracted several followers among young West Indians.⁴ They believe that Ras Tafari, who claimed descent from Solomon, was the Messiah who will lead them back to Ethiopia, the promised land. The men may wear their hair in long ringlets (“dreadlocks”) and wear the “tam,” a knitted woollen hat in the Ethiopian colours of red, yellow, and green. The women do not cut their hair, which is worn under a scarf. Marijuana (“ganga”) is sometimes used as a means of enlightenment and a health giving drug; there is no evidence that the health of their children is in any way affected. Most Rastafarians are strict vegetarians (vegans); when the diet is strictly observed the children may develop nutritional deficiencies.

LANGUAGE

In the early days of immigration children who spoke at home what is termed Creole by linguists and patois or dialect by West Indians² had considerable difficulties on entry to school, but this is no longer so as the present generation of preschool children is usually fluent in the local form of English.

Conditions found in the children of Afro-Caribbean families

SICKLE CELL DISEASE

In sickle cell disease the red blood cells contain an abnormal haemoglobin (HbS), which causes them to form a sickle shape when exposed to low oxygen tension (fig 1). Sickle cells aggregate in the microcirculation, causing infarction, and have an increased fragility and shortened life, resulting in chronic haemolytic anaemia. The heterozygous sickle cell trait (HbAS), in which there are no symptoms under normal conditions, should be distinguished from the homozygous sickle cell anaemia (HbSS), which is usually severe, occasionally disabling, and sometimes fatal, though a few patients inexplicably have few symptoms (table I shows the distinction between sickle cell trait and sickle cell anaemia).

Figure 2 shows the geographical distribution of the sickle cell gene. Outside Africa the distribution corresponds to that of people of African origin who were dispersed by the slave trade to the West Indies and America; secondary migration has occurred recently to the United Kingdom¹ (fig 3) and elsewhere. The sickle cell gene also occurs in the indigenous population of the north coast of the Mediterranean, Saudi Arabia, round the Persian Gulf, and in some parts of India, mainly in aboriginal tribes.

In some parts of Britain the cord blood of all infants of Afro-Caribbean or African origin is examined by haemoglobin electrophoresis; both heterozygotes and homozygotes can be detected. The children who are heterozygous (sickle cell trait, HbAS) will not develop symptoms, but their parents should be told of the diagnosis and its benign outlook. Infants who are homozygous (HbSS) usually

FIG 2—Geographical distribution of haemoglobin S (reproduced with permission from editor and publishers from *Sickle cell disease*. In: Fleming AF, ed. Edinburgh: Churchill Livingstone, 1982.

FIG 3—Davis LR, Huekins ER, White SM. Sickle cell anaemia in England and Wales. *Br Med J* 1981;283:1519-21. (Reproduced with permission of the authors.)

trait, and in some areas parents who are both heterozygous or have already produced a child with sickle cell anaemia are offered antenatal testing to detect the homozygous fetus. Testing during the first trimester (8-11 weeks) is now available at some centres; it is better accepted and less distressing than testing during the second trimester, which may necessitate a late termination, usually at roughly 20 weeks. In sickle cell anaemia the affected child may present in several ways.

In infancy—The infant presents at the age of 3-4 months with failure to thrive, recurrent infections, anaemia, jaundice, and splenomegaly (the spleen usually shrinks progressively and becomes impalpable by the age of 8-10 years).

Acute illness at any age over 4 months—Whatever the diagnosis a child who is acutely ill and is known to have, or is suspected of having, sickle cell anaemia should be admitted to hospital immediately. Acute pneumococcal infections, pneumonia, septicaemia, and meningitis are particularly common. Severe sickling crises may be precipitated by any acute infection, acidosis, dehydration, hypoxia, trauma, exposure to cold, or by a general anaesthetic if special precautions are not taken. Severe shock may develop, particularly in young children with enlarged spleens when sequestration of blood occurs in the spleen. Aplastic crises are not common and may be due to infections with parvovirus,⁵ sometimes affecting several members of the same family simultaneously; megaloblastic crises due to dietary folic acid deficiency, however, are common in the West Indies and may occur in Britain in children having inadequate diets (see under Religion). The "bilateral chest syndrome" and extensive mesenteric infarction may occasionally prove fatal.

