The World Health Organization is a specialized agency of the United Nations with primary responsibility for international health matters and public health. Through this organization, which was created in 1948, the health professions of some 150 countries exchange their knowledge and experience with the aim of achieving the highest possible level of health throughout the world.

By means of direct technical cooperation with its Member States, and by stimulating such cooperation among them, WHO promotes the development of comprehensive health services, the prevention and control of diseases, the improvement of environmental conditions, the development of health manpower, the coordination and development of biomedical and health services research, and the planning and implementation of health programmes.

These broad fields of endeavour encompass a wide variety of activities, such as developing systems of primary health care that reach the whole population of Member countries; promoting the health of mothers and children; combating malnutrition; eradicating smallpox throughout the world; controlling malaria and other communicable diseases including tuberculosis and leprosy; promoting mass immunization campaigns against a number of preventable diseases; improving mental health; providing safe water supplies; and training health personnel of all categories

Progress towards better health throughout the world also demands international cooperation in such matters as establishing international standards for biological substances, pesticides and pharmaceuticals; recommending international nonproprietary names for drugs; administering the International Health Regulations; revising the international classification of diseases and causes of death; and collecting and disseminating health statistical information.

Further information on many aspects of WHO's work is presented in the Organization's publications.

THE MANAGEMENT OF NUTRITIONAL EMERGENCIES IN LARGE POPULATIONS

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CONTENTS

Preface

This guide is intended for use by health personnel responsible for the field management of nutritional emergencies in populations, namely, the medical or allied personnel from national or provincial health services or from relief agencies in the country affected.

It is particularly concerned with severe nutritional emergencies, that is, mass starvation caused by the interruption of food supplies to the population over a long period. Unusual food shortages may be caused by major crop failures, war and civil conflicts, or natural disasters. Relief personnel responsible for short-term food distribution following a major disaster such as an earthquake or cyclone may also find these guidelines useful, although they were specifically prepared for the management of situations in which populations suffer from widespread and severe malnutrition.

No mention is made of social, cultural, or political factors that are critical during famines, nor of rehabilitation. The guide is concerned, as it were, with fire-fighting rather than fire prevention or reconstruction.

No short booklet can provide guidelines applicable to each and every situation. Adaptation and improvisation will be necessary to some extent. All the examples given are based on experience, and it is hoped that they will be helpful in the preparation of local procedures and guides for the on-site training of relief workers in each country.

ACKNOWLEDGEMENTS AND REFERENCES

Acknowledgement is due to many individuals both in and outside WHO for their valuable advice and criticism based upon long experience of field work. The authors are also grateful to the League of Red Cross Societies, the Catholic Relief Services, and Oxfam for their valuable assistance in the preparation of this guide.

Material and ideas have been drawn from many sources, but particularly from the following publications:

Guide to food and health relief operations in disasters. New York, Protein-Calorie Advisory Group (PAG) of the United Nations System, 1977.

BLIX, G., HOFVANDER, Y. & VAHLQUIST, V., ed. *Famine: a symposium dealing with nutrition and relief operations in times of disaster.* Uppsala, Almqvist & Wikell for Swedish Nutrition Foundation and Swedish International Development Authority, 1971.

KING, M.H. *Nutrition for developing countries.* Nairobi, Oxford University Press, 1972.

Food emergency manual. Rome, World Food Programme (new edition in preparation).

CAMERON, M. & HOFVANDER, Y. *Manual on feeding infants and young children.* 2nd edition, New York, Protein-Calorie Advisory Group of the United Nations System, 1976.

A debt of gratitude is also owed to the Literary Executor of the late Sir Ronald A. Fisher, F.R.S., to Dr Frank Yates, F.R.S., and to Longman Group Ltd, London, for permission to reprint Table A in Annex 6 from their book *Statistical Tables for Biological, Agricultural and Medical Research* (6th edition, 1974)

1. Normal and emergency needs

Basic facts about food and nutrition are given in Annex 1, which should be consulted by readers who are not thoroughly familiar with nutritional concepts. Energy and protein requirements in normal and emergency situations are briefly summarized below.[1]

Normal situations

Recommended intakes

The energy and protein intakes considered as safe by WHO/FAO for each age group and physiological condition[2] are shown in Annex 1.

