Chapter 7 Protein-Energy Malnutrition

Protein-energy malnutrition (PEM) is currently the most widespread and serious health problem of children in the world. At any time approximately 100 million children suffer from the moderate or severe forms of PEM. In the 1990s, the number of underweight children in developing countries declined from 177 million to 149 million. The prevalence of severe malnutrition also declined, but even then severe malnutrition remains an important problem. In any one country the prevalence rates will be influenced by the season, the availability of food, the incidence of infection, and the state of development of the health services. As can be expected, the peak incidence is immediately after epidemics of infectious illnesses and diarrhoea or in the "hungry' months. Results of community surveys in the past 10 years in 17 different countries and involving 173 000 children reveal an aggregate prevalence rate of 20 per cent (Table 7.1).

Table 7.1 Prevalence of childhood malnutrition

Area	No. of surveys	No. of children examined (thousands)	Severe forms Range (%)	Severe forms Median (%)	Moderate forms Range (%)	Moderate forms Median (%)
Latin America	1 1	109	0.5- 6.3	1.6	3.5-32.0	18.9
Africa	7	25	1.7- 9.8	4.4	5.4-44.9	26.5
Asia	7	39	1.1-20.0	3.2	16.0-46.4	31.2
Total	25	1 73	0.5-20.0	2.6	3.5-46.4	18.9

Taking the median values, an approximate estimation of the geographical distribution of childhood malnutrition can be made in Table 7.2 and Fig. 7.1.

Table 7.2 Geographical distribution of childhood malnutrition

Area	Population aged 0-5years (millions)	No. of children with PEM (millions)	
		Severe	Moderate
South America	46	0.7	8.8
Africa	61	2.7	16.3
Asia	206	6.6	64.4
Total	313	10.0	89.5

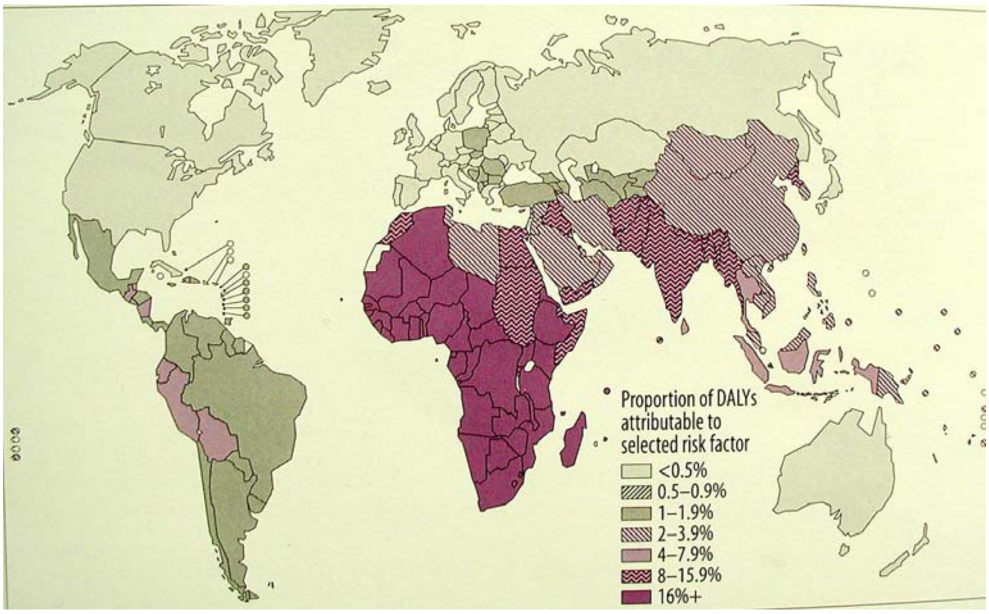


Figure 7.1 Distribution of Malnutrition by Regions

The data in Table 7.2 provide only an estimate of the size of the problem. Exact statistics will not become available until health services of the developing countries are able to achieve universal coverage of the population and efficient methods for the collection of health data are established. At present only a small proportion of rural populations receive health care on a regular basis, and health coverage of the urban poor is even less in some countries. Hence most health statistics are rudimentary. Thus community surveys at regular intervals provide the only means of measuring the size of the problem. As a result of such surveys it is possible to generalize that at any time 10 per cent of children in an average rural community in the developing world will show signs of growth failure and some of them will have clinical signs of malnutrition. Only 24 per cent of children in such communities show adequate growth, and the remaining 66 per cent experience faltering of growth from time to time.

The level of preschool (1-5 years) mortality in a country may also indicate the prevalence of childhood malnutrition. This is because of the well-known synergism between undernutrition and infection. The preschool mortality in the average developing country is about 40 times that of western countries, which again reveals the size of the problem of malnutrition. In a study of the patterns of childhood mortality in 13 areas in South America, sponsored by the Pan American Health Organization, 7318 deaths in children between the ages of 1 and 4 years were studied. Malnutrition was found to be the primary cause of death in 9 per cent (range 0-18 per cent) and an associated cause of death in 48.4 per cent (range 0-61 per cent). The general conclusion was that malnutrition was directly or indirectly responsible for 57.4 per cent of deaths of children aged 1-4 years.

CLASSIFICATION AND DEFINITION

It is paradoxical that such a widespread, serious and extensively studied form of nutritional disorder still continues to be a controversial subject in almost every aspect. This is especially so with regard to classification and pathogenesis. The reason is that the presenting features of nutritional deficiency vary from one part of the world to another, due mainly to the great variation in the nutrient content of the diet, the prevalence of antecedent illnesses, the variability of the host, and the time over which the causative factors operate. Two distinct clinical syndromes have been described, viz. kwashiorkor and marasmus, and represent the severe forms of PEM. They occupy the two ends of a spectrum with a mixture of the clinical features of both in between. Biochemical features also form a spectrum though they are more evident in kwashiorkor than in marasmus. It is not unusual to find that a child diagnosed as suffering from kwashiorkor shows the typical features of marasmus after the oedema (see "Clinical features') subsides, while a child with nutritional marasmus often develops oedema and progresses to marasmic kwashiorkor.

Of the two classical syndromes, kwashiorkor has received a great deal of interest and attention because of its striking clinical features and extensive changes in the body's chemistry. However, there are now clear indications that marasmus is on the increase, especially in the city slums and shanty towns of the developing countries. The rapid decline in breast feeding has a great deal to do with this. Moreover, since marasmus usually occurs at a younger age than kwashiorkor its long-term effects are more severe. I n both forms of malnutrition, recognition at an early stage is important in order to avoid the serious after-effects of established malnutrition. Hence there has been great interest in accurate classification and especially in identifying early signs.

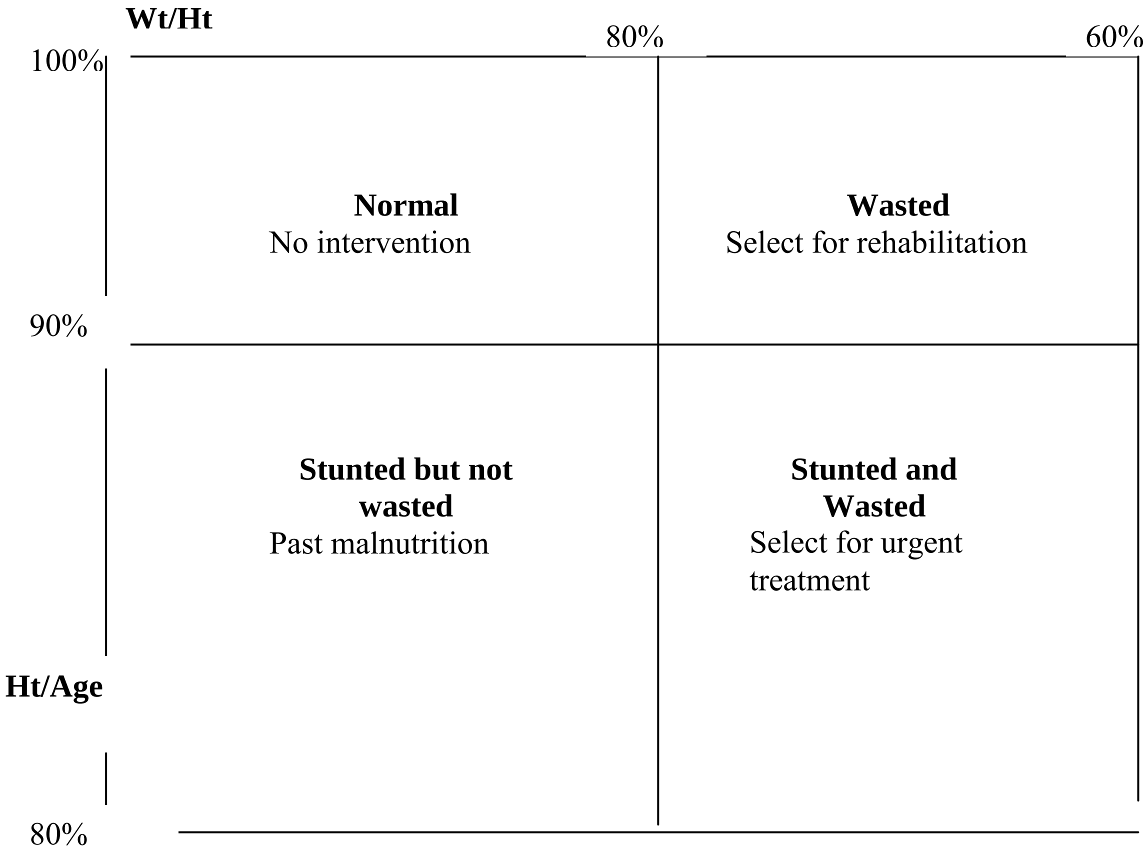
Mild-to-moderate malnutrition

In defining the stages of malnutrition, two processes have to be taken into account. These are: (1) the period over which malnutrition occurs, so as to decide whether it is acute or chronic, or acute on chronic. Acute forms chiefly affect body weight more than height, whereas in the chronic form both height and weight are affected. (2) The aetiological factors. The classical explanation that kwashiorkor is due to protein deficiency with relatively adequate energy supply while marasmus is due to the overall deficiency of proteins and calories arose out of the observations that in countries where roots, tubers and plantain (all with 1-2 per cent protein) form the staple foods kwashiorkor is more common. This view has been challenged, as we saw in the previous chapter, and the role of infection has come to be emphasized in the aetiology as well as treatment of severe malnutrition.

In every locality the identification of the important aetiological factors is necessary for instituting early intervention. When food is inadequate, the organism adapts first of all by reducing growth and the clinical signs are those of such adaptation. Thus, weight gain slows down, and so weight for age has been commonly used to assess the degree of mild to moderate malnutrition. Those children who weigh less than the mean weights of children in their age group are thus called "*wasted*' and the degree of wasting is an indication of the degree of malnutrition they have suffered. A common difficulty is that in most cases parents do not know the ages of their children. Height for age also suffers from the same difficulty. Thus there is a need for age-independent criteria of malnutrition. It has been suggested that the ratio weight/ height may overcome this difficulty besides providing a sensitive measure of wasting. Refinements have been added to this measure in the form of regression lines and various indices like $weight/height^{2}$ or $weight/ height^{1.6}$. One may question the value of these complexities which require mathematical manipulation.

