Current Practice

MEDICINE IN THE TROPICS

Malnutrition in Childhood in the Tropics

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Of the many causes of ill-health among children of the tropics malnutrition is undoubtedly the most important. Apart from its direct effects on health, malnutrition also exerts an indirect effect by diminishing resistance to infectious diseases.

The infant and child mortality rates in tropical countries are high. In India, according to the latest census figures, while infants and children under the age of 5 years constitute 16.5% of the total population, deaths within this age group account for 40% of the total deaths in the country. Infant and toddler mortality rates in some of the tropical countries are shown in Table I. A curious reflection of such high mortality in infancy and childhood is that in contrast with the situation in Europe the average expectation of life in tropical countries is actually higher at the age of 5 years than at birth. These data indicate the magnitude of ill-health among infants and children in tropical countries.

TABLE I.—*Infant and Toddler Mortality Rates in Different Countries*

Developed Countries	I.M.R.	T.M.R.	Developing Countries	I.M.R.	T.M.R.
Australia	19.1	1.0	Columbia	90.0	15.6
Belgium	25.8	1.0	El Salvador	65.5	22.7
Canada	24.7	1.1	Guatemala	91.6	42.7
France	23.4	1.2	Guinea	42.0	55.4
Japan	20.4	1.9	Mexico	66.3	14.7
Netherlands	14.8	1.1	Costa Rica	86.4	N.A.
Sweden	14.2	0.8	Honduras	50.0	13.5
U.S.A.	24.8	1.0	Venezuela	48.0	5.5
			India	81.0	12.0
			Malaya	57.0	34.0
			Philippines	67.4	8.1

I.M.R. = Infant Mortality Rate per 1,000 live births.
T.M.R. = Toddler Mortality Rate per 1,000 population.

Among poor communities in the tropics undernutrition and an unhygienic environment are inseparable factors, which tend to interplay with and aggravate each other. It is therefore difficult to decide how far these mortality figures are attributable to malnutrition and how far they are the result of insanitary and unfavourable environment. However, a careful scrutiny of the data offers some suggestive indications.

A considerable proportion of infant deaths in the tropics occurs in the neonatal period. Analysis reveals that nearly 75% of these deaths are directly attributable to "premature birth." A study of over 16,000 obstetric cases in Madras in South India¹ showed that the incidence of premature births (using the criterion of body weight of 5 lb. (2.2 kg.) as normal) was 8.1% among the poor general-ward patients as against 1.6% among the well-to-do patients admitted to the special wards. Using the international criterion of a birth weight of 5½ lb. (2.5 kg.) in full-term infants as evidence of prematurity, Varkki *et al.*² found that the incidence of prematurity among infants in Coonoor (South India) was three times as high in poor communities as in the well-to-do group. Such a high incidence of prematurity may be considered as a reflection of maternal malnutrition.

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The extreme severity of the malnutrition in pregnant mothers in the poorest sections of populations in the tropics can be gauged from the results of recent surveys. In a study of the diet of pregnant women of the low socio-economic groups in South India the average daily intake of protein was 44 g. (animal protein 6 g.), of calcium 300 mg., and of iron 18 mg. The total calorie intake was 1,575.

Nutrition in Early Infancy

If the infant in the tropics manages to survive the hazards of the neonatal period he is relatively safe till the end of the first year in spite of his insanitary environment. During at least the first six months the infant receives, through his mother's milk, the nutrients which he needs in amounts which are probably not too inadequate. It is gratifying that women of the low socio-economic group in the tropics are able to breast-feed their infants successfully for prolonged periods. Instances of inability to breast-feed infants, which are frequent among the sophisticated high socio-economic groups, are extremely rare among the poor. This is surprising in the light of the diet of the nursing mothers, which provides less than 50 g. of protein daily, mainly derived from vegetable sources.

In a study among poor South Indian women it was observed³ that the daily output of breast milk varied from about 600 ml. within the first six months of lactation to about 350 ml. between the 18th and 24th months. The composition of the breast milk obtained at different stages of lactation showed that the milk was quite satisfactory with regard to the proximate principles, though not with regard to vitamins and minerals (Table II). These observations point to the need for encouraging breast-feeding by poor communities in the tropics, though it must be borne in mind that these underfed mothers are able to maintain satisfactory lactation only at the expense of their own tissues.