Vulnerable groups

The energy and protein requirements of women are increased during pregnancy—by +1.5 MJ (350 $kcal_{th}$) and +15 g protein per day—and during lactation—by +2.3 MJ (550 $kcal_{th}$) and +20 g protein per day—over and above their normal requirements.

Preschool children (0–5 years) require proportionally more energy and protein for each kg of body weight than adults. They are more vulnerable to malnutrition.

Emergency situations

The WHO/FAO safe intakes of energy and protein[2] have not yet been attained by the majority of people in developing countries. In nutritional emergencies caused by food shortage, relief planning based on these standards is unrealistic. The maintenance of energy intake at a level adequate for survival must be the primary consideration.

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[1] Energy values are expressed in the SI unit, the megajoule (MJ) The equivalents in the superseded unit, the thermochemical kilocalorie ($kcal_{th}$) are given in parentheses. 1 MJ = 239 $kcal_{th}$. 1000 $kcal_{th}$ = 4.184 MJ.

[2] PASSMORE, R. ET AL. *Handbook on human nutritional requirements*, Geneva, World Health Organization, 1974 (Monograph Series, No. 61).

Table 1 shows the minimum amount of energy required to sustain life.

TABLE 1. EMERGENCY ENERGY INTAKE PER PERSON[a]

Group	Height (cm)	Emergency subsistence (for a few weeks) MJ ($kcal_{th}$) per day	Temporary maintenance (for many months) MJ ($kcal_{th}$) per day
0–1 years[b]	under 75	3 4 (800)	3.4 (800)
1–3 years	75– 96	4 6 (1 100)	5 4 (1 300)
4–6 years	96–117	5 4 (1 300)	6.7 (1 600)
7–9 years	117–136	6 3 (1 500)	7.5 (1 800)
10 years or over:	over 136		
male		7 1 (1 700)	8 4 (2 000)
female		6 3 (1 500)	7 5 (1 800)
Pregnant or lactating women		8 0 (1 900)	9 2 (2 200)
Average per day per person		about 6.3 MJ (1 500 $kcal_{th}$)	about 7 5 MJ (1 800 $kcal_{th}$)

[a] Adapted from Mayer, J. Famine relief: what kind of organization and what types of trained personnel are needed in the field. In: Blix, G. et al. *Famine: a symposium...*, Uppsala, 1971

[b] Levels for infants are similar to those recommended for normal situation.

The emergency subsistence level is the estimated level below which large-scale starvation and death should be expected if the population is of normal body size and is required to perform some work.

A prolonged maintenance diet at the level indicated above is likely to result in some loss of body weight. Supplementary feeding of vulnerable groups is essential to provide extra energy and nutrients.

Even under "normal conditions", without any emergency, the energy intake of some populations is comparable to or less than the temporary maintenance level—7.5 MJ (1800 $kcal_{th}$). When resources are scarce, it may not be justifiable to provide this amount to some segments of the population, and a level as low as 6.3 MJ (1500 $kcal_{th}$) will have to be maintained for extended periods. The decision will depend on local conditions.

2. Major deficiency diseases in emergencies

- **Protein-energy malnutrition** (PEM) is the most important health problem during a nutritional emergency.
  Severe PEM can present several forms:
  - *Nutritional marasmus* is characterized by a severe wasting away of fat and muscle ("skin and bone"). It is the commonest form in most nutritional emergencies.
  - *Kwashiorkor* is characterized by oedema, usually starting at the lower extremities.
  - *Marasmic kwashiorkor* is a combination of wasting and oedema. The treatment of severe forms of PEM is presented in Chapter 5.

- **Mineral and vitamin deficiencies** may also be important.
  - *Severe anemia* is common and requires a daily intake of iron for an extended period of time.
  - *Vitamin A deficiency,* the most important vitamin deficiency, is characterized by night blindness and/or eye lesions which may lead to permanent total blindness. The severe forms are usually associated with PEM.
  - Other deficiency conditions are less common: beriberi, pellagra, scurvy, rickets.
  - Mineral and vitamin deficiencies must be identified and the individuals affected or at risk treated by administration of the missing nutrient.

