PART 3:

DIAGNOSING, MEASURING AND TREATING HEALTH NEEDS THROUGH A SUPPLEMENTARY FEEDING PROGRAM

LESSON 3

CAUSES OF MALNUTRITION

Malnutrition is one of the most important problems facing refugees. Malnutrition decreases resistance to infection causing greater rates of sickness and death and stunts height. It also lowers working capacity, and hence productivity.

Malnutrition occurs when the diet supplies insufficient nutrients to cover the body's requirements. It therefore is most common among young children and pregnant and nursing women whose needs are relatively high.

Major nutritional 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 most common form of malnutrition in nutritional emergencies.

- Kwashiorkor is characterized by edema, usually starting at the lower extremities.

- Marasmic kwashiorkor is a combination of wasting and edema. The treatment of severe forms of PEM is presented in Lesson 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: beri-beri, pellagra, scurvy, and rickets.

- Mineral and vitamin deficiencies must be identified and the affected individuals or those 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 or nutrient losses caused by infection.

Chronic PEM has many short-term and long-term physical and mental effects, including growth retardation. A malnourished child is often 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 such as rapid weight-loss which affect a significant number of older children, adolescents, and adults, as well as young children.

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 of PEM can occur rapidly.

Severe forms of PEM include: nutritional marasmus, kwashiorkor, and maramsmic kwashiorkor.

Nutritional marasmus results from prolonged starvation (see Fig. 3-1).

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

Associated signs can be:

- A thin "old man" face.

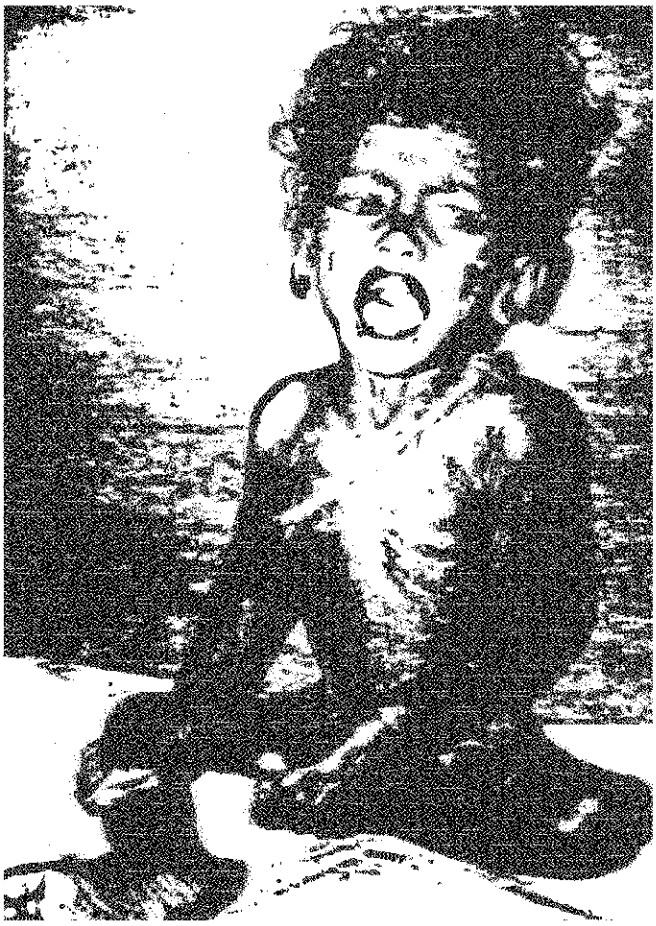
- "Baggy pants" (the loose skin of a child's buttocks hanging down).

- The children concerned are usually active and may appear to be very alert in spite of their condition.

- There is no edema detectable when pressure is applied to the lower extremities.

Figure 3-1

Child suffering from nutritional marasmus



Kwashiorkor (see Fig. 3-2 and Fig. 3-3). The main sign is edema, usually starting at the lower extremities and extending, in more advanced cases, to the arms and face. Edema may be detected by the production of a definite pit in the pretibial region as a result of the application 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).

- Skin lesions and depigmentation: skin may peel off (especially on the legs), and ulceration may occur. The skin lesions may look 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 edema may be the only visible sign; in others all the associated signs may be present.

Figure 3-2

A severe case of kwashiorkor showing edema and skin and hair changes

Figure 3-3

A 13-month old boy with severe kwashiorkor

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

Specific deficiencies

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 3-A.