TABLE II.—*Composition of Human Milk*

Duration of Lactation	Protein g./100 ml.	Fat g./100 ml.	Lactose g./100 ml.	Vitamin A i.u./100 ml.	Vitamin B ₁ mg./100 ml.	Vitamin B ₂ mg./100 ml.	Vitamin C mg./100 ml.	Calcium mg./100 ml.	Phosphorus mg./100 ml.
1-6 months	1.06	3.34	7.47	79	15.3	19.0	1.9	36.3	11.7
7-12 months	1.01	3.23	7.54	66	14.5	18.1	2.5	34.1	12.0
13-18 months	1.05	3.79	7.54	65	16.5	17.2	3.2	29.8	12.6
More than 18 months	1.13	3.41	7.51	66	15.1	11.5	2.9	36.6	11.1

Growth and Development

At least for four months after birth the growth rates of Asian infants are not significantly lower than those of infants of the technically developed regions of the world. While this is true of growth rates, the actual weights of Asian infants even

within the first six months of birth are lower than those of normal infants elsewhere. In Fig. 1 the heights and weights of infants and children belonging to the poor socio-economic groups in India have been compared with those of American infants and children of corresponding ages. Among European and American infants chest circumference has been reported normally to overtake head circumference around the first year. In Asian infants of the poor socio-economic groups, however, this has been found to take place at about the third year. It is, however, interesting that no significant retardation has been observed with regard to the attainment of milestones of development in infants of the tropics.

It is the universal practice among poor communities in the tropics that infants are breast-fed for prolonged periods, often extending to two years. It is also the usual practice in many poor communities that infants are maintained solely on breast milk till the end of the first year. Supplementary foods are invariably unmodified versions of the adult diets and are almost solely cereal-based. Milk and animal protein foods figure only occasionally. Due to ignorance and prejudices green leafy vegetables and fruits are also not often included. Even such qualitatively deficient diets are often fed in quantities which are inadequate. The general impression that infants and children in the tropics are "overstuffed" with starchy foods providing adequate or more than adequate calories is not borne out by actual surveys. In view of the remarkable growth retardation the protein intake on the basis of actual body weights may appear to be only marginally deficient. However, in view of the inadequacy of calories in the diets a considerable proportion of the protein must be utilized for energy purposes. The intake of vitamins, especially of vitamin A, either preformed

or as carotene, is also low; the intake of calcium rarely exceeds 400 to 500 mg.

An elaborate account of the classical nutritional deficiency diseases affecting children will not be attempted here. Excellent descriptions of such classical syndromes as scurvy, rickets, and infantile beriberi, are available in standard textbooks. In this paper major nutritional problems affecting children of the tropics will be briefly discussed.

Protein-Calorie Malnutrition

Among the various types of malnutrition in children of the tropics protein-calorie malnutrition is the most widespread and important. Two major clinical forms have been recognized, kwashiorkor and marasmus.

Kwashiorkor

Kwashiorkor is generally seen in children between 1 and 3 years of age. The onset of the disease is insidious. Towards the latter half of the first year the child shows failure to grow and a lack of interest in the surroundings. He has frequent attacks of diarrhoea and other infections. There is often loss of appetite and increasing apathy, and the child may develop discoloration of the hair and skin and a characteristic fullness of the face. Later the ankles swell, and oedema gradually spreads upwards. It is at this stage that the child is generally brought to the hospital (Figs. 2 and 3).

Growth failure and hypoproteinaemic oedema are accepted as minimal diagnostic criteria for the disease. Oedema is maximal in the dependent parts, but ascites is uncommon. There is generally no albuminuria, and this differentiates the oedema from that of renal origin. The clinical severity of the oedema bears no relation to the ultimate prognosis or to the response of the case to treatment.

The hair can be plucked off easily and is often discoloured. The discoloration may not be present throughout the length of the hair, but only in segments, and this is termed the "flag

FIG. 1.—Comparison of height and weight of American and Indian children.