Protein-energy malnutrition (PEM)

Protein-energy malnutrition is a problem in many developing countries, even in normal times. Most commonly it affects children between the ages of 6 months and 5 years (especially around 18–24 months), i.e., at the time when they are most vulnerable to the common infectious diseases such as gastroenteritis and measles. PEM may simply be due to shortage of food, or it may be precipitated by lack of appetite and an increase in nutrient requirements and losses caused by infection.

Chronic PEM has many short- and long-term physical and mental effects, including growth retardation, a malnourished child being lighter and shorter than a better-fed child of the same age.

In times of nutritional emergency it is primarily the more acute forms of PEM that have to be dealt with. These are characterized by a rapid loss of weight and may be evident in a much wider range of age groups than usual. For example, significant numbers of older children, adolescents, and adults may also be affected.

Past experience has shown that many emergencies affect the supply of food to only a proportion of the population concerned. The situation will obviously vary from place to place, but it is often the case that only a small proportion of the total population presents clinical signs of severe PEM. For each case of severe clinical PEM there may well be 10 moderate cases and 100 children of "near normal" nutritional status. Progression from moderate to clinically severe forms is rapid.

Severe forms of PEM [1]

The severe forms of PEM are:

nutritional marasmus
kwashiorkor
marasmic kwashiorkor

Nutritional marasmus results from prolonged starvation (see Fig. 1).

The main sign is a severe wasting away of fat and muscle. The child is very thin ("skin and bone"), most of the fat and muscle mass having been expended to provide energy. It is the most frequent form of PEM in cases of severe food shortage.

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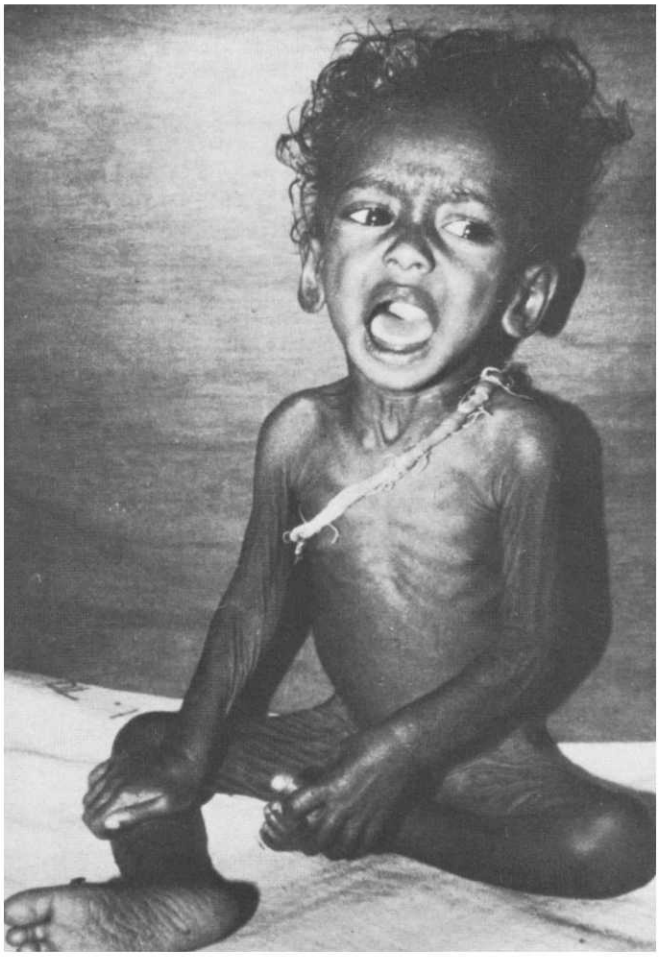
[1] For treatment, see Chapter 5.

Associated signs can be:

- A thin "old man" face.
- "Baggy pants" (the loose skin of a child's buttocks hanging down).
- The children concerned are usually active and may appear to be very alert in spite of their condition.
- There is no oedema (swelling that pits on pressure) of the lower extremities.