Reduced growth as a consequence of adaptation to lack of food also affects height. Weight can swing up and down, but obviously this is not the case with height. All that happens is that growth in height slows down and the individual will end up short. Those children whose heights are less than the mean heights of children in their age group are called 'stunted'. Catch-up growth in both height and weight can occur if the slowing of growth was temporary, as for example after an acute illness. If dietary deficiency is prolonged, full catch-up does not occur and the deficit in height becomes fixed and permanent. Thus deficits of height indicate long-standing malnutrition. Children who grow slowly in an adverse environment and retain normal weight for height may be adapting to the environment compared to those who are wasted or have developed oedema.

Deficits in weight for height and in height for age can be put together in a 2x2 table for community surveys or for triage in emergencies. This manoeuvre helps to identify four groups of children as follows:



Besides growth in height and weight, the body compartments most affected in malnutrition are those of energy reserve - subcutaneous fat and the protein store of skeletal muscle. Measuring these two body compartments can shed extra light on the pathophysiological mechanisms. Thus, in malnutrition muscle is wasted not only because of lack of protein in the diet, but because muscle is used up to supply energy. Deficient muscle with adequate body fat will be one indication of protein deficiency. Conversely, adequate muscle with lack of fat suggests lack of energy reserve.

The adaptive changes to dietary deficiency are not always successful. For example, we know that undernourished individuals have a predisposition to infectious illnesses. Diarrhoeal disease is not only more prevalent in undernourished children, but also tends to be more severe. In one study of village children aged between 6 and 32 months in Nigeria it was found that the frequency of diarrhoea during the rainy season was greatest amongst children who were described as "wasted" (i.e. < 80 per cent of weight for height). Such children suffered 47 per cent more episodes of diarrhoea. Pre-existing malnutrition affected the duration of diarrhoea which was 79 per cent longer in "wasted' children compared to well-nourished controls. In children who were under-weight (< 75 per cent weight for age) the duration of diarrhoea was 33 per cent longer and in stunted (< 90 per cent height for age) children it was 37 per cent longer compared to those who were well-nourished. In Zaire, childhood infections, especially measles, had occurred in more than half the children in the weeks immediately preceding an outbreak of kwashiorkor. Associated deficiencies besides those of protein and calories also occur with malnutrition. Hence in identifying early malnutrition it is useful to remember that the differing proportions of protein and calories in the diet, the duration of malnutrition, the associated deficiency of other nutrients, and the effects of infectious illnesses together give rise to a wide spectrum of signs and symptoms. The presence of intestinal parasites may also contribute to the typical pattern of malnutrition encountered in a given locality. For example, heavy hookworm infection may contribute to loss of iron as well as protein in the gut;

Strongyloides infection also acts in a similar manner with added malabsorption, and giardiasis causes impaired absorption. In recent years the epidemic of HIV / AIDS has resulted in a harrowing picture of severe malnutrition associated with gastro-intestinal infection with cryptosporidium.

A further twist has been added to a complex situation by the trend of urban migration and rapid urban growth in most developing countries. The new social problems of unemployment, the need to change from subsistence to cash economy and a deteriorating family life, added to the promotion of a variety of "junk' foods and beverages, means that multiple deficiencies occur on a background of protein and energy lack.

The Need for Simplification

Such a complex situation has led to a variety of methods of classification. Such methods, however useful as research tools, cannot be easily taught to auxiliaries and village health workers, for whom a more simplified and action-oriented approach is necessary. With this need in mind, several simple methods of measuring nutritional status have been evolved. The Wellcome classification is one such simple method for the diagnosis of clinical malnutrition.

Wellcome classification

Weight (% of standard)	**Oedema** Present	Absent
80-60	Kwashiorkor	Undernourished
< 60	Marasmic kwashiorkor	Marasmus

For children within the community who are at risk of mild-moderate malnutrition a similar simple tool for the selection of early cases is required. The Gomez classification was first suggested in the late 1950s as a method of diagnosing mild-moderate forms of malnutrition in the community and for the early identification of marasmus. The classification is based on weights of healthy American children under the age of 5 years, and the fiftieth percentile is taken as the standard. Malnutrition is graded into three degrees of increasing severity according to the percentage reduction in weight from the standard.

Gomez Classification

First-degree malnutrition	< 80% of the standard
Second-degree malnutrition	< 70% of the standard
Third-degree malnutrition	< 60% of the standard

The Gomez classification has been criticized on two counts. Firstly, it does not take height into consideration. Secondly, in some communities more than half the children fall in the category of third-degree malnutrition, and health workers doubted whether the growth standards of one community were applicable to another. With further experience it is now realized that the place on the growth chart where a child's weight falls is not so important as the shape of his growth curve compared to the standard. This knowledge has helped to remove a great deal of controversy and contributed to the spread of weight charts in most countries of the Third World. When Morley described the first weight chart in Nigeria it carried two curves. The upper curve represented mean weights of children from the upper social class and the lower curve did the same for the lower social class. Since then there has been considerable debate with regard to local standards, definition of "normal, children and so on. This controversy delayed the development of local weight charts. When it was realized that it was the shape of the child's weight curve as compared to normal which was important, and not the actual weight, the use of weight charts based on the Harvard standards received a great boost. More recently a working party convened by the World Health Organization has developed a weight chart for international use (figure 7.2) based on American data. In these charts the upper lines represent the 97th and 50th percentiles respectively. The lower lines are 3rd percentile, -3 standard deviation and -4 standard deviation respectively. Thus, further development in the concept first proposed by Gomez and colleagues has facilitated the regular use of weight charts in children's clinics in many countries.

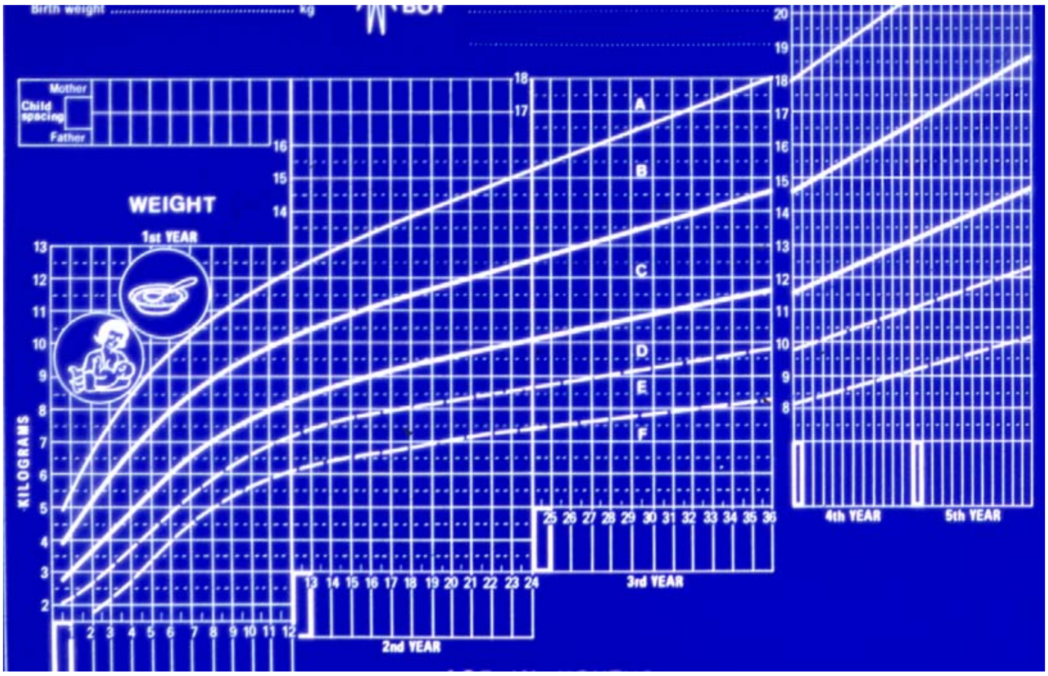


Figure 7.2 Weight Chart for International Use

Present experience shows that it is possible to train auxiliaries and even lesser-trained health personnel in the charting and interpretation of growth records. In the present state of development of health services even these are not available in all areas and the new trend is to train part-time village health workers who are not always literate. A yet simpler way of assessing nutritional status of children is therefore required. Circumference of the mid-arm as an indicator of muscle mass has been used as one of the parameters for measuring nutritional status. It is known that in the normal child between the ages of 1 and 5 years the arm circumference changes very little. Here then is a parameter which is age-independent. (Fig.7.3).

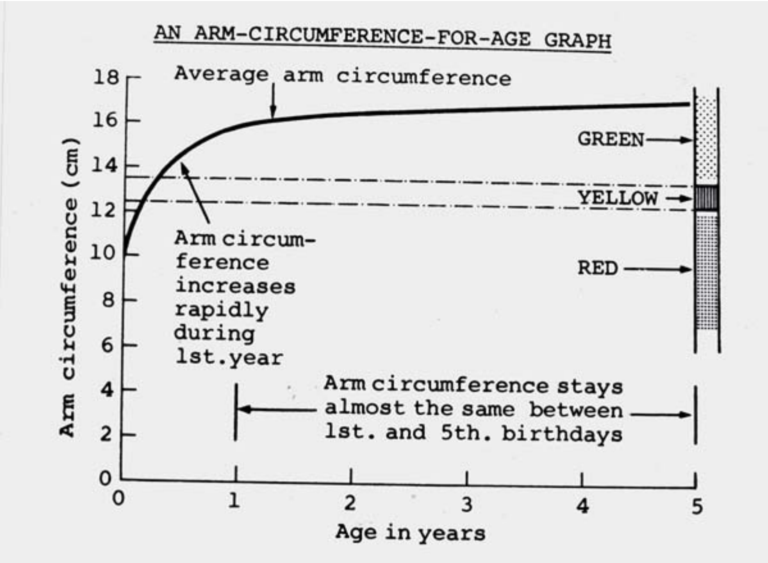


Figure 7.3 The mid-arm circumference as a measure of nutritional status

The first practical use of this concept was made during the Biafran war for selecting malnourished individuals in the refugee camps for intensive rehabilitation. The arm circumference was compared with the height of the individual and grades of malnutrition were identified in accordance with the percentage reduction in the arm circumference. A height measuring stick is marked off in arm circumference measurements (80% and 85% of normal) against height. The method came to be known as the *Quac Stick.* It compares mid-arm circumference with the height of the child. (See fig. 7.4)

Figure 7.4 Quac Stick in use in Biafra

More recently, Shakir has shown in Baghdad children that measurement of the arm circumference was a useful tool for diagnosing malnutrition. Children whose arm circumference was less than 75 per cent of the standard also had a body weight less than 60 per cent of the Harvard standard in nine cases out of ten in his series. The practical value of this observation is that primary-school children and illiterate village health workers can be trained to use a string or a strip of plastic with a mark and colours in green (over 14.0 cm), yellow (12.5-14.0 cm) and red (less than 12.5 cm) for assessing malnutrition in village children (figure 7.5)

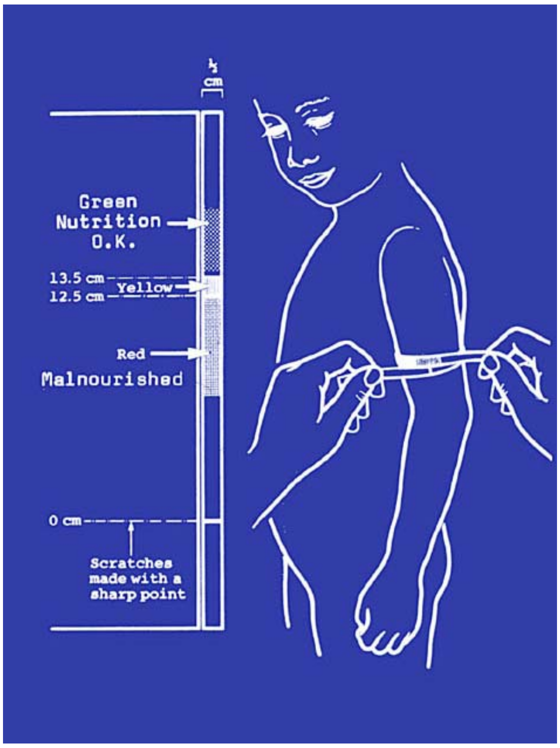


Figure 7.5 Measuring mid-arm circumference

Experience in several countries has shown the practical usefulness (Figure 7.4). of the above methods of measuring less severe forms of malnutrition. In the Narangwal study conducted in fourteen villages in the Punjab, North India, 3000 children aged 1-36 months were observed regularly for several years. It was found that taking weight for age as the parameter according to the Gomez classification, the risk of death for a child between 70 and 80 per cent of the Harvard mean was more than For a child between 60 and 70 per cent it was more than 10 times. When annual mortality rates were computed for each nutritional level it was found that an approximate doubling of mortality occurred with each 10 per cent drop in nutritional status. (See fig. 7.6).

