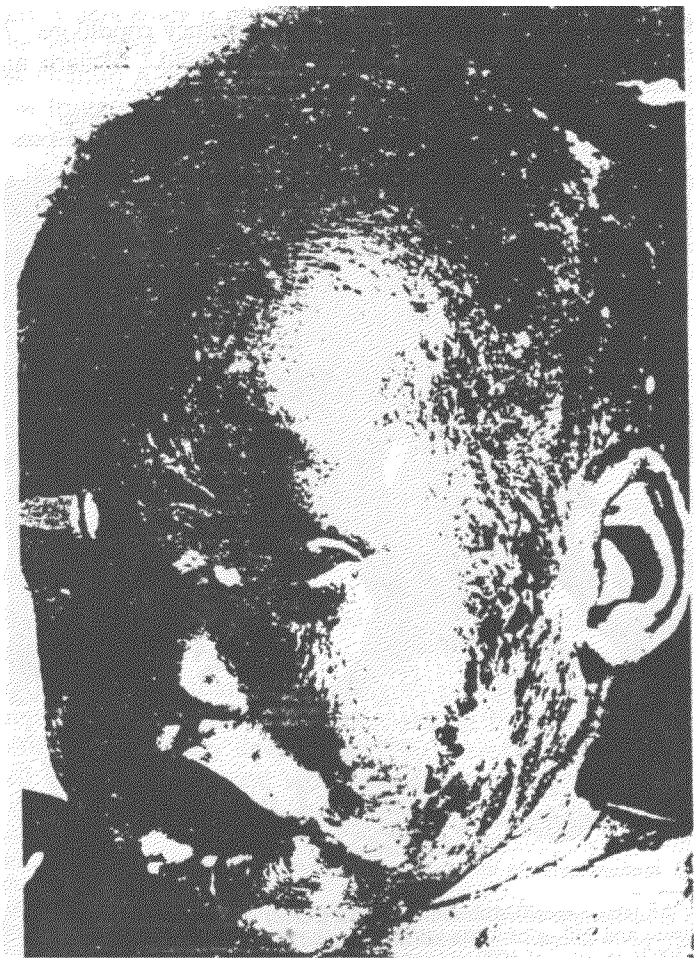
- Skin lesions and depigmentation: dark skin may become lighter in some places, especially in the skin folds; skin may peel off (especially on the legs), and ulceration may occur. The skin lesions may look very like burns.
- Children with kwashiorkor are usually apathetic and miserable and show no signs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



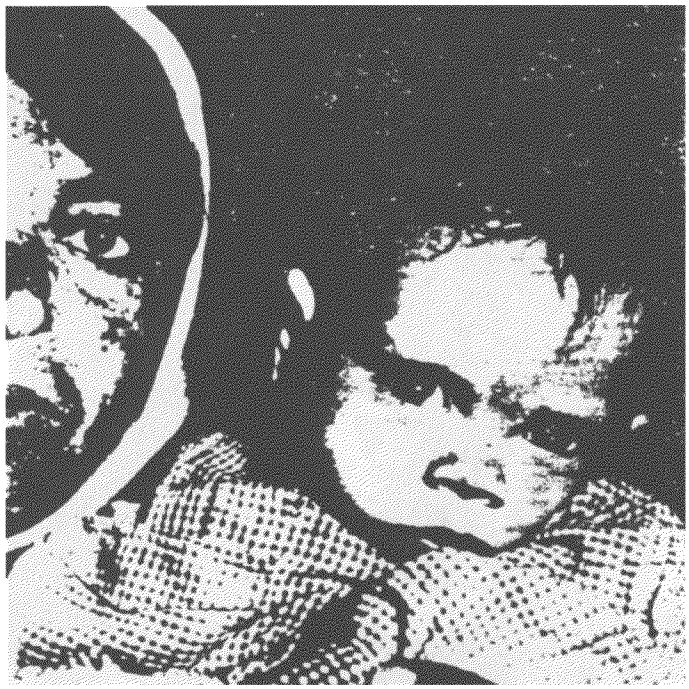
Specific deficencies

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 15500.10 (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

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[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