FIG. 2.—Child suffering from kwashiorkor showing oedema and skin

sign." The skin is scaly and shows characteristic changes which have been designated "crazy pavement dermatosis." The lesions start as black patches, especially over pressure points, which later exfoliate, exposing hypopigmented areas bordered by hyperpigmented zones.

Alimentary disturbances are the rule. There is marked anorexia, and diarrhoea is frequent. Pathogenic bacteria can be found in the stools in only a small percentage of the cases. However, in view of the high incidence of parasitic infestations in the tropics, ova of parasites, especially roundworm ova, are frequently seen. The faeces often show microscopic fat globules. The diarrhoea responds to high-protein therapy, and can therefore be regarded as a manifestation of protein deficiency.

The liver may or may not be enlarged. Liver biopsy invariably reveals extreme degrees of fatty infiltration, but contrary to previous belief this never leads to hepatic cirrhosis. General signs of associated vitamin deficiencies are usually present, commonly those of vitamin A and the vitamin-B complex.

There is a marked hypoproteinaemia, the reduction being greater in the albumin fraction. The serum protein and albumin levels are of the order of 4.5 and 1.9 g./100 ml. respectively. There is a reduction in many of the serum enzymes, the blood urea, and the blood cholesterol.

FIG. 3.—Child suffering from kwashiorkor showing oedema, skin changes, and moon-face.

Anaemia is often present, and is mainly due to iron deficiency, but concomitant deficiency of other haematinic factors should also be borne in mind.

Marasmus

Marasmus is seen in a younger age group, usually between 6 and 12 months of age. These infants are usually exclusively breast-fed, and the milk available is not adequate for proper growth. There is therefore a gross calorie undernutrition. A typical case of marasmus is characterized by gross underweight, marked muscular wasting, and loss of subcutaneous fat.

TABLE III.—Differences Between Kwashiorkor and Marasmus

	Kwashiorkor	Marasmus
Age of incidence	12–36 months	6–18 months
Oedema	Invariably present	Absent
Muscle wasting	Mild to moderate	Severe
Loss of subcutaneous tissue	Mild	Marked
Hair	Discoloration; easy pluckability	Normal
Skin	Scaly; dyspigmentation: crazy pavement dermatosis	Normal
Plasma proteins	Markedly reduced	Reduced
Serum enzymes	Low	Normal
Blood urea	Low	Normal
Liver	Fatty infiltration	Normal

Oedema is not seen in these infants, and the liver is free from fatty change. Skin and hair changes are absent.

Hypoproteinaemia is seen in marasmus, but to a lesser extent than in kwashiorkor. The serum protein and albumin concentrations are around 5 and 3.5 g./100 ml. respectively. Blood urea levels are normal, and serum enzyme activity is not much reduced. The major differences between kwashiorkor and marasmus are set out in Table III.

Marasmic Kwashiorkor

These descriptions of kwashiorkor and marasmus are of well-defined cases and may be considered to represent two ends of the spectrum of protein-calorie malnutrition. In practice there is often considerable overlap between these two clinical conditions. In India and Indonesia protein-calorie malnutrition often manifests in the clinical form designated "marasmic kwashiorkor." A picture of marasmus may precede the onset of kwashiorkor in these children. Again, after the disappearance of oedema in response to treatment the subjects may appear marasmic. The differences between kwashiorkor and marasmus are probably not so much due to differences in dietary patterns as to different "adaptation responses" to the deficient diets.

Treatment of Protein-Calorie Malnutrition

Therapy consists in feeding the children diets providing adequate protein and calories. The best protein source for these diets is milk. The therapeutic regimen described in this article is that used in the Nutrition Research Laboratories, Hyderabad.

Each child is given a diet providing 4 g. protein and 200 cal./kg. body weight, the composition of which is given in Table IV. Skimmed milk constitutes 80% of the total protein and wheat 20%. The former is diluted in a ratio of 1:6. The rest of the calories are provided by sugar, bananas, and butter. Since fat gives more calories per unit, butter helps in raising the calorie content of the diet and in keeping the bulk low. These children usually have concomitant vitamin and iron deficiencies, so they are given supplements of vitamins and iron.