Jaundice may be due to haemolysis in a sickling crisis; to hepatitis A or B, which may also provoke sickling, hepatic crises with pooling and sickling in the liver, and signs of obstructive jaundice; or, in children over 10 years, to obstruction by bile pigment gall stones.

Localised infarction producing pain and swelling—In infants the fingers and toes are often affected ("dactylitis," the "hand-foot" syndrome). Disease of the mesenteric vessels may mimic an abdominal emergency. Hemiplegia or other neurological infarction syndromes are particularly disabling. Haematuria occurs but is rare.

SICKLE CELL DISEASE AND THE FAMILY DOCTOR

The family doctor should ensure that all his Afro-Caribbean and African patients are tested for sickle cell disease, and those with a positive result and members of their families should be further investigated. About one in 10 Afro-Caribbeans have the sickle cell trait and about one in 400 infants will have sickle cell anaemia. Anyone with sickle cell anaemia should wear a Medialert necklace or bracelet, and the parents of an affected child should contact a local branch of the Organisation for Sickle Cell Anaemia Research or the Sickle Cell Society (see box).

Prophylactic dental care is important and should be encouraged. When this is local policy parents should be strongly advised to have their children immunised against pneumococcal infection (Pneumovax, Morson) and to maintain their daily prophylactic penicillin. Folic acid supplements should be maintained if this is local policy, though present evidence suggests that in Britain aplastic crises are more likely to be due to infection with parvovirus,⁵ while megaloblastic crises due to folic acid deficiency are rare. Oral iron should not be prescribed for anaemic patients in whom iron deficiency has not been proved. Analgesia at home, for painful crises, should be confined to aspirin or paracetamol. Dihydrocodeine, dipanone, dextropropoxyphene with paracetamol (Distalgesic), and similar habit forming drugs should be prescribed only in hospital or in exceptional circumstances at home. Flying in commercial aircraft, which are normally pressurised at 2300 m (7000 ft), is a risk to those with sickle cell anaemia but not to those with the trait. The airline should be informed well before the flight and the availability of an oxygen supply other than the emergency one should be discussed; splenic infarction appears to be the commonest complication of flying, and those who have previously had an infarct are likely to do so again.

Useful addresses

The Sickle Cell Society, c/o Brent Community Health Council, 16 High Street, Harlesden, London NW10 4LX (01 451-3293).

The address of other counselling centres can be obtained from this society, which publish *Handbook on sickle cell disease: a guide for families* and other literature.

Organisation for sickle cell anaemia research (OSCAR), 200a High Road, London N22 (01 899-3300).

HAEMOGLOBIN SC DISEASE AND SICKLE CELL THALASSAEMIA

Haemoglobin SC disease and sickle cell thalassaemia are much less common than sickle cell anaemia,⁶ while HbCC disease is extremely rare. Haemoglobin SC disease and most types of sickle cell thalassaemia have less severe symptoms than does homozygous sickle cell disease. HbCC disease presents as an anaemia.

GLUCOSE 6 PHOSPHATE DEHYDROGENASE (G6PD) DEFICIENCY

Glucose 6 phosphate dehydrogenase deficiency, inherited through an X linked intermediate gene and therefore seen predominantly in men, occurs in the fairly benign African form in Afro-Caribbeans. Homozygous women are not uncommon and may be as severely affected as are the men; rarely, a heterozygous woman may be mildly affected clinically. Favism, an acute and severe haemolysis due to contact with the broad bean (*Vicia faba*) or its pollen, appears to be confined to Mediterranean and Oriental types (for further discussion see article 5: Families from the Mediterranean and Aegean Regions and article 7: Chinese and Vietnamese families.) In affected subjects the red blood cells haemolyse after