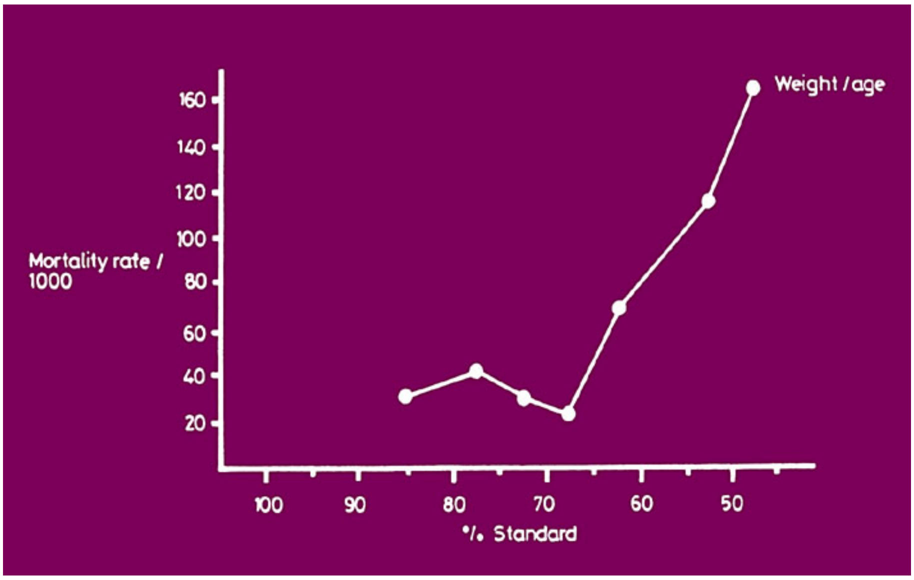


Figure 7.6 Mortality by age for weight

A similar study in rural Bangladesh assessed the risk of mortality in 2019 children aged 13-23 months over a period of 2 years. Severely malnourished children according to all the common indices like weight/age, weight/height, height/age, arm circumference/age and arm circumference/height experienced substantially higher (3- to 7-fold) mortality rates. Even though all indices were found to discriminate mortality risks weight for age and arm circumference for age were the strongest and weight/height the weakest. For each index, a threshold of risk was noted instead of a gradation. (See Fig. 7.7)

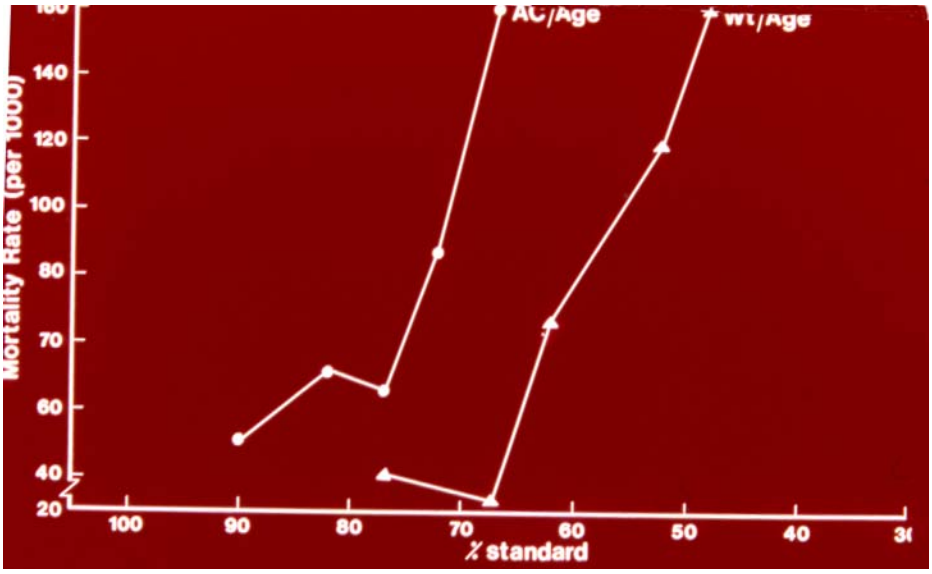


Figure 7.7 Mortality rates by arm circumference for age and weight for age

Field studies in several countries have shown that there is a range of growth status over which the risk of dying changes only slightly until a lower threshold below which mortality risks rise steeply. Severe malnutrition invariably carries life threatening risk. In moderate malnutrition mortality risk is less clear. This is true for all the measures of malnutrition – weight/height; height/age; and arm circumference. The relationship between any given measure and mortality risk is non-linear, and there is a distinct threshold effect.

In conclusion, whatever the anthropometric criterion used for diagnosing sub-optimal nutrition the relationship between mortality risks and nutritional status stands out. When mortality risks are plotted against nutritional status the risks are high for severe forms of malnutrition and relatively low for the mild-moderate forms of malnutrition. An impression is created that mild/moderate malnutrition does not pose much risk and in many countries governments stressed severe forms of anthropomorphic deficits in their national plans. However, when the same data are plotted on a log scale taking logarithms of mortality risks against severity of malnutrition straight line slopes are obtained. This observation indicates that risks increase exponentially with severity of malnutrition and not just linearly as previously thought. When this approach was applied to data from 53 developing countries it could be shown that 56% of child deaths overall were attributable to malnutrition's potentiating effect, with three-quarters arising from mild-moderate malnutrition.

In the case of the newborn, birth weight is a useful indicator of mortality risks in the neonatal period and the first half of infancy. In the first 3 years of life, weight for age can effectively identify those children who are at risk of death. After the age of 1 year and up to the age of 5, the arm circumference is a useful quick technique for identifying those at risk. Children who are both wasted and stunted face greatly increased danger and should be carefully supervised.

CLINICAL FEATURES

In the early stage of malnutrition clinical signs are few and even absent, and diagnosis requires both biochemical tests and anthropometric measurements.

In the severe forms, growth failure is obvious. In addition, activity is reduced so that the child is listless and apathetic or irritable. Because of this irritability communication between the child and the parents is minimal, and often resentment builds up. There is also discoloration of hair and skin, anaemia of varying severity, signs of associated deficiencies and presence of infection.

Kwashiorkor

Kwashiorkor (See figure 7.8) presents with failure to thrive, oedema, apathy, anorexia, diarrhoea and discoloration of the skin and hair.

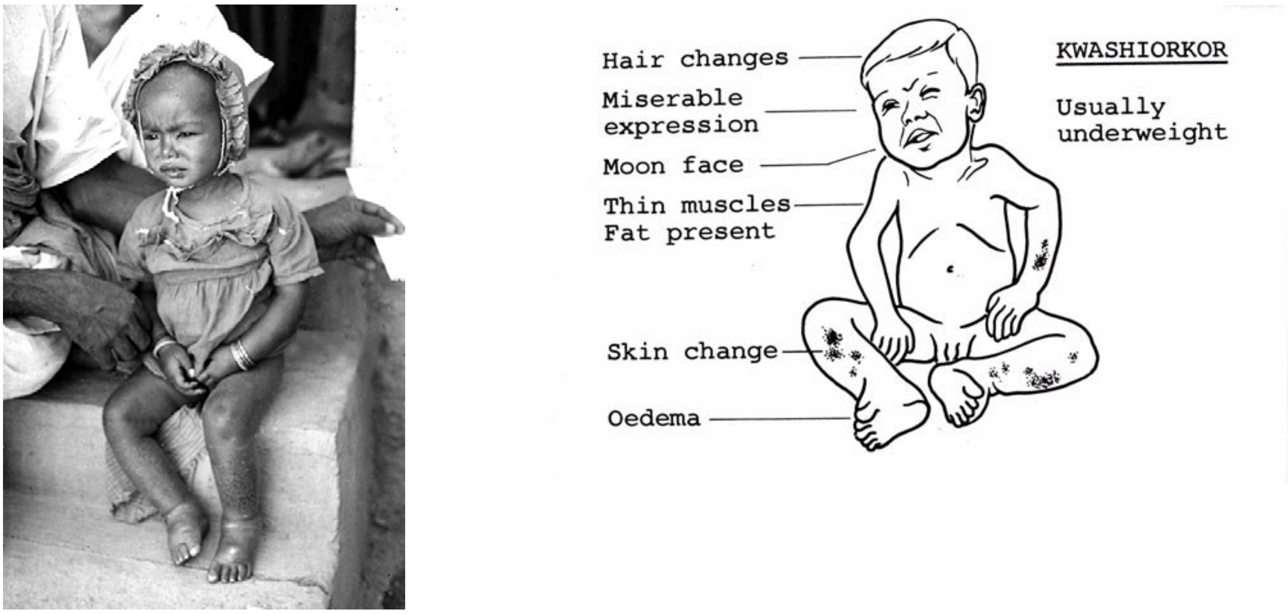


Figure 7.8 Kwashiorkor

The general appearance may be that of typical "sugar baby', with chubby features and bloated body, so that at the time parents may think the child is doing well, and they cannot be convinced that he is malnourished.

Failure in growth is marked and weight is reduced in spite of the presence of oedema. Varying degrees of muscle wasting are present. The discoloration of hair and skin gives the child a characteristic "red baby' appearance. In addition, various forms of skin disorder can also occur. These can vary from the characteristic "flaky paint' dermatoses to fissures and at times raw ulcerating areas chiefly at the flexures and the buttocks.

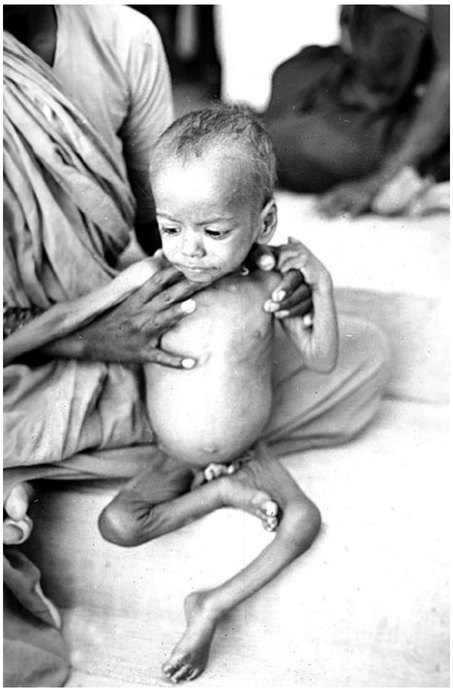
Oedema is the characteristic clinical sign of kwashiorkor. It appears first on the dorsum of the feet and ankles and spreads upwards to involve the rest of the body. Oedema fluid can represent 5-20 per cent of body weight so that change in the appearance of the child when the fluid is lost can be striking.