FIG. 1. CHILD SUFFERING FROM NUTRITIONAL MARASMUS



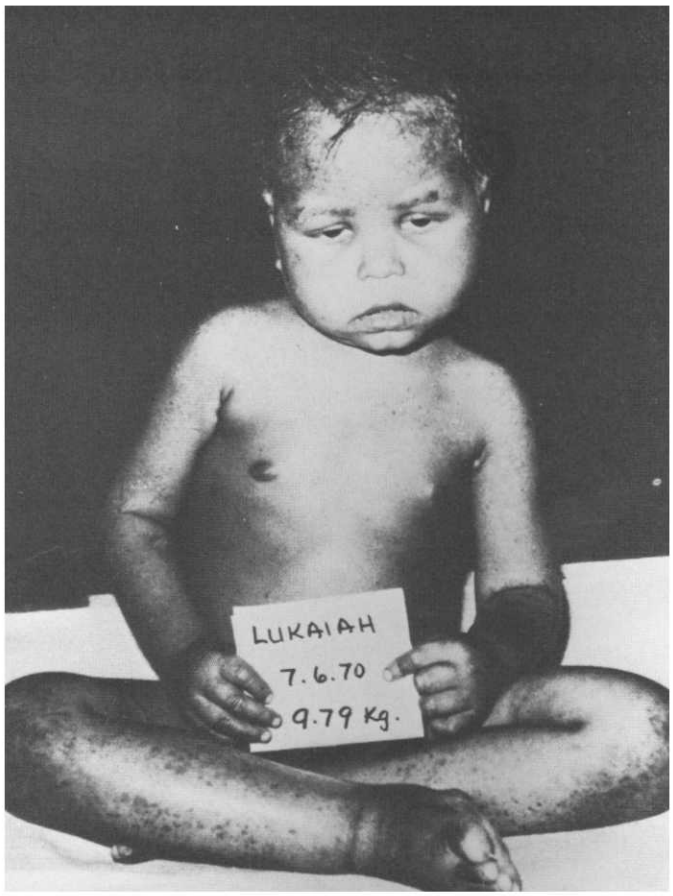
Kwashiorkor (see Fig. 2 and Fig. 3). The main sign is oedema, usually starting at the lower extremities and extending, in more advanced cases, to the arms and face. Oedema may be detected by the production of a definite pit in the pretibial region as a result of moderate pressure for three seconds with the thumb over the lower end of the tibia.

The child may look "fat" so that the parents regard him as well-fed.

Associated signs can be:

- Hair changes: loss of pigmentation, curly hair becomes straight (an African child may appear to have much longer hair), easy pluckability (the hair comes out easily with a very gentle pull).