TABLE II—Drugs that may cause haemolysis in G6PD deficiency

Antimalarials	Primaquine Pamaquine Pentaquine
Sulphonamides	Sulphanilamide Sulphacetamide Sulphapyridine Sulphamethoxazole (in Bactrim, Septrin) Silver sulphadiazine (surface application for burns)
Sulphones	Dapsone (in Maloprim, prophylaxis for malaria) Thiazolesulphone
Nitrofurans	Nitrofurantoin (Furadantin)
Analgesics	Acetanilide
Miscellaneous	Methylene blue Nalidixic acid (Negram) Naphthalene Nitridazole Phenylhydrazine Toluidine blue Trinitrotoluene

Table modified from Wintrobe MM, Lee GR, Boggs DR, *et al. Clinical Hematology*. 8th ed. Philadelphia: Lea and Febiger, 1981:792, with permission from authors and publishers.

the patient has taken certain drugs (table II) or under conditions of severe hypoxia or acidosis, particularly in diabetic ketoacidosis and during acute infections such as hepatitis. Clinically, G6PD deficiency takes two forms: jaundice in the neonatal period or an acute self limiting haemolysis precipitated by one of the drugs listed in table II. In the African form spontaneous jaundice without drug contact may develop in the full term infant as well as in the preterm infant, usually on the second or third day of life, reaching a maximum on or around the third to the sixth day and subsiding by the end of the first week. Infants may also become jaundiced after contact with one of the offending drugs through the placenta, in breast milk, as part of treatment, after applications to the cord (usually menthol preparations), or after contact with babies'

clothes impregnated with naphthalene (mothballs). The timing of jaundice obviously depends on the time of contact. In Britain jaundice from G6PD deficiency is not common in Afro-Caribbean infants, but the diagnosis must nevertheless be considered when there is no obvious cause for non-obstructive jaundice. Other factors such as incompatibility of blood groups, sepsis, or reabsorption of extravasated blood may be contributory factors.

In the older child or adult a sudden haemolysis lasting about 36 hours may develop after drug contact; jaundice is not evident at the time of the haemolysis because the process is so quick. Deaths from haemolysis are rare in Afro-Caribbeans (for further discussion and a table showing the differences between the three types of G6PD deficiency see articles 5 and 7: Families from the Mediterranean and Aegean Regions and Chinese and Vietnamese families).

ACQUIRED DISEASES

In the neonatal period subaponeurotic haemorrhage appears to be more common in infants of Afro-Caribbean or African origin, though the reason for this is not known. Neonatal jaundice may occur (for further discussion see G6PD deficiency), and the breast enlargement that is sometimes seen in the first week of life may persist in some Afro-Caribbean girls until the age of 4-5 years (see also below, under premature thelarche).

During infancy and childhood the following conditions may occur.

Umbilical hernia is common in Afro-Caribbean and African children; if large or persistent (until the age of 3-4 years) it may require operation.

Keloid formation is more likely to develop in postoperative or post-traumatic scars or after burns than in other racial groups.

Nutritional deficiencies—Normally Afro-Caribbean and African children are well nourished, though a mild iron deficiency anaemia is common. Children brought up in a Rastafarian family on a strict vegetarian diet may develop nutritional rickets,⁸ and there is a risk of megaloblastic anaemia and glossitis due to deficiency of vitamin B₁₂ or folic acid.^{9,10}

Premature thelarche (premature development of breasts) seems to occur more often in Afro-Caribbean girls than in other ethnic groups. The enlargement may occur as early as 4-5 years but is not accompanied by other evidence of puberty or signs of endocrine disease. Provided that the breast enlargement remains an isolated symptom there is no reason for anxiety or specialised investigation. Such children should, however, be seen at least once by a paediatric endocrinologist and should then be examined regularly until normal puberty is established. No treatment is available or is required except reassurance.