Another major characteristic is the change in personality. Most children with kwashiorkor are apathetic or extremely irritable and miserable. Marked improvement in temperament occurs on treatment, and many clinicians stress that the return of the smile is the first sign of improvement.

Physiological functions of the various systems are markedly disturbed, with diarrhoea, electrolyte disturbance, circulatory insufficiency, metabolic imbalance and poor renal function. Hence the child with kwashiorkor should be thought of as an emergency in need of intensive medical and nursing care, and not just simply malnourished.

Marasmus (See figure 7.9)

This usually occurs in younger children, with failure to thrive. Affected children are short and light for their age. In appearance they are shrunken and wizened due to lack of subcutaneous fat. Until recently kwashiorkor had aroused maximum interest and attention, but it is now increasingly realized that marasmus is a fast-growing disease of the large urban slums and shanty towns in the cities of the Third World. The sharp increase in bottle feeding amongst the urban poor and the new migrants to the cities is largely responsible for the increase in the incidence of marasmus. Since the slums and shanty towns are also the "septic fringes' of the cities, the marasmic child commonly suffers from infections of all sorts, though more commonly respiratory and diarrhoeal illnesses.



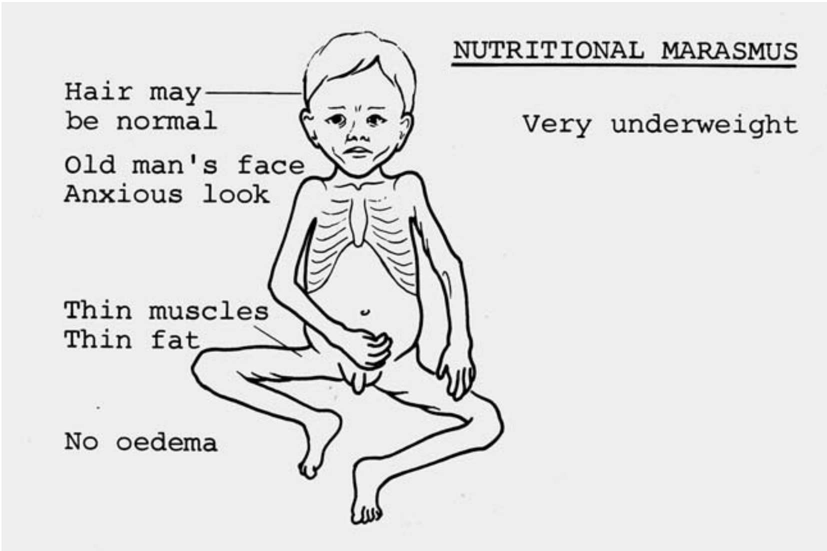


Figure 7.9 Marasmus

ASSOCIATED DEFICIENCIES

Nutritional deficiency is very rarely restricted to just one or two nutrients. As a rule the deficiency is generalized so that, besides clinical signs of protein and calorie deficiency, there also exist signs of vitamin and other deficiencies.

Many of the illnesses which precipitate protein-calorie malnutrition also provoke loss of nutrients from the body in the same way as they cause a negative nitrogen balance. The type of local food staple, the age of the child, and the time over which the child's diet has been insufficient also help to determine the severity and the nature of associated deficiencies, the most common of which are those of the fat-soluble and water-soluble vitamins and of iron.

Xerophthalmia (See fig. 7.10)

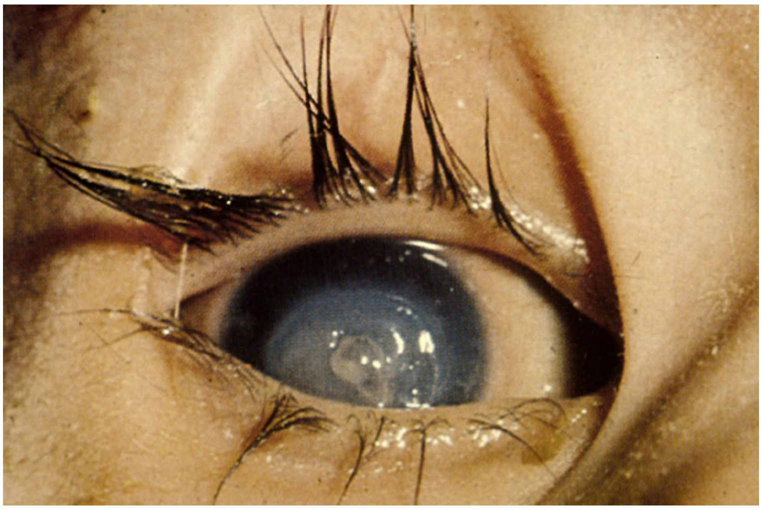


Figure 7.10 Xerophthalmia with keratomalacia

In the rice-eating countries of South East Asia, deficiency of vitamin A is endemic and is commonly associated with protein-energy malnutrition. In Indonesia about three-quarters of all cases of kwashiorkor are reported to also have xerophthalmia. In Thailand the incidence is 40 per cent, but in East and West Africa and in the West Indies the reported incidence is only about 1 per cent. Clinical deficiency is only the tip of the iceberg, because in endemic areas children with PEM but no clinical eye signs invariably have low levels of vitamin A and depleted liver stores of the vitamin. Presence of eye lesions therefore indicates a long-standing deficiency and is often a danger signal, since the mortality from PEM in such cases is about four times that of children who have no ocular lesions.

Rickets (See Fig. 7.11)

Figure 7.11 Rickets in malnutrition

Rickets is a common finding in cases of PEM from the urban slums and inner-city areas. It is more common in the younger child suffering from marasmus than in the older child suffering from kwashiorkor.
Rickets, like xerophthalmia, has been more commonly reported from South East Asia, where prevalence rates of 15- 18 per cent have been recorded. It is a rare finding in East and West Africa and in the West Indies. In endemic areas malnourished children without clinical rickets have low blood levels of the active form of vitamin D.

Vitamin B deficiency (See fig. 7.12)

Figure 7.12Angular stomatitis of Vitamin B deficiency

Laboratory tests show that children

with PEM have depleted stores of the water-soluble vitamins, chiefly those of the B group. In many cases there are visible manifestations of deficiency disease. The effects of protein deficiency on the mucosal lining of the mouth, skin and gastrointestinal tract may alter the classic manifestations of B-complex deficiency. Many vitamins act as co-enzymes in several key metabolic reactions in the cell. Their deficiency, together with the deficiency of protein and calories, can seriously disrupt cell function. Supplementation with vitamins during treatment is essential in order to replenish tissue stores and to ensure optimal function of the new tissue generated during growth.

Anaemia

Anaemia is also a common accompaniment of protein-calorie malnutrition. The commonest form of anaemia is of the iron deficiency type, which is as expected, since iron deficiency is so widespread in the tropics. But there is very little response to iron therapy until such time as recovery from malnutrition also begins. In several countries the anaemia is reported to respond better to folic acid and B 12 than to iron. Hence during recovery it is important to administer several haematinics, the more so because treatment is usually based on an artificial formula containing high energy and protein, and not on a complete food as such.

PATHOLOGICAL FEATURES AND CHANGES IN METABOLISM

As the fat stores of the body are consumed and muscle tissue depleted total body water increases as a percentage of body weight. A direct relationship can be demonstrated between weight deficit and total body water. A proportionate increase occurs in the extra cellular fluid. On recovery, some of the excess extra cellular fluid is taken up by the re-generating cells and some is lost by diuresis.

As the tissue cells break down, potassium and nitrogen are lost in equal proportions initially. Later there is increased loss of potassium in diarrhoeal stools causing a cellular deficit of potassium. The total body protein is severely reduced, ranging from 55 to 80 per cent (average 59 per cent) of normal. Non-collagen protein is depleted more than collagen protein. Muscle mass is greatly diminished and may be only 30 per cent of normal for age. Similarly, in marasmus, body fat may fall as low as 5 per cent of total body weight, compared to the normal of 19 percent.

Changes in the digestive system

Liver (See fig. 7.13)

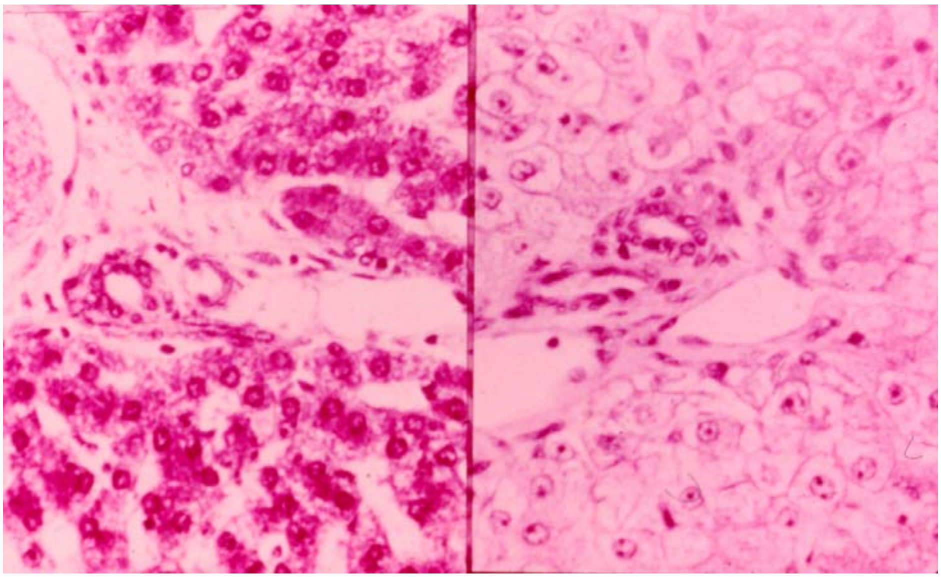


Figure 7.13 Fatty degeneration in the section on the right with normal liver tissue on the left for comparison.

(Note the wide separation of the nuclei in the section on the right due to accumulation of lipid in the cells.)

Fatty liver is characteristic of kwashiorkor. The fat content of the liver may be as high as 50 per cent of the total wet weight. As recovery occurs, fat gradually disappears. Electron microscopy of liver tissue obtained by biopsy reveals that after 3 weeks of treatment on a balanced diet the liver cells are still not visibly normal, even though serum protein levels have reached normal levels. Recognizable liver pathology can be identified even after 10-12 weeks of treatment. Total recovery eventually occurs and liver biopsy 5 years later on light microscopy has shown no signs of residual damage. There are two obvious reasons for the fatty liver. There is an increased flux of fatty acids from adipose tissue for the production of energy. At the same time there is decreased hepatic synthesis of P-lipoproteins which normally transport triglycerides from the liver. The synthesis of the apo-protein part of this fat transporting mechanism is particularly sensitive to lack of protein in the diet.