FIG 2 A SEVERE CASE OF KWASHIORKOR SHOWING OEDEMA AND SKIN AND HAIR CHANGES

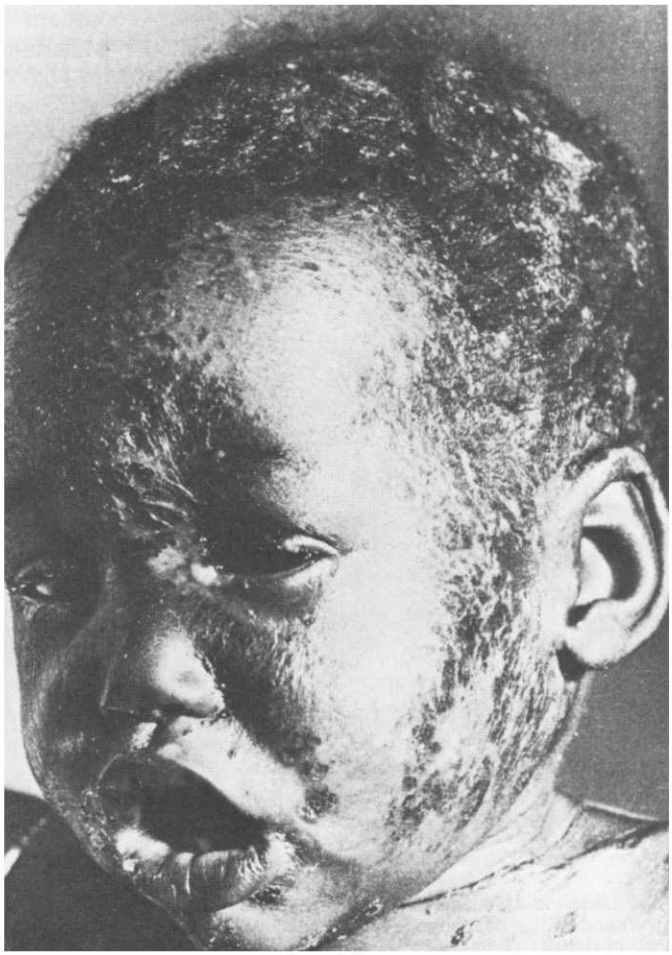


- Skin lesions and depigmentation: dark skin may become lighter in some places, especially in the skin folds; skin may peel off (especially on the legs), and ulceration may occur. The skin lesions may look very like burns.
- Children with kwashiorkor are usually apathetic and miserable and show no signs of hunger. It is difficult to persuade then to eat.

The associated signs of kwashiorkor do not always occur. In some cases oedema may be the only visible sign, in others all the associated signs may be present.

Marasmic kwashiorkor. This is a mixed form with oedema occurring in children who are otherwise marasmic and who may or may not have the other associated signs of kwashiorkor.

FIG 3 A 13-MONTH-OLD UGANDAN BOY ADMITTED TO HOSPITAL FOR THE TREATMENT OF SEVERE KWASHIORKOR



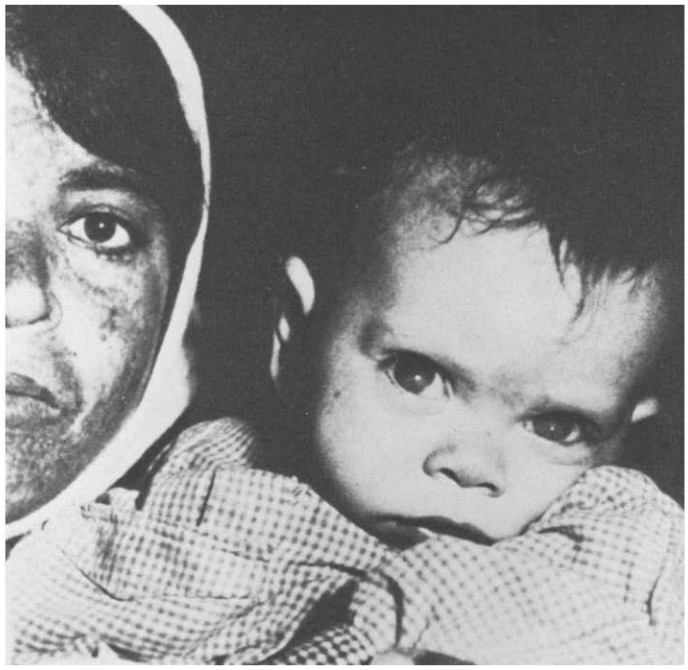
Specific deficencies

While severe PEM is usually the most important health problem during a nutritional emergency, mineral and vitamin deficiencies may also be important. Their treatment is summarized in Table 2.

Anaemia

Nearly all malnourished children are anaemic as a result of iron deficiency and often of folic acid deficiency. Moderate or severe anaemia is diagnosed by pulling down the lower eyelid and looking for pallor of the conjunctiva. The causes are generally multiple (nutritional deficiencies, e.g., of iron and folic acid, malaria, hookworm infestation, etc.). Treatment of moderate forms consists of the daily administration of iron and folic acid for several weeks or months throughout recovery. Supervision of treatment can be difficult under emergency conditions. The daily dose is 100–150 mg of iron with 100 μg of folic acid.[1] Malaria and hook-

FIG. 4. XEROPHTHALMIA IS DIFFICULT TO DETECT AND CHILDREN ARE OFTEN BROUGHT TO HOSPITAL MUCH TOO LATE TO SAVE THEIR EYES



[1] UNICEF tablets specified as containing 0.2 g dried iron sulfate (equivalent to 368 mg of elemental iron) and 250 μg of folate are recommended for routine use—UNIPAC catalogue number 15 500 10 (bottles of 1000 tablets).

worm infestation should be treated and children (and perhaps women) need to receive additional food for faster recovery. Very severe anaemia (extreme pallor, white mucous membranes, difficulty in breathing) should be referred to medical facilities for blood transfusion where possible.