Premature pubarche (premature development of pubic hair) also appears to be fairly common in Afro-Caribbean girls. As with premature thelarche there is no cause for anxiety or for endocrine investigation if there is no evidence of endocrine disease or other signs of incipient puberty. The management is as for premature development of the breasts.

Emotional, social, and school problems

Baby minding and deprivation—The slow development of a deprived child may be due solely to the unstimulating environment provided by a baby minder; the circumstances that lead to this situation have been described previously.

Poor school progress may be due initially to deprivation but is now more likely to be related to poor self esteem and a sense of hopelessness induced by the probability of unemployment on leaving school.

Parental attitudes to punishment—Physical punishment as a form of parental discipline has traditionally been used by Afro-Caribbean families. The use of a belt or strap, usually by the father, does not indicate a disturbed family relationship or a sadistic parental attitude and should not normally be dealt with as "child abuse" unless there is severe or repeated injury.

African families

Most African families in Britain are West Africans from Nigeria or Ghana. They usually come to Britain for further education or to obtain a professional qualification. If both parents are studying, as is often the case, the need to complete their course successfully is in competition with traditional expectations, their own and those of their families at home, that they should have a child. This may result in their child being sent, often when very young, to a baby minder during the day; or fostering may be arranged on a long term basis, often with a white couple. The problems of a Nigerian couple in London have been described by Buchi Emecheta in *Second Class Citizen*.¹¹

Religion—In Ghana about 60% of the population are Christians of various denominations; the remainder are Moslems. Dietary restrictions on pork and pork products are similar to those for Asian Moslems. Also in Nigeria Islam and Christianity are the most important religions.

Language—The main languages in Nigeria are Yoruba, Ibo, and Hausa; in Ghana Akan, Gã, and Ewe. In both countries English is the second language. Language is unlikely to be a problem to a child who has been with an English speaking baby minder or whose parents have found them a place in a day nursery. In areas where there are many children for whom English is the second language special attention is given at entry to school; but language difficulties are, nevertheless, not uncommon, particularly in Nigerian children. Difficulties may arise for a child who has remained in Africa with grandparents and come to Britain at the age of 8-9 years, to a new country and to parents and perhaps siblings who are complete strangers.

Conditions found in the children of African families

GENETICALLY DETERMINED CONDITIONS

Sickle cell disease, HbSC disease, HbS thalassaemia, HbCC disease, and G6PD deficiency all occur in African children with the same symptoms as those described for Afro-Caribbeans. In West Africans the incidence of sickle cell trait is, however, about one in five of the population, and therefore one in 200 infants can be expected to develop symptoms of sickle cell anaemia.

ACQUIRED DISEASES

For subaponeurotic haemorrhage, neonatal jaundice, umbilical hernia, and keloid formation see under the paragraphs dealing with Afro-Caribbean children.

Schistosomiasis—Both *Schistosoma haematobium* and *S. mansoni* are common throughout rural Africa and are acquired by contact with infected water from rivers but rarely infect children before they can walk independently. *S. haematobium* infection should be suspected in children who have recently been in Africa if they develop recurrent attacks of haematuria, with dysuria and frequency, or an apparently straightforward attack of acute pyelonephritis that fails to improve. Infection with *S. mansoni* is more likely to produce chronic diarrhoea with blood and mucus in the stool. Both types of infection can produce malaise without localised symptoms, with or without hepatosplenomegaly. A child in whom schistosomiasis is suspected should be referred to someone with experience of this condition.

Malaria is common in most parts of Africa, and chloroquine resistance is particularly common in Tanzania and the coastal regions of Kenya. Children should be tested for G6PD deficiency before being given primaquine (for further discussion of malaria and map see article 4: Conditions found in children of Asian families).

Hookworm (ankylostomiasis) should be considered in a child who has recently arrived from Africa and has an unexplained or iron resistant anaemia. Ankylostomiasis is unlikely in infants, town dwellers, or children from hot, dry parts of Africa (for further

discussion of hookworm see article 4: Conditions found in children of Asian families).