PANCREAS

There is a marked atrophy of the acinar cells, and exocrine secretion is reduced in keeping with the atrophic changes. Enzyme activity of the pancreatic juice has been reported to be as low as 50 per cent of normal.Recovery takes place within the first few days of instituting treatment. Investigation of B cell function reveals that in both kwashiorkor and marasmus insulin secretion is abnormally low after oral administration of glucose. Improvement occurs after 3-6 weeks of treatment, though there are instances where an abnormal response persisted up to 10 months after recovery from malnutrition.

GASTROINTESTINAL TRACT (See fig. 7.14)

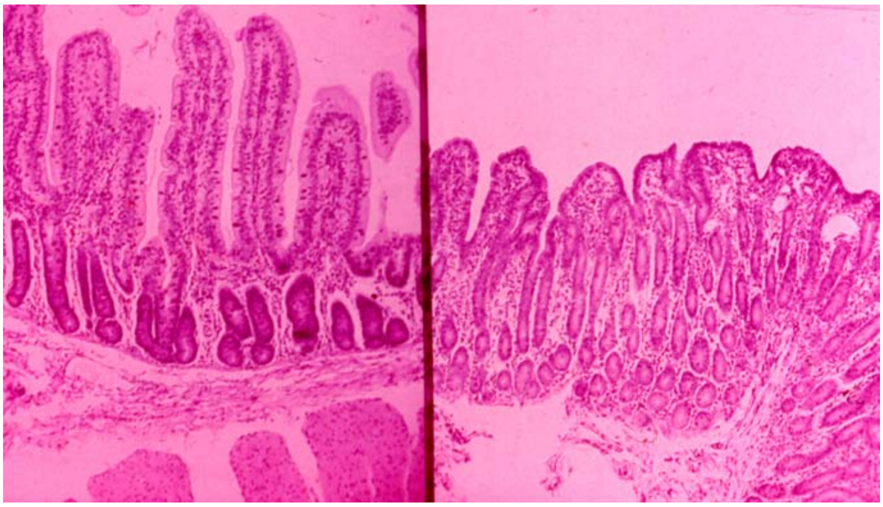


Figure 7.14 Villous atrophy in malnutrition seen in section on the right. Normal gut tissue for comparison on the left.

(Note the long feathery villi in the normal gut as compared to the blunted villi in the section on the right

Striking morphological changes occur in the jejunum, especially in kwashiorkor. In particular, villous atrophy may be severe. Enzyme activity is reduced within the cells in keeping with the morphological changes. The enzymes most affected are the ones located in the brush border. Of these, lactase has been studied extensively because it is more severely affected and because most diets used for recovery are based on milk and contain large quantities of lactose. Besides these morphological changes, the small intestine also suffers from bacterial overgrowth with invasion of the proximal gut by the bacterial flora of the distal part of the small intestine. Malabsorption of fat has been correlated with bacterial degradation of bile salts so that the concentration of conjugated bile salts in the gut lumen falls below the critical level necessary for forming micelles with fat. Intestinal permeability is increased as measured by the inert sugars lactulose and L-rhamnose. Decreased L-rhamnose recovery in urine indicating malabsorption, and increased lactulose recovery indicating permeability serve as independent predictors of mortality. Improvement in intestinal permeability is slow to occur with nutritional therapy, and can lag much behind improvement in weight.

Some atrophy of the gastric mucosa is common in the majority of cases. Fasting gastric pH is often in the neutral range. Thus the function of the stomach acid as a barrier to intestinal contamination is much reduced. This is supported by the observation that fasting stomach contents in children suffering from PEM have high bacterial and fungal counts.

The changes in gut morphology, together with reduction in the amounts of pancreatic enzymes and bacterial overgrowth of the small gut as well as parasitic disease, are together responsible for the common occurrence of diarrhoea in malnutrition. Impairment of absorption is also likely, but, in most cases, is not severe enough to interfere with recovery. For example, up to 33 g of fat containing unsaturated fatty acids is tolerated daily by malnourished children. Clinical experience with diets containing large quantities of vegetable fat supports this observation and cottonseed oil is now a common ingredient of many dietary regimens. With regard to protein digestion, it has been found that even though faecal nitrogen excretion in malnourished children is on average twice the normal, there is no serious malabsorption of nitrogen. More than three-quarters of the nitrogen in the diet is absorbed and is usually sufficient to allow the initiation of a cure except in very severe diarrhoea. Intolerance of lactose can present a serious problem at times, but even here the incidence of practical difficulties with feeding is small (less than 10 per cent). Thus, knowledge of the alteration indigestion and absorption is helpful in dealing with complications when they arise, but these are rare and in most cases it is possible to treat and rehabilitate children suffering from malnutrition without the need for sophisticated laboratory support.

HEART (See Fig. 7.15)

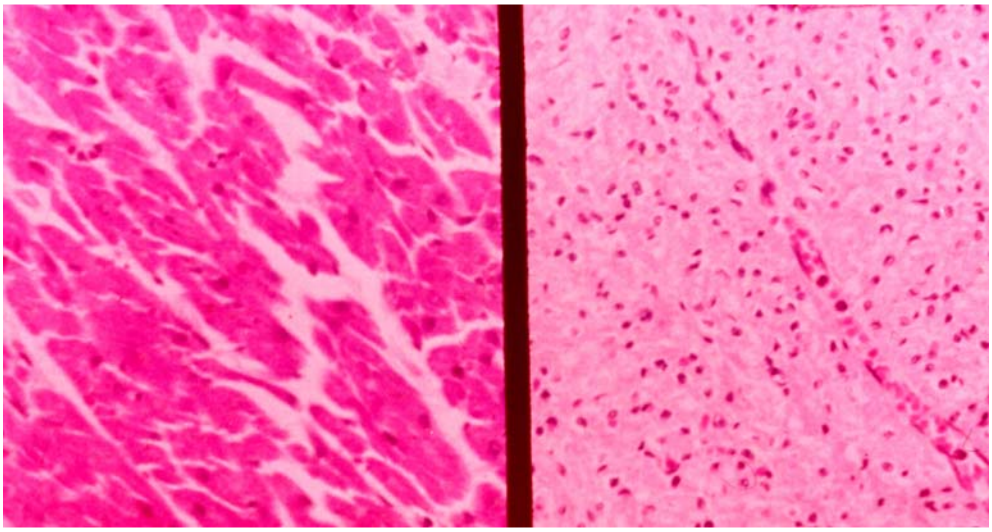


Figure 7.15 Normal myocardium on the left for comparison with section of heart muscle obtained at post mortem in a child who died of kwashiorkor.

(Note the degenerative changes with, intense vacuolation and cells of varying size in the section on the right).

The heart muscle suffers in the general atrophy of all muscle tissue. Cardiac output is reduced in accord with the reduced body metabolism. Doppler echocardiograms show reduced thickness of septum and posterior cardiac wall compared to age matched controls. The total left ventricular mass and cardiac output are reduced in proportion to decrease in body size. Institution of treatment, by stimulating metabolism, can often precipitate congestive cardiac failure. The salt content of the therapeutic diet and presence of anaemia may contribute to congestive cardiac failure.

HAEMOPOIETIC SYSTEM

A mild to moderate anaemia is a common accompaniment of PEM. Deficiency of nutrients such as protein, iron and folic acid, in addition to bone marrow depression due to infection, are all undoubtedly involved in the aetiology of the anaemia. Parasitic infections such as malaria and hookworm are also of relative importance in the tropics where childhood malnutrition is common. The fall in haemoglobin is related to reduction in the erythrocyte mass which commences with tissue wasting and loss of body weight. Megaloblastic changes in the bone marrow frequently occur and in some areas, e.g. the Sudan, the anaemia is reported to respond to folic acid administration. In the first few days of treatment, with regeneration of plasma proteins and the expansion of plasma volume, the concentration of haemoglobin may fall further, thereby accentuating the anaemia.

Immune System

Profound changes occur in cell-mediated immunity, the complement system, and in the function of polymorphonuclear lymphocytes. The thymus is consistently reduced in size. Seroconversion in response to vaccination seems to be preserved. The complement system shows consistent abnormality with decreased C3 levels. Alterations in defense mechanisms are further discussed below.

MUSCLE (See Fig. 7.16)

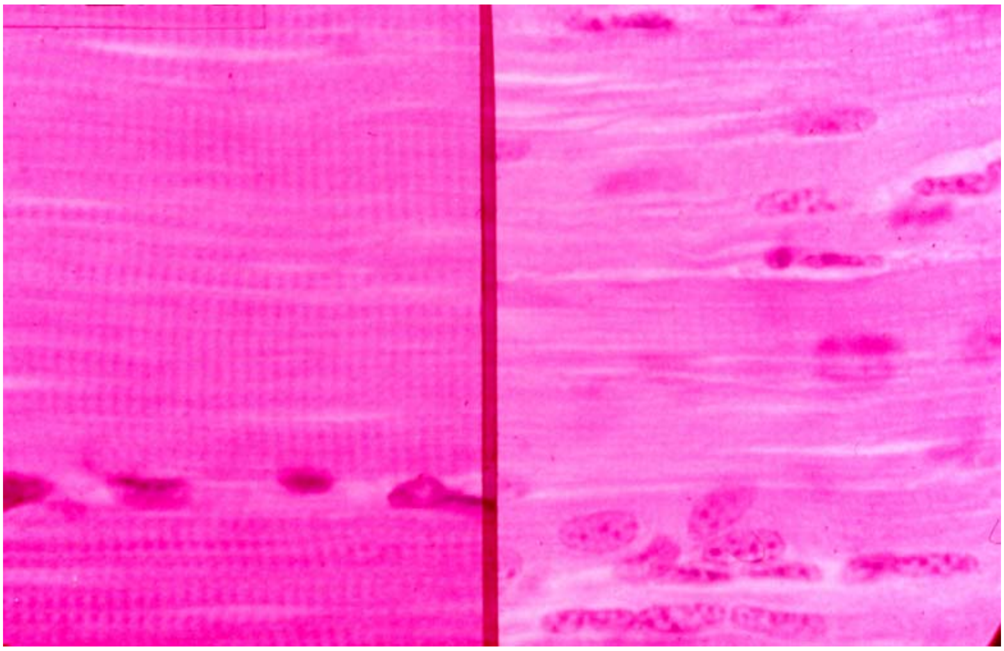


Figure 7.16 Skeletal muscle in severe malnutrition on the right hand section.

(Note the nuclei are concentrated in the right hand section indicating loss of cytoplasm from the cells. Both specimens show striations which are typical of skeletal muscle)

The muscle compartment of the body comprises a large mass of protein which is both labile and sensitive to dietary changes. Muscle wasting is an early result of PEM. Electron microscopy shows depletion of myofibrils, mitochondrial swelling, glycogen depletion and disorganization of sarcomere. At recovery the average muscle mass is usually about twice that during the malnourished state. Muscle and fat biopsies in malnourished Peruvian infants at the time of admission and again 4-9 months later, after recovery, show that there is a gross reduction of muscle cell size in malnourished infants. Improvement takes place with recovery, but the cell size continues to remain subnormal after recovery. Thus clinical recovery does not always reflect cellular maturity.