Vitamin A deficiency and xerophthalmia[1]

Vitamin A deficiency is the leading cause of permanent blindness in preschool children. It is almost always associated with some degree of protein-energy malnutrition. Xerophthalmia is the term used to describe the eye signs caused by vitamin A deficiency (see Fig. 4). The daily requirement of retinol rises from 300 μg for an infant to 750 μg for an adult and 1200 μg for a pregnant woman. If the vitamin A is provided by vegetable foods (carotenes) and not by animal products (which includes full cream milk) the amount should be multiplied by six, i.e., range from 1800 μg to 7200 μg (average: about 4000 μg per day per person).

Vitamin A deficiency is most likely to be a problem in areas where the diets of the very poor, even in normal times, do not meet requirements. Since most vitamin A is derived from vegetable sources (green vegetables, most yellow fruits and vegetables, e.g., mangoes, papayas, and carrots, red palm oil, etc.) and these are, in many countries, only seasonally available, there may be a higher incidence of xerophthalmia towards the end of the dry season, when liver stores of the vitamin are depleted.

Since vitamin A is stored in the liver, a sudden deterioration in the diet does not necessarily produce an immediate sharp rise in the incidence of cases, and there may well be a delay of several months until vitamin A deficiency occurs.

Symptoms. One of the first symptoms of vitamin A deficiency is night blindness. Those affected are unable to see at low light intensities e.g., after sunset or inside a hut) when normal individuals can still see reasonably well.

This symptom is difficult to confirm in small children, but there may be a local word for it and mothers may recognize that the night vision of their children is impaired. Indeed, the existence of a local word should be taken as suggesting at least a potential problem.

The following eye lesions are caused by vitamin A deficiency:

— areas on the conjunctiva which become dry, opaque, and dull (xerosis[2])
— accumulation (often triangular in shape) of foamy material on the conjunctiva, usually towards the outer side of the iris (Bitot's spots[2])
— the cornea becomes dry and dull (corneal xerosis), and this may be followed by the most severe signs, namely, clouding, ulceration, and

[1] See also: SOMMER, A. *Field guide to detection and control of xerophthalmia,* Geneva, World Health Organization, 1978.

[2] Slight degrees of xerosis and Bitot's spots may be stained by the application of a small drop of lissamine green or rose bengal 1 % solution with a 10 μl Eppendorf pipette. See: SAUTER, J.J. *Trop. Doc.,* 6. 91-93 (1976).

(even within the space of a few hours) perforation of the cornea, leading to loss of eye contents and permanent blidness. Ulceration and perforation may occur with alarming rapidity, especially in young children who are suffering from measles or some other acute febrile illness.

The presence of any of these signs in even a few children indicates that many more children are at risk.

Prevention. The best way to prevent xerophthalmia is to provide sufficient carotenes or vitamin A in the diet. Note that vitamin A is removed from dried skim milk (DSM) with the fat during processing. Some DSM is supplemented in the factory (e.g., milk provided by UNICEF, WFP, or EEC). This should be indicated on the package and is an important fact to check.

In high-risk areas it may be necessary to administer a high dose of vitamin A periodically to every child, lactating mother, and woman pregnant for more than 6 months. This should be seriously considered if:

— the diet is grossly deficient in vitamin A;

— more than 2% of children under 5 years of age have conjunctival xerosis including Bitot's spots;

— old corneal lesions (scars) are found in one or more children in every 1000.

A single UNICEF soluble capsule containing 110 000 μg of retinol palmitate (200 000 IU of vitamin A) will provide protection for 4–6 months. It should be repeated at an interval of 4 months. Half a dose should be given to children under 1 year. Administration of vitamin A should be recorded on the child's ration card. Overdosage (indicated by headache, vomiting, etc.) is exceptional and may be caused by too frequent (e.g., daily) administration of a high dose.