Female circumcision and infibulation—In Britain only late complications of the most extensive form of female circumcision, the pharaonic type (infibulation), are likely to be seen. Though widely practised in Africa, it is not performed in Ghana but is common in Nigeria, in both Moslem and Christian families. Regrettably, female circumcision has also been performed in private clinics in London. The operation, which is often carried out before the age of 10 days, consists of removing the clitoris and labia minora and all or part of the labia majora; the cut edges are held together but a small aperture is left for the passage of urine and menstrual discharge. In Nigeria a modified operation is sometimes performed with removal of the clitoris and trimming of the labia minora round the clitoris. Complications that may arise are: urinary tract infection, a poor urinary stream related to obstruction from meatal scarring or urethral stricture, complete or almost complete labial fusion, rectovaginal fistulas, implantation dermoids, keloid scarring, and in older girls, introital stenosis.

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References

- 1 Davis LR, Huekins ER, White SM. Survey of sickle cell disease in England and Wales. *Br Med J* 1981;283:1519-21.
- 2 Edwards VK. *The West Indian language issue in British schools*. London: Routledge and Kegan Paul, 1979.
- 3 Scarrs S, Caparalo BK, Ferdman BM, Tower RB, Caplan J. Developmental status and school achievements of minority and non-minority children from birth to 18 years in a British midlands town. *British Journal of Developmental Psychology* 1983;1:31-48.
- 4 Springer L, Thomas J. Rastafarians in Britain: a preliminary study of their food habits and beliefs. *Hum Nutr Appl Nutr* 1983;37:120-7.
- 5 Serjeant GR, Topley JM, Mason K, et al. Outbreak of aplastic crises in sickle cell anaemia associated with a parvovirus-like agent. *Lancet* 1981;ii:595-7.
- 6 Mann JR. Sickle cell haemoglobinopathies in England. *Arch Dis Child* 1981;56:676-83.
- 7 Gibbs WH, Gray R, Lowry M. Glucose-6-phosphate dehydrogenase deficiency and neonatal jaundice in Jamaica. *Br J Haematol* 1979;43:263-74.
- 8 Ward PS, Drakeford JP, Milton J, James JA. Nutritional rickets in Rastafarian children. *Br Med J* 1982;285:1242-3.
- 9 Campbell M, Lofters WS, Gibbs WN. Rastafarianism and the vegans syndrome. *Br Med J* 1982;285:1617-8.
- 10 Close GC. Rastafarianism and the vegans syndrome. *Br Med J* 1982;286:473.
- 11 Ennecheta B. *Second class citizen*. Glasgow: Fontana and Collins, 1974.

Further reading

- Ellis J. *West African Families in Britain*. London: Routledge and Kegan Paul, 1978.
- Fleming AF, ed. *Sickle cell disease*. Edinburgh: Churchill Livingstone, 1982.
- Anionwu E, Hall J. *Handbook on sickle cell disease: a guide for families*. London: Sickle Cell Society, 1983.
- Lehmann H, Huntsman RG. *Man's haemoglobin*. Amsterdam and Oxford: North Holland Publishing Company, 1974.
- Pollak M. The care of immigrant children. In: Hart C, ed. *Child care in general practice*. 2nd ed. Edinburgh: Churchill Livingstone, 1982.

(This article is the sixth in a series.)

What fluid intake is recommended during marathon type races (in terms of litres/hour and composition, with particular reference to sodium chloride and sugar)?

The volume of fluid lost by perspiration and evaporation during marathon racing obviously depends on the ambient temperature but may amount to 20% of body weight (around 1.5 l for someone weighing 70 kg). Under hot conditions the losses are greater still. The important thing is to drink little and often throughout the race. Inexperienced runners seldom drink enough and only rarely too much. Several "electrolyte" preparations are available commercially, but a simple solution of glucose in water with sufficient salt to make the mixture palatable is perfectly adequate and a good deal cheaper.—M G HARRIES, consultant physician, London.