TABLE IV.—Diet for Treatment of Protein-Calorie Malnutrition Based on Milk Protein

Ingredient	g./kg. body weight	cals/kg. body weight
Skimmed milk	9	36
Bread	20	70
Butter	7	63
Sugar	7	28
Bananas	2	

Though milk is the best protein for these children, it is expensive and rare in developing countries. Attempts are being made to incorporate locally available vegetable protein foods into these therapeutic diets. Groundnut (*Arachis hypogaea*), Bengal gram (*Cicer arietinum*), and cottonseed have been successfully used. However, these proteins have to be used at a level of 6 g./kg. body weight. Moreover, where possible incorporation of small quantities of skimmed milk gives better results. The composition of one such diet is given in Table V.

TABLE V.—Diet Containing Vegetable Protein Mixture

Ingredient	g./kg. body weight	cals/kg. body weight
Wheat flour	18	63
Cottonseed flour	6	24
Sugar	10	40
Ghee	7	63
Skimmed milk	3	12

The diets are well accepted by the children, though anorexia may restrict the quantities taken in the first three to four days. Clinical improvement, characterized by mental alertness, loss of oedema, and consequent loss in body weight, is evident within seven to ten days. Complete recovery occurs within three to four weeks, when there will have been a gain in body weight and the serum albumin will have returned to near normal levels. Recovery is retarded by the presence of infection, and tuberculosis should always be borne in mind. If it is present a response to therapy will be observed only after the institution of specific antituberculosis treatment. Antibody production is impaired in protein malnutrition, and a negative Mantoux reaction does not necessarily rule out tuberculosis. The practice followed in our clinic, therefore, is that all children who fail to improve with dietary therapy are treated simultaneously with antituberculosis drugs, whether or not they show evidence of the infection.

Troublesome diarrhoea is treated with binding reagents like pectin or kaolin. Where pathogenic organisms are found in the stools specific therapy is instituted. Since most of the cases have diarrhoea, potassium chloride is given routinely.

Vitamin-A Deficiency

After protein-calorie malnutrition the nutritional deficiency most widely prevalent in children of the tropics is that of vitamin A. Some 8% of the child population at any given time have ocular signs of vitamin-A deficiency.

The milder signs of vitamin-A deficiency are night-blindness, Bitot spots, and xerosis of the conjunctiva. The more severe form of vitamin-A deficiency is xerosis of the cornea, leading to keratomalacia, and eventually to blindness. The most common lesion, however, is conjunctival xerosis. Children with a history of night-blindness do not necessarily show any other ocular sign; and children with ocular lesions may not necessarily give a definite history of night-blindness.

The incidence of vitamin-A deficiency is particularly high in children suffering from kwashiorkor, for diets poor in protein are also deficient in vitamin A. Moreover, a close relationship exists between the metabolism of both nutrients. Serum vitamin-A levels are very low in these children, and nearly 65% of the children presenting with this dual deficiency suffer from the most severe form of vitamin-A deficiency, keratomalacia.

Early detection of vitamin-A deficiency, especially corneal xerosis, is vital because administration of vitamin A can reverse the ocular lesion in as short a period as 24 hours. Mild cases of xerosis are often missed owing to examination in poor light.

Treatment

Treatment depends on the severity of the lesion and on the presence of associated protein malnutrition. In children suffering from vitamin-A deficiency alone and in whom there is no corneal involvement 10,000 to 25,000 I.U. of vitamin A daily is administered orally until the lesions disappear, and this usually requires about three to four days. Smaller doses may then be given for a further seven to ten days.

Corneal lesions must be treated as a medical emergency with 50,000 to 100,000 I.U. vitamin A daily until complete disappearance of the lesion. The dose should then gradually be tapered off to 10,000 I.U. Corneal involvement is one of the last stages of vitamin-A deficiency, and the body stores of the child at this stage may be considered to be practically nil. Vitamin-A supplements should be continued for a period of six to eight months. Local application of an antibiotic is advocated until the lesions disappear.

In cases of kwashiorkor with vitamin-A deficiency, contrary to general belief, the oral route of administration is superior to the parenteral route. However, the vitamin should be given with a fat base either in the form of butter or some vegetable oil to facilitate absorption.