Vitamin B1 deficiency (beriberi)

The problem of vitamin B1 or thiamine deficiency is less common and is confined to certain areas, e.g., those where the diet is of white polished rice or where people have had to live exclusively on a starchy staple food such as cassava. Several forms exist:

- the "dry" form with neuritis leading to paralysis of the limbs;
- the "wet" form with acute swelling of the body (oedema) and other signs of cardiac failure, leading to sudden death (especially among infants);
- the moderate form, which can be very common, characterized by loss of appetite, malaise, and severe weakness, especially in the legs. These signs may last for many months.

An average intake of approximately 1 mg thiamine daily is sufficient to prevent beriberi; sources are undermilled cereals, legumes, green leaves, etc. Parboiling of rice should be encouraged. Rice for camp use should not be too polished.

Niacin deficiency (pellagra)

Pellagra is characterized by a bilaterally symmetrical skin rash found only on those surfaces of the body exposed to sunlight. It is often marked by severe diarrhoea and mental deterioration.

This deficiency is found mostly among maize- and sorghum-eating populations and is prevented by an average intake of 15–20 mg of niacin per day per person. Sources are legumes and cereals (undermilled).

Vitamin C deficiency (scurvy)

Scurvy is easily recognized: the gums are swollen, particularly between the teeth, and bleed easily. The big joints (knee, hip, etc.) may also appear swollen, although bleeding can take place in any tissue. Haemorrhages on the surface of the bone (subperiosteal) are painful and can cause a pseudo-paralysis in infants. Scurvy can be prevented by providing at least 10 mg daily of ascorbic acid (vitamin C)—i.e., 15 ml of citrus juice, one quarter of an orange, a small tomato, or 20 g of leafy vegetables. If gum swelling does not respond to vitamin C, the cause is not scurvy but poor mouth hygiene.

Vitamin D deficiency (rickets)

Rickets is characterized by deformed, soft bones. The skull has an irregular square form with bossing, while the long bones are bowed with enlarged extremities. Walking is delayed. The best way to prevent rickets is by exposing the unclothed body of the child to sunlight.

Specific deficiencies and nutritional relief

The distribution of multivitamin tablets to the entire population of the affected area is a waste of time and money. The best means of providing vitamins is an adequate diet. Most multivitamin preparations contain only very small quantities of individual vitamins and must be taken at least daily to be of any use. The following approach to the problem of vitamin deficiencies should be adopted:

1. *Identify the deficiencies of public health importance.* For instance, is xerophthalmia a potential problem? Is scurvy or beriberi to be reasonably expected? Such questions are best answered by:

— evaluating the respective intakes of major vitamins in the actual diet,

— initiating a surveillance system (see Chapter 3).

2. Should an obvious dietary deficiency be identified or the presence of typical signs of a specific clinical deficiency be reported and confirmed, *the diet should be corrected by providing foods rich in the missing vitamins and/or minerals.*

3. Should this be impossible or insufficient, mass administration of the *specific* vitamin is indicated. It should be given in adequate quantities.

TABLE 2 CURATIVE TREATMENT OF SPECIFIC DEFICIENCIES (IN BRIEF)

What	When	How much and how long
Moderate to severe anaemia	● marked pallor of conjunctiva	100–200 mg of iron with 100 μg of folic acid daily in 2 or 3 divided doses several weeks or months until recovery
Xerophthalmia	● night blindness ● any ocular sign of vitamin A deficiency ● severe PEM	intramuscular injection of 55 000 μg water-miscible retinol palmitate (100 000 IU of vitamin A) followed the next day by oral administration of 110 000 μg (200 000 IU of vitamin A, adequate protein intake is essential
Beriberi	● any suspicion of vitamin B 1 deficiency	50 mg of thiamine followed by 10 mg daily until recovery
Pellagra	● any suspicion of niacin deficiency	300 mg of niacin per mouth daily until recovery (usually a few days in acute cases)
Scurvy	● any haemorrhagic symptom in a malnourished child	500 mg or more ascorbic acid daily until recovery
Rickets	● any suspect bone deformation in young child	no more than 100 000–300 000 IU (2 5–7 5 mg) of colecalciferol (vitamin D) in a single dose or 1 000 IU (25 μg) daily for 10–30 days; [a] exposure of the skin to sunlight.

[a] An overdose of colecalciferol is dangerous.