Anemia

Nearly all malnourished children are anemic as a result of iron deficiency and folic acid deficiency. Moderate or severe anemia 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 ug of folic acid.2 Malaria and hookworm infestation should be treated and children (and perhaps women) need to receive additional food for faster recovery. Very severe anemia (extreme pallor, white mucous membranes, difficulty in breathing) should be referred to medical facilities for blood transfusion where possible.

Anemia in pregnancy is associated with low birth weight and increased infant death. Work capacity is reduced in anemic adults and is an important cause of low productivity in some countries.

SFPs can help to control anemia in participants if the rations include foods rich in iron (liver, meat, fish, green leaves, blended foods, soy fortified cereals) as well as some meat, fish or soybean, and vitamin C-rich foods to improve total iron absorption (p __). Nutrition education can improve the situation in most cases. A complementary activity in some SFPs is the giving of iron supplements and/or anti-parasitic treatment to vulnerable groups. Breast, rather than artificial, feeding will help prevent anemia in infants.

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

Vitamin A deficiency and xerophthalmia [3]

Vitamin A deficiency is the leading cause of permanent blindness in young children. It is common among refugees in many developing countries who have some degree of protein-energy malnutrition. A serious deficiency of vitamin A causes a disease of the eye called xerophthalmia (see Fig. 3-4). If the disease reaches the final stages the lens of the eye softens (keratomalacia) and blindness occurs. Xerophthalmia usually occurs in children with PEM and the death rate among such children is very high.

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. The daily requirement of retinol rises from 300 ug for an infant to 750 ug for an adult and 1200 ug for a pregnant woman. If the vitamin A is provided by vegetable foods (carotenes) and not by animal products (such as full cream milk) the amount should be multiplied by six, i.e., range from 1800 ug to 7200 ug (average: about 4000 ug per day per person). 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

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3 See also: SOMMER, A. "Field guide to detection and control of xerophthalmia, Geneva, World Health Organization, 1978.

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 signs are caused by vitamin A deficiency:

- areas on the conjunctiva become dry, opaque, and dull (xerosis 4)

- accumulation (often triangular in shape) of foamy material on the conjunctiva, usually towards the outer side of the iris (Bitot's spots 4)

- the cornea becomes dry and dull (corneal xerosis), and this may be followed by severe signs of clouding, ulceration, and (even within the space of a few hours) perforation of the cornea. Ulceration and perforation may occur with alarming rapidity, especially in young children who are suffering from measles or some other acute febrile illness. This can lead to loss of eye contents and permanent blindness.

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

4 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 ul Eppendorf pipette. See: SAUTER, J.J. "Trop.Doc.," 6:91-93 (1976).

Eye signs that may be recognized by non-health staff [5] which indicate that the child may have xerophthalmia are:

- inability to see well at night (night blindness)
- dislike of bright light (photophobia)
- grey/white foamy patches on the white surface of they eye (Bitot's spots)
- surface of eye looks dry.

All SFP staff must be on the look-out for any of these signs and should refer any affected child immediately for treatment. The disease can progress very rapidly especially if the child is being fed unfortified DSM.

Prevention. The best way to prevent xerophthalmia is to provide sufficient carotenes or vitamin A in the diet. SFP rations and meals should contain a source of vitamin A in areas where vitamin A is deficient. Good inexpensive sources are dark green leaves, yellow and orange fruits and vegetables and red palm oil. Rich but expensive sources are liver and egg yolk. Donated foods rich in the vitamin are yellow maize, WSB, CSM, dried egg, dried apricots, DWM, fortified DSM and cheese.

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5 Health workers should be able to recognize conjunctival and conjunctival and corneal xerosis which positively indicate xerophthalmia.

Vitamin A is removed from dried skim milk (DSM) with the fat during processing. Some DSM is fortified or supplemented with vitamin A 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 women 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 ug 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.

Figure 3-4

Xerophthalmia is difficult to detect and children are often brought to a hospital much too late to save their eyes

Vitamin B1 deficiency (beri-beri)

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 (edema) 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 beri-beri; 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 diarrhea 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 (scurvey)

Scurvy is easily recognized by swollen gums, particularly between the teeth, that bleed easily. The big joints (knee, hip, etc.) may also appear swollen, although bleeding can take place in any tissue. Hemorrhages on t 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 beri-beri to be reasonably expected? Such questions are best answered by:

- evaluating the intake of major vitamins in the actual diet,
- initiating a surveillance system (see Lesson 4).

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 3-A

Curative treatment of specific deficienciencies

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 66 000 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 B1 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 calecalciferol (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 calecalciferol is dangerous.