BRAIN AND THE NERVOUS SYSTEM

Research in the effects of malnutrition on the brain encounters severe difficulties. There is the virtual inaccessibility of the brain for study. Secondly, our ignorance of the physical basis of higher mental function is such that it is impossible to relate structural change to function Thirdly, the important effect of environmental stimulation on intellectual ability makes it difficult to untangle the effects of lack of stimulation from that of undernutrition, since often the two co-exist. However, animal experiments together with observations on long-term effects of malnutrition in children have helped to identify several basic principles. The vulnerability of the growing brain to periods of malnutrition is now widely accepted. The period of growth of the human brain corresponding to a "vulnerable period' in animal experiments would seem to extend from about mid-pregnancy to the second birth day.

The type of damage suffered is closely related to the timetable of development, and will depend upon the developmental events at the time of the insult. Growth impairment is more in the form of failure of assembly of certain components (or their sizes or numbers) rather than destructive lesions. Thus, the mature product may turn out to be not only deficient or small, but also distorted. Metabolic and biochemical functions of the brain may be altered, and several of these, like catechol amine metabolism, are related to higher mental functions. Hence at a time when a large number of components are being formed and assembled, the vulnerability is greatly increased, as for example during the period of the growth spurt of the brain.

The timetable of brain development is such that many of the event have possibly only a single opportunity to occur. If conditions are no optimal at a given time, that opportunity is lost and compensation may be difficult. This means that different parts of the brain may be affected to a variable extent by the same insult. The cerebellum, for example, is selectively affected and within the cerebellum certain structures bear the major brunt.

How do the above principles derived from laboratory studies an animal experiments relate to the situation in the human? In one study 74 J Jamaican school-age boys, who had suffered severe malnutrition in the first 2 years of life, were compared with male siblings closest in age and classmates or neighbours matched for age and sex. The IQ was found to be significantly lower in all aspects of measurements in the index cases, and in particular the full scale and verbal IQ. In this study, however, no relationship could be established between the level of IQ and the age at which malnutrition occurred. Another study from South Africa followed up 20 children who were grossly u undernourished in infancy, until they were 15-18 years old. All the children scored low on full scale and verbal quotient. Other tests showed a marked disturbance of visual-motor perception in 17 of these children. Even though a catch-up in height had occurred in all the children, the difference in head circumference as compared to controls got worse. The persistence of low IQ well into the teens indicates the permanence of damage to mental function. Similar studies from Uganda have confirmed these observations and have related the mental deficit to the chronic undernutrition suffered by such children.

When magnetic resonance imaging technique was applied to children admitted with severe kwashiorkor brain shrinkage was found in every one of those investigated. White and grey matter were found to be equally affected. Following three months of treatment the changes in brain show improvement in keeping with gain in weight and improvement in serum protein levels. (See Figs. 7.17 and 7.18)

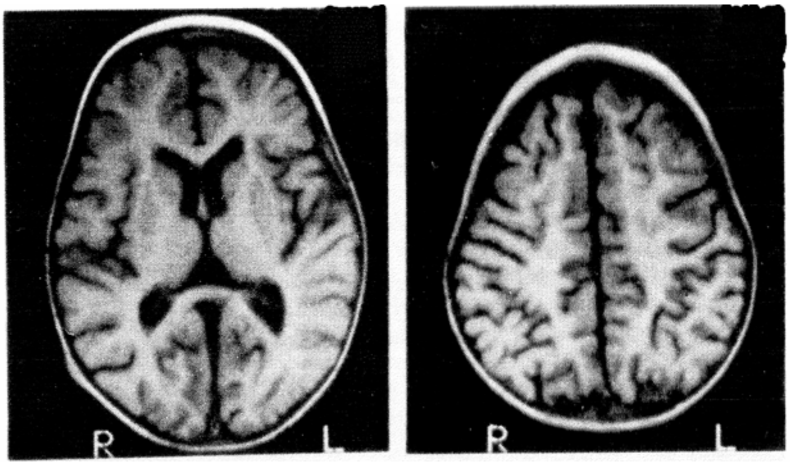


Figure 7.17 MRI scan in kwashiorkor

Typical MRI changes seen on admission (mid (left) and high (right) axial views)

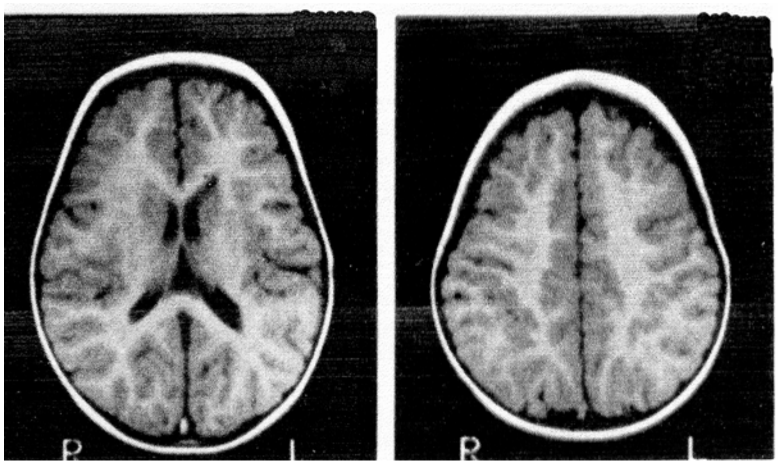


Figure 7.18 MRI scans after 90 days of treatment.

Mid and high axial views showing improvement.

Histology of brain sections taken from infants who died of severe malnutrition show shorter apical dendritic spines in the neurons as well as reduced number of dendrites as compared to normal control who died of other causes. These anatomic abnormalities may be related to the neuropsychological deficits that occur in malnutrition.

To conclude, if malnutrition is a measure of disadvantage and of deficiencies in the child's environment, then the damage or distorted growth suffered by the delicate nervous system represents a tragic outcome of such disadvantage. The stunting of brain growth condemns the victims of malnutrition to a lifetime of failure of learning.

Metabolic changes

CARBOHYDRATE METABOILISM

Low blood sugar is a common accompaniment of PEM. Two types of hypoglycemia have been identified - asymptomatic, from which recovery occurs with feeding; and the profound irreversible type, associated with severe mal nutrition, hypothermia or infection. As a general rule, if the child has hypothermia it is almost certain that hypoglycemia is also present.

FAT METABOLISM

Fat malabsorption is common in PEM, but the degree is rarely serious enough to cause steatorrhoea. Probably the most serious result of fat malabsorption is the impairment of absorption of fat-soluble vitamins.
It has been found that vegetable fats are better absorbed than animal fats and this is the rationale behind the use of cottonseed oil as a source of energy in the treatment of kwashiorkor.

PROTEIN METABOLISM

Protein digestion, though inefficient because of low levels of pancreatic trypsin is sufficient for recovery to occur when an adequate diet is being fed. On average, absorption of nitrogen from a milk-based diet is 70-80 per cent as compared to 90 per cent in the normal child. Similarly, in the absence of complicating infection, nitrogen is well retained, being in the range of 20-40 per cent of the intake. Thus, protein repletion through a greatly enhanced anabolism is usual as soon as sufficient protein is given. Nitrogen retention continues to be high until a normal growth rate has been attained.

Albumin synthesis and its level in plasma are very sensitive to protein intake. There is an immediate fall when dietary deficiency occurs, and a rise when the deficiency is corrected. Serum albumin levels are also sensitive to infection. The longitudinal study of Ugandan children referred to earlier showed that a combination of respiratory infection, diarrhoea and malaria caused a dramatic fall in serum albumin.

Defense mechanisms

The child with malnutrition is susceptible to infection. The body's defenses are unable to mount an adequate response to microbial challenge so that the mildest infection tends to spread and become generalized. In severe cases the clinical response to infection, like fever and phagocytosis, may be absent and the first sign of widespread infection may be sudden deterioration in the general condition, refusal to take food and hypothermia.

Studies of the body's defense mechanisms reveal adequate capacity for humoral immunity. Thus immunoglobulin levels in the blood are normal and there are normal numbers and proportions of B lymphocytes, which produce immunoglobulins. Secretory IgA in salivary and naso-pharyngeal secretions, and in the gastrointestinal tract, is reduced and does not rise in response to antigenic challenge. This impairment of secretory antibody response in malnourished children explains their slow recovery from enteric infections and viral illnesses like measles. In contrast to humoral immunity, cellular immunity (T-cell function) is profoundly impaired. T-lymphocytes are reduced in number to about a third of normal and various tests of their functions also show impairment. In keeping with the lowered cellular immunity, all lymphoid organs show atrophy, especially the thymus. Several studies have now established that in protein-energy malnutrition and/or deficiency of single nutrients that take part in nucleic acid metabolism generally there is atrophy of lymphoid tissue and dysfunction of cell mediated immunity. Deficiencies of single nutrients can impair the production of key proteins. A useful clinical sign for assessing the involution of lymphoid organs is the size of the tonsils in malnourished children. Phagocytic function is also inefficient and several workers have emphasized its correlation with iron deficiency, especially lower levels of serum transferrin.

Accompanying deficiency of micronutrients often compounds the difficulty. Of the micronutrients vitamins A, C, E and B6 and folic acid are important to consider. Metals like zinc, selenium, iron, copper have influences on immune response, and many are deficient in severe malnutrition.

Immunological recovery in response to treatment lags much behind improvement in weight. Hence the child remains prone to cross infection for a long time after clinical recovery and discharge from hospital.

Management of Severe Malnutrition

Severe malnutrition is a serious disease. The median case fatality from the disease has remained unchanged over several decades at 20 – 30 per cent. A likely cause could be faulty case management with diets that are high in protein, energy and sodium as well deficient in micronutrients placing undue stress on run down enzyme systems. Updated treatment guidelines have been issued by the World Health Organization on which the following description is based.

The objectives of treatment are:

To achieve stabilization and institute correction of chemical imbalances.

To treat complications and reduce case fatality - the present mortality rates as high as 20-30 per cent can be reduced by careful reversal of the metabolic and physiologic slowdown in the organ systems.

To achieve rehabilitation on a well-balanced diet;

To prevent relapse and future deterioration through education of the parents;

To achieve long-term follow-up with a view to restoring the child to full health.

The initial requirement is to carefully assess for and treat complications like shock, dehydration, hypoglycemia, hypothermia, and infection. The early days are critical as the child is desperately ill and the body's metabolic processes are at an ebb. Suspect **shock** if the child is lethargic or unconscious in addition to the other clinical signs of shock like cold extremities and thready pulse. Shock from dehydration and sepsis is likely to coexist, and what is more, response to intravenous therapy in septic shock is slow. While preparing for intravenous fluids for treatment of shock check for and treat hypoglycemia and hypothermia.
Hypoglycemia is an ever present risk for all severely malnourished children. Immediately on admission they should receive oral glucose or sucrose solution or a feed if it is quickly available. If a feed cannot be made quickly available, dissolve 1 rounded teaspoon of sugar in 3 ½ tablespoons of water and feed it to the child to be followed by the first feed as soon as possible. If the child is unconscious 5 ml/kg of 10% glucose should be given by a nasogastric tube. Testing for hypoglycemia (blood sugar < 3 mmol/L or < 54 mg/dl) by dextrostix should be carried out immediately on admission for all patients.

Hypothermia, if present with hypoglycemia, is often a sign of serious infection and associated with high fatality. Axillary temperature of < 35^{0}C or rectal temperature <35.5^{0}C indicates hypothermia. Treatment includes immediate feeding with 10% glucose or sucrose as above, followed by oral feeds, covering with bed clothes and nursing in close proximity to a source of heat including mother’s body heat.