What, if any, would be the effect on the eyes of a person sitting in a room with closed windows, no ventilation, and an ambient temperature of 26-30°C?

Normally the posterior three quarters of the globe is at body temperature so, assuming that under the conditions stated in the question body temperature did not change, this part of the eye would not be expected to change either. The anterior quarter, being exposed, has a rather lower temperature than the rest of the eye. Under the conditions stated the temperature of the front part of the eye would rise and depending on relative humidity within the room evaporation from the precorneal tear film could then increase. Increased evaporation would stimulate an increased blink rate and, probably increase tear production. No discomfort or change to the cornea would occur unless these reflex mechanisms proved unable to prevent drying.—ROGER A HITCHINGS, consultant ophthalmic surgeon, London.

Is it possible to sterilise a baby's bottle and teat in a microwave oven?

Strictly speaking sterilisation is not necessary, since bacterial spores need not be killed. Vegetative bacteria, however, must be destroyed. I have been unable to find any publications on the use of microwaves to disinfect infant feeding bottles and have therefore performed a series of experiments. These included tests on identical polycarbonate bottles with rubber teats that had been coated with saliva and allowed to dry. Water was added to the uppermost marking of one bottle and both were placed in the centre of the microwave. When the water was observed to boil in the bottle, both were removed and samples taken from inside the bottle, the tip of the teat, and the teat flange. No significant killing occurred at any site in the empty bottle. The full bottle was sterile, but the teat was heavily contaminated. These and other experiments showed that intracellular water in bacteria does not kill

the organism when exposed to microwaves. The feeding bottle may be disinfected when it contains water or feed, but the teat must be removed since it does not reach a high enough temperature. Because cold spots occur in domestic microwaves I recommend that bottles are processed individually and that the process is continued until the contents boil. The teat may be processed by immersing it in a fluid filled container and treating similarly. Proper cleaning of all items should not be neglected.—R H GEORGE, consultant microbiologist, Birmingham.

Ascorbic acid is used to improve the function of both baking powder and yeast not only by housewives but also by the large bakeries. Has this any justification?

The traditional method of breadmaking includes many stages that take time. The rising stage may be appreciably cut down if ascorbic acid is added. Vitamin C acts as a flour improver and as such is a key component in the Chorleywood bread process, by which about 75% of commercially baked bread is made. Although chemically a reducing agent, ascorbic acid is converted to dehydroascorbic acid, which helps retain the carbon dioxide produced by yeast in the dough. The resulting loaf is bigger and lighter. As baking destroys the original ascorbic acid bread cannot be considered a useful source of vitamin C. Makers of homemade bread can take advantage of this process by adding 25 mg of vitamin C (half a small tablet) to the liquid used with 50 g of flour.—E MORSE, nutritionist, London.

Thewlis BH. Vitamin C in breadmaking. In: Birch GG, Parker K, eds. *Vitamin C*. London: Applied Science Publishers, 1974.

A fit elderly man complains of short bouts of strange and sometimes unpleasant smells, for which there is no external cause. What might be the cause of this, and is any treatment advised?

Brief episodes might result either from local sepsis in the mouth or nose or from temporal lobe epilepsy. Local sepsis as, for example, in the teeth may usually be confirmed or excluded easily enough. Attacks of temporal lobe epilepsy of which the only manifestation was an unpleasant smell would be unusual, and it would be most important to have an eyewitness's description of whether there is any abnormal behaviour at these times such as clouding of consciousness or automatic movements—in particular, swallowing or smacking the lips. The onset of such fits in old age may be due to the effects of cerebral infarction or to a tumour, and the question of treatment clearly depends on establishing, if possible, a precise diagnosis of the cause of the symptoms.—W B MATTHEWS, professor of clinical neurology, Oxford.