Doses higher than those mentioned above are not necessary. It has been found that in a number of children treated with higher doses of the vitamin signs of acute toxicity such as vomiting and bulging of the fontanelle occur. Even at a dose of 50,000 to 100,000 I.U. a small percentage of children may develop these signs, but they are likely to be transient.

Phrynoderma

Phrynoderma, or follicular hyperkeratosis, a nutritional deficiency disorder not uncommonly encountered in the children of the tropics belonging to the low income groups, is a condition generally seen in late childhood and adolescence.

The earlier belief that phrynoderma is a manifestation of vitamin-A deficiency still persists. However, this hypothesis is not substantiated by epidemiological, clinical, biochemical, or therapeutic studies. Vitamin-A deficiency is seen most commonly in the first six years of life, a period at which phrynoderma is relatively uncommon. Many children with lesions of phrynoderma have no other signs of vitamin-A deficiency, and the condition cannot be cured by vitamin-A therapy.

It has been established that phrynoderma is due to a deficiency of essential fatty acids. Vitamin-B complex deficiency also seems to have an associated role, for 60–70% of the patients manifest signs of vitamin-B deficiency.

The disease is characterized by the presence of projecting horny papules on the skin. The papules have a round or oval base and a sharp peak. The papules are generally hyperpigmented, vary in diameter from the size of a pinhead to that of a millet, are discrete, and tend to appear in groups. The lesions are distributed symmetrically, but localized lesions may also be seen occasionally. They are commonly present on the extensor surfaces, especially on the elbows (Fig. 4) and knees,

FIG. 4.—Elbow showing typical lesions of phrynoderma.

and in the gluteal region. They may also be seen on the back and on the posterior and lateral surfaces of the arms and thighs.

Nearly 60–80% of the patients also have signs of vitamin-B deficiency, such as glossitis, cheilosis, angular stomatitis, and nasolabial dyssebacea.

Histopathological examination reveals superficial hyperkeratosis of the epidermis and of the hair follicles. The sebaceous follicles are found to be dilated and plugged with dense keratotic material.

Treatment

The condition can be cured in most cases by the oral administration of fats rich in essential fatty acids. The improvement has been observed to be quicker when vitamins of the B-complex are given simultaneously. Parenteral administration of B-complex vitamins *per se* can also cure phrynoderma, but it has been found that recurrence is more frequent and quicker in such cases as compared with those treated with essential fatty acids. We have routinely employed safflower oil (linoleic acid content: 70%) in doses of 5 ml. thrice daily in combination with an oral B-complex preparation (thiamine 3 mg.; ribo-

flavine 1 mg.; pyridoxine 0.5 mg.; pantothenate 1 mg.; and niacin 30 mg.) with satisfactory results. Improvement is obvious in about two weeks, though six to eight weeks of therapy is needed for complete disappearance of the lesions. It needs to be stressed that even massive doses of vitamin A do not bring about any improvement.

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MEDICINE TODAY

Management of Parkinsonism

"Medicine Today" is the television series for doctors produced by the B.B.C. Advice on the preparation of the programme is given by the Association for the Study of Medical Education.

The programme on B.B.C. 2 on 5 December was on the subject of Parkinsonism. Printed below is an article prepared with the help of expert contributors to complement the television programme, which will be repeated on B.B.C. 1 on 13 December at about 11 p.m.

Many Parkinsonian patients have now been treated successfully by stereotaxic procedures, and there is considerable agreement on the indications for operation. Young patients with non-progressive and predominantly unilateral tremor often gain gratifying relief; those with marked rigidity may also respond satisfactorily. Frail patients with progressive and advanced disease do poorly, and surgery should be withheld from those with marked hypokinesia, severe disorders of gait and balance, bulbar symptoms, and evidence of intellectual deterioration. Even the most dramatic results are essentially palliative, and successfully placed lesions in selective patients do not alter the natural history of the underlying pathological process. Tremor and rigidity often recur.