Infection. The usual signs of infection are not present, and yet prevalence of pneumonia, bacteraemia, and urinary tract infection is high. WHO advises empirical treatment with broad spectrum antibiotics for the first seven days. Intravenous administration of antibiotics is recommended if hypothermia or hypoglycemia is present, or the child is lethargic or appears very toxic and ill. It is the usual experience that the majority of children have some or all of the following: malaria, worm infestation, respiratory infection some with tuberculosis, skin sepsis, or urinary infection. Screening for, and treating these conditions if present, is an essential part of management.

Dehydration. If the child is passing watery stools one should assume some dehydration and the use of oral rehydration is recommended. The WHO ORS solution has a high sodium and low potassium content and is not considered suitable for severely malnourished children. Instead WHO recommends a modified solution named ReSoMal, to be administered at the rate of 5 ml/kg every 30 minutes for the first 2 hours and 5 ml/kg/hour for the next 4 to 10 hours. ReSoMal is prepared as shown in Table 7.3:

Table 7.3 Formula for ReSoMal

Ingredient	Amount
Water	2litres
WHO – ORS	One 1-litre packet
Sugar (Sucrose)	50g
KCl solution*	45 ml
or	or
Electrolyte-mineral solution**	40 ml

* KCl solution is prepared by dissolving 100g of KCl in 1 litre of water
** Electrolyte-mineral solution is prepared according to the formula in Table 7.4 below.

If shock has been diagnosed, intravenous treatment with any of the following should be commenced:

Ringer's lactate with 5% glucose
or
half-strength Darrow's solution with 5% glucose
or
half normal saline with 5% glucose.

A careful and measured approach is necessary bearing in mind the weakened state of the myocardium and of the cardio vascular system as a whole. In the first hour 15 ml/kg of the fluid is administered to be repeated if the child shows improvement. If there is no change consider septic shock.

Feeding during the initial phase of stabilization.

Feeding should commence as soon as possible after admission. If the child is breast feeding it should be continued. Initial feeds should provide just enough energy and protein to maintain physiologic processes, bearing in mind the run-down state of metabolic processes. Too much food, particularly protein, in the initial phase aggravates the metabolic imbalance. Hence a cautious approach is needed to begin with, and once the initial crisis is over intake of energy and protein can be increased to make up the shortfall. Initial feeds should be:

low in osmolality and lactose provide 100 kcal/g/day, and 1 to 1.5 g/kg protein per day in 130 ml/kg/day of fluid frequent and small if the child can take by mouth; if not, administered by nasogastric tube.(see Fig. 7.19)

Figure 7.19 Feeding with nasogastric tube.

In addition to protein and energy severely malnourished children are often deficient in a number of **micronutrients** like zinc, iron, copper and selenium as well as vitamins A, B complex, folic acid, and so on. These need to be provided with the feeds to help replenish body stores. Many of them are important constituents of cells. Moreover, deficiencies in zinc and vitamin A impair the function of the immune system, and are needed for the structure and function of mucosa. Zinc supplementation reduces the incidence of diarrhoea and pneumonia, and improves growth. Vitamin A supplementation is known to reduce mortality and morbidity due to diarrhoea and measles. Iron supplementation improves cognition and growth. Finally, many of the trace metals like copper and selenium are important constituents of body cells, and would be needed as tissue regeneration commences.

Electrolyte imbalance is common in severe malnutrition. Potassium and magnesium are important constituents of cells. As the patient recovers in response to diet and homeostasis gets established, electrolyte imbalance gets corrected but over a period of two weeks or more. WHO recommends supplying extra potassium at the rate of 3 to 4 mmol/kg daily, and magnesium at the rate of 0.4 to 0.6 mmol/kg daily. In the early stage of stabilization when only milk based feed are tolerated minerals and electrolytes can be supplemented together in the form of an electrolyte-mineral solution added to the feeds. A concentrated form of electrolyte-mineral solution can be made up to serve as a stock solution. When stored in a fridge the stock solution has a shelf life of about 1 month. It should be discarded if it turns cloudy, and a fresh stock solution be made. Add 20 ml of the concentrated solution to each 1000 ml of milk feed. The formulae for the electrolyte-mineral solution and the starter milk feeds are given below in tables 7.4 and 7.5.

Table 7.4 Composition of concentrated electrolyte-mineral solution

Ingredient	Weight (g)
Potassium chloride (KCl)	224
Tripotassium citrate	81
Magnesium chloride	76
Zinc acetate	8.2
Copper sulphate	1.4
Water to make up	2500 ml

Add 20 ml of the above concentrated electrolyte-mineral solution to each 1000 ml of milk feed.

Each 20 ml of the solution contains the following:

Potassium chloride	24 mmol
Tripotassium citrate	2 mmol
Magnesium chloride	3 mmol
Zinc acetate	300 µmol
Copper sulphate	45 µmol

Use 40 ml of the concentrated electrolyte-mineral solution for the ReSoMal solution described in Table 7.3

Table 7.5 Starter milk formula for treating malnutrition

	Starter formula using skimmed milk powder	Starter formula using cereal flour* and skimmed milk
Dried skimmed milk (g)	25	25
Sugar (g)	100	70
Cereal flour (g)	-	35
Vegetable oil (g)	27	27
Electrolyte-mineral solution (ml)	20	20
Water to make up final solution to (ml)	1000	1000

* The advantage of adding cereal flour is that of low osmolality which is of benefit for children with persistent diarrhoea. The disadvantage is that it needs to be cooked for 4 minutes.

A comparable starter formula based on dried full cream milk powder can be made from:

Whole dried milk powder (g)	35
Sugar (g)	100
Oil (g)	20
Electrolyte-mineral solution (ml)	20
Water to make (ml)	1000 ml

If using fresh cow's milk, the dried whole milk powder in the above list of ingredients may be replaced with 300 ml milk.

The above formulae provide nutrients per 1000 ml as follows:

Nutrient per 1000 ml	**Starter formula using skimmed milk**	**Starter formula using cereal flour and skimmed milk**
Energy (kcal)	75	75
Protein (g)	0.9	1.1
Lactose (g)	1.3	1.3
Potassium (mmol)	4.0	4.2
Sodium (mmol)	0.6	0.6
Magnesium (mmol)	0.43	0.46
Zinc (mg)	2.0	2.0
Copper (mg)	0.25	0.25
% energy from protein	5	6
% energy from fat	32	32
Osmolality (mosmol/l)	413	334

Feeding Schedule.

A recommended feeding schedule with gradual increase in the volume and a proportional decrease in frequency of feeds is as follows:

Days	Frequency	Volume /kg/feed	Volume/kg/day
1 – 2	2 hourly	11 ml	130 ml
3 – 5	3 hourly	16 ml	130 ml
6 onwards	4 hourly	22 ml	130 ml

Night feeds are essential. If there is staff shortage or if the carer accompanying the child cannot be trained a nasogastric tube may be the only option. It is important, however, not to exceed 100 kcal/kg/day in this critical initial phase. The volume of feed to be administered by the child's weight is given in Table 7.6 below.

Table 7.6 Volume of Starter formula per feed by child's weight.

Child's weight (kg)	2-hourly (ml / feed)	3-hourly (ml / feed)	4-hourly (ml/feed)
2.0	20	30	45
2.2	25	35	50
2.4	25	40	55
2.6	30	45	55
2.8	30	45	60
3.0	35	50	65
3.2	35	55	70
3.4	35	55	75
3.6	40	60	80
3.8	40	60	85
4.0	45	65	90
4.2	45	70	90
4.4	50	70	95
4.6	50	75	100
4.8	55	80	105
5.0	55	80	110
5.2	55	85	115
5.4	60	90	120
5.6	60	90	125
5.8	65	95	130
6.0	65	100	130
6.2	70	100	135
6.4	70	105	140
6.6	75	110	145
6.8	75	110	150
7.0	75	115	155
7.2	80	120	160
7.4	80	120	160
7.6	85	125	165
7.8	85	130	170
8.0	90	130	175
8.2	90	135	180
8.4	90	140	185
8.6	95	140	190
8.8	95	145	195
9.0	100	145	200
9.2	100	150	200
9.4	105	155	205
9.6	105	155	210
9.8	110	160	215
10.0	110	160	220

Vitamin supplements.

Protein energy malnutrition is a multi nutrient deficiency. In addition to minerals and electrolytes supplementation with vitamins is important since many of them serve as co-factors in the chemical reactions within cells. WHO recommends particular attention to **vitamin A** (50 000 i.u. for children less than 6 months old; 100 000 i.u. for those aged 6 to 12 months; and 200 000 i.u. for older children) on day 1.
Folic acid 5 mg. should be administered on day 1 then, 1 mg daily.
A multivitamin preparation to provide vitamins of the **B complex** group should also be administered daily.

The Catch up phase

Return of appetite and improvement in the mental state of the patient signals that the critical early phase has been effectively dealt with, and the time for catching up on lost ground has come. This is approximately a week or so after admission. The goal of treatment now is to regenerate tissue growth and to achieve weight gain greater than 10g/kg/day. The energy cost of laying down 1g of muscle tissue is 2kcal/g body weight, and that of laying down 1g of fat is between 8 to 10 kcal/g/body weight. Thus catch up growth will depend on the provision of calories, protein and other nutrients in excess of normal requirements. Even then a cautious approach is needed for fear of stressing the slowly recovering physiological processes. Frequent feeds, amount depending upon the patient's appetite help to achieve energy and protein intake of 150 – 220 kcal/kg/day and 4 to 6 g /kg/day of protein. A switch over to a stronger formula for catch up growth that provides 100 kcal and 2.9 g protein per 100 ml can now be considered. The recipe for such a formula is in Table 7.7 below.

Table 7.7 Recipe for catch up formula

Ingredients	Formula based on dried skimmed milk powder	Formula based on dried whole milk powder	Formula based on fresh cow's milk
Milk (g)	80	110	880 ml
Sugar (g)	50	50	75
Vegetable oil (g)	60	30	20
Electrolyte-mineral solution (ml)	20	20	20
Water to make up final volume (ml)	1000	1000	1000

The catch up formula provides nutrients as follows:

Nutrient / 100 ml	
Energy (kcal)	100
Protein (g)	2.9
Lactose (g)	4.2
Potassium (mmol)	6.3
Sodium (mmol)	1.9
Magnesium (mmol)	0.73
Zinc (mg)	2.3
Copper (mg)	0.25
% energy from protein	12
% energy from fat	53
Osmolality (mosmol/l)	419

Daily administration of the multivitamin supplement and folic acid must continue in the catch up phase. Modified porridge and other foods may be offered additionally. These can be modified as appropriate to provide energy and protein content as in the catch up formula. The catch up phase is also the time for providing iron supplement orally if indicated.

Frequency of feeds.

The feeding schedule should continue as in the initial stabilization phase for the first 2 or 3 days, the only difference being that the catch up formula now replaces the previous starter formula. If tolerated each successive feed can be increased by 10 ml until some feed remains unconsumed, which is approximately when intake reaches about 200ml/kg/day. If the child is breast feeding it should continue.

As catch up begins and the blood volume expands there is a danger of precipitating cardiac failure. The child should be carefully monitored for tachypnoea and tachycardia. Cardiac failure is unlikely if a gradual transition to catch up formula is followed. If suspected the volume of feeds should be reduced to 100 ml/kg/day for 24 hours, and then gradually increased to 115 ml/kg/day for the next 24 hours and 130 ml/kg/day for following 48 hours.

Monitoring growth in the catch up phase.

If weight gain is more than 10 g/kg/day the child is progressing well and the treatment should be continued. If weight gain is between 5 – 10 g/kg/day it is unlikely that intake targets of nutrients are being met, or that underlying infection has been missed. If weight gain is < 5g/kg/day a full assessment is called for. Feeding schedules and targets should be reviewed. Investigations for infection, particularly tuberculosis should be undertaken. The quality of mothering and nursing care should be assessed.

Nutrition Rehabilitation phase

Once the child is eating with full appetite, oedema has disappeared, weight gain is satisfactory and the full course of antibiotics has been completed it is a matter of time before (usually 2 to 4 weeks) weight for length is reached 90%. (See table 7.8).

Table 7.8 Weight for Length (WHO/NCHS standard)

Boys' Weight(kg)						Girls' Weight (kg)				
-4SD 60%	-3sd 70%	-2SD 80%	-1sd 90%	**Median**	**Length (cm)**	**Median**	-1sd 90%	-2sd 80%	-3sd 70%	-4sd 60%
1.8	2.1	2.5	2.8	3.1	**49**	3.3	2.9	2.6	2.2	1.8
1.8	2.2	2.5	2.9	3.3	**50**	3.4	3	2.6	2.3	1.9
1.8	2.2	2.6	3.1	3.5	**51**	3.5	3.1	2.7	2.3	1.9
1.9	2.3	2.8	3.2	3.7	**52**	3.7	3.3	2.8	2.4	2
1.9	2.4	2.9	3.4	3.9	**53**	3.9	3.4	3	2.5	2.1
2	2.6	3.1	3.6	4.1	**54**	4.1	3.6	3.1	2.7	2.2
2.2	2.7	3.3	3.8	4.3	**55**	4.3	3.8	3.3	2.8	2.3
2.3	2.9	3.5	4	4.6	**56**	4.5	4	3.5	3	2.4
2.5	3.1	3.7	4.3	4.8	**57**	4.8	4.2	3.7	3.1	2.6
2.7	3.3	3.9	4.5	5.1	**58**	5	4.4	3.9	3.3	2.7
2.9	3.5	4.1	4.8	5.4	**59**	5.3	4.7	4.1	3.5	2.9
3.1	3.7	4.4	5	5.7	**60**	5.5	4.9	4.3	3.7	3.1
3.3	4	4.6	5.3	5.9	**61**	5.8	5.2	4.6	3.9	3.3
3.5	4.2	4.9	5.6	6.2	**62**	6.1	5.4	4.8	4.1	3.5
3.8	4.5	5.2	5.8	6.5	**63**	6.4	5.7	5	4.4	3.7
4 4.3	4.7	5.4	6.1	6.8	**64**	6.7	6	5.3	4.6	3.9
	5	5.7	6.4	7.1	**65**	7	6.3	5.5	4.8	4.1
4.5	5.3	6	6.7	7.4	**66**	7.3	6.5	5.8	5.1	4.3
4.8	5.5	6.2	7	7.7	**67**	7.5	6.8	6	5.3	4.5
5 1	5.8	6.5	7.3	8	**68**	7.8	7.1	6.3	5.5	4.8
5.3	6	6.8	7.5	8.3	**69**	8.1	7.3	6.5	5.8	5
5.5	6.3	7	7.8	8.5	**70**	8.4	7.6	6.8	6	5.2
5.8	6.5	7.3	8.1	8.8	**71**	8.6	7.8	7	6.2	5.4
6	6.8	7.5	8.3	9.1	**72**	8.9	8.1	7.2	6.4	5.6
6.2	7	7.8	8.6	9.3	**73**	9.1	8.3	7.5	6.6	5.8
6.4	7.2	8	8.8	9.6	**74**	9.4	8.5	7.7	6.8	6
6.6	7.4	8.2	9	9.8	**75**	9.6	8.7	7.9	7	6.2
6.8	7.6	8.4	9.2	10	**76**	9.8	8.9	8.1	7.2	6.4
7	7.8	8.6	9.4	10.3	**77**	10	9.1	8.3	7.4	6.6
7.1	8	8.8	9,7	10.5	**78**	10.2	9.3	8.5	7.6	6.7
7.3	8.2	9	9.9	10.7	**79**	10.4	9.5	8.7	7.8	6.9
7.5	8.3	9.2	10.1	10.9	**80**	10.6	9.7	8.8	8	7.1
7.6	8.5	9.4	10.2	11.1	**81**	10.8	9.9	9	8.1	7.2
7.8	8.7	9.6	10.4	11.3	**82**	11	10.1	9.2	8.3	7.4
7.9	8.8		10.6	11.5	**83**	11.2	10.3	9.4	8.5	7.6
8.1	9	9.9	10.8	11.7	**84**	11.4	10.5	9.6	8.7	7.7

Boy's weight (Kg)							Girls' weight (kg)			
-4sd 60%	-3sd 70%	-2sd 80%	-1sd 90%	Median	Length (cm)	Median	-1sd 90%	-2sd 80%	-3sd 70%	-4sd 60%
7.8	8.9	9.9	11	12.1	**85**	11.8	10.8	9.7	8.6	7.6
7.9	9	10.1	11.2	12.3	**86**	12	11	9.9	8.8	7.7
8.1	9.2	10.3	11.5	12.6	**87**	12.3	11.2	10.1	9	7.9
8.3	9.4	10.5	11.7	12.8	**88**	12.5	11.4	10.3	9.2	8.1
8.4	9.6	10.7	11.9	13	**89**	12.7	11.6	10.5	9.3	8.2
8.6	9.8	10.9	12.1	13.3	**90**	12.9	11.8	10.7	9.5	8.4
8.8	9.9	11.1	12..3	13.5	**91**	13.2	12	10.8	9.7	8.5
8.9	10.1	11.3	12.5	13.7	**92**	13.4	12.2	11	9.9	8.7
9.1	10.3	11.5	12.8	14	**93**	13.6	12.4	11.2	10	8.8
9.2	10.5	11.7	13	14.2	**94**	13.9	12.6	11.4	10.2	9
9.4	10.7	11.9	13.2	14.5	**95**	14.1	12.9	11.6	10.4	9.1
9.6	10.9	12.1	13.4	14.7	**96**	14.3	13.1	11.8	10.6	9.3
9.7	11	12.4	13.7	15	**97**	14.6	13.3	12	10.7	9.5
9.9	11.2	12.6	13.9	15.2	**98**	14.9	13.5	12.2	10.9	9.6
10.1	11.4	12.8	14.1	15.5	**99**	15.1	13.8	12.4	11.1	9.8
10.3	11.6	13	14.4	15.7	**100**	15.4	14	12.7	11.3	9.9
10.4	11.8	13.2	14.6	16	**101**	15.6	14.3	12.9	11.5	10.1
10.6	12	13.4	14.9	16.3	**102**	15.9	14.5	13.1	11.7	10.3
10.8	12.2	13.7	15.1	16.6	**103**	16.2	14.7	13.3	11.9	10.5
11	12.4	13.9	15.4	16.9	**104**	16.5	15	13.5	12.1	10.6
11.2	12.7	14.2	15.6	17.1	**105**	16.7	15.3	13.8	12.3	10.8
11.4	12.9	14.4	15.9	17.4	**106.**	17	15.5	14	12.5	11
11.6	13.1	14.7	16.2	17.7	**107**	17.3	15.8	14.3	12.7	11.2
11.8	13.4	14.9	16.5	18	**108**	17.6	16.1	14.5	13	11.4
12	13.6	15.2	16.8	18.3	**109**	17.9	16.4	14.8	13.2	11.6
12.2	13.8	15.4	17.1	18.7	**110**	18.2	16.6	15	13.4	11.9

This period is best utilized for educating parents regarding home care and nutrition. Since full immunologic recovery usually lags far behind nutritional recovery it is best to move the child away from the risk of cross infection in the acute care unit to a recovery unit. Also parental education is more effective in amore settled and quiet environment away from the rush and commotion of an acute pediatric ward. Many hospital authorities have found it useful to have a nutrition rehabilitation centre as an annex to the pediatric ward for this purpose.

The **nutrition rehabilitation centre** was first described in the 1960s as a convalescent place or staging post between the hospital ward and the home. Two advantages were immediately obvious. Firstly, it took the pressure off the crowded hospital wards where treatment costs were becoming prohibitive. Secondly, nutrition education of the mothers was easier to organize and more effective in the relatively quiet environ-ment of the nutrition rehabilitation centre in comparison with the bustle in the acute paediatric ward. Soon the activities of the nutrition rehabilitation centre were expanded to include training in mother craft, in improved techniques of farming, raising poultry, environmental sanitation and other similar activities. When the activities of the nutrition rehabilitation centres were integrated with those of the out-patient services and under-5s clinics, they became the focal points of maternal and child health work and of disseminating new knowledge in the community. Mothers admitted to such centres often take on the role of advisers in child feeding in their immediate neighbourhood. This has the advantage of creating a locally available source of knowledge in infant feeding for the community.

The nutrition rehabilitation centre is basically a school for parents who come together as either residents or day visitors to learn about better ways of feeding their children. They learn by participation and by observing their children thrive on foods which they have cooked a the centre. Thus, in leading the child back to health the emphasis now shifts from the hospital environment to the home environment. (See Fig. 7.20)

Figure 7.20 Nutrition Rehabilitation Unit with Pediatric wards in the distance

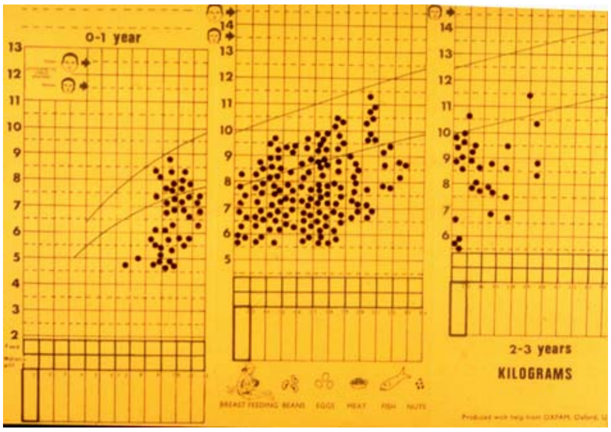
Figure 7.21 Mothers learning to cook a nutritious meal.

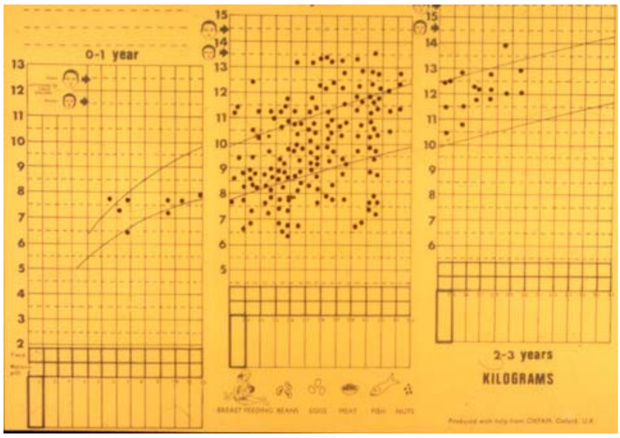
Figure 7.22 A group discussion with vegetable garden in background

Health workers now step back and allow the auxiliaries to take over as educators and demonstrators. The emphasis is on locally available foods, prepared