Regrettably the greater number of patients are elderly and unsuitable for surgery. Every effort should be made by their doctors to encourage independence for as long as possible. Each patient poses an individual problem in management, and practical advice—from rubber-tipped walking-sticks to strategically placed aids to daily living—is greatly appreciated. Intelligent patients can often teach their doctors useful tips which can be passed on to others. Some patients with postencephalitic Parkinsonism cope remarkably well with stairs and on uneven surfaces. They should be encouraged to persist with these activities despite the anxiety often engendered in their relatives. During transient and minor illnesses patients with Parkinsonism should not spend any longer in bed than is absolutely necessary. After minor setbacks short and intensive courses of physiotherapy are often helpful, but there is little justification for sporadic visits to the clinic scattered over many months.

Drugs are generally disappointing in the treatment of Parkinsonism. Few patients gain dramatic and sustained relief, whereas undesirable side-effects are all too frequent. Though sceptics have related any amelioration of the patient's disabilities to the prescriber's enthusiasm, there can be little doubt that some patients do get useful improvement of tremor and rigidity; and a few show unequivocal relapse when anti-Parkinsonian drugs are stopped abruptly.

Most of the currently prescribed drugs are anticholine compounds, and their use stems from an early clinical observation that belladonna conferred some relief. Today naturally occurring alkaloids such as tincture of belladonna are rarely used, but a host of synthetic analogues are available. There is no convincing evidence that any one preparation is superior to the

others, and there is no rational guide to the sequence of new drugs when others have failed. The general principle is to introduce one drug at a time, beginning with a small dose thrice daily and gradually increasing the amount until optimum relief is obtained or toxic effects become intolerable. This empirical process of adjusting dosage and drug in the hope of discovering a beneficial regimen is often an exacting and frustrating task, taxing the patience and fortitude of both patient and doctor. Visual hallucinations, toxic confusional states, impaired visual accommodation, and excessive dryness of the mouth often cause patients to give up medication spontaneously before their doctor suggests this. Even in a patient who is apparently gaining satisfactory relief it is always worth while stopping treatment from time to time to ensure that the patient agrees that he is better off with his drugs.

Approved Name	Proprietary Name	Tablet (mg.)	Price/100 Tabs.
Belladonna alkaloids	Rabellon		3s. 2d.
Benzhexol	Artane	2 and 5	6s. 10d. and 13s. 8d.
Benzhexol hydrochloride	Pipanol	2 and 5	6s. 10d. and 13s. 8d.
Benztropine methanesulphonate	Cogentin	2	18s.
Biperiden	Akineton	2	11s. 6d.
Chlorphenoxamine	Clorevan	50	16s.
Diphenhydramine hydrochloride, hyoscine hydrobromide	Benacine	25 } 0.325 }	11s. 8d.
Ethopropazine	Lysivane	50	14s.
Mephenesin	Myanesin	500	12s. 8d.
Mephenesin carbamate	Tolseram	500	16s.
Methixene hydrochloride	Tremonil	5	14s.
Orphenadrine hydrochloride	Disipal	50	13s. 11d.
Phenglutarimide	Aturbane	5	13s. 4d.
Procyclidine hydrochloride	Kemadrin	5	15s. 4d.
Tigloidine hydrobromide	Tiglyssin	250	47s. 6d.

Benzhexol 2 mg. t.d.s., orphenadrine 50 mg. t.d.s., benztropine 0.5 mg. t.d.s., and ethopropazine 50 mg. t.d.s. are approximately equivalent initiating doses of commonly used drugs. Some physicians favour combinations of drugs at sub-toxic levels, but there is no agreement on optimum combinations and certainly little justification for giving more than two drugs at the same time. When increments are made gradually many Parkinsonian patients may tolerate remarkably large doses of anticholine drugs, but it is important to ensure that such heroic measures are desirable and justifiable.

There is little to choose between the various synthetic anticholine drugs, and there is very little mystique in the use of these preparations. Few other useful drugs are available. Dextroamphetamine (5 to 10 mg. twice daily) is occasionally helpful to hypokinetic patients, and antidepressant drugs are often indicated in the management of their chronic and often refractory diseases. Beta-adrenergic blocking agents and dopamine derivatives are still in the experimental stage of assessment, and their safety and place in routine clinical problems has yet to be accepted. Finally, it must be recalled that reserpine, phenothiazines and methyl dopa may cause Parkinsonian syndromes—and these drugs should not be given to patients suffering from disorders of the basal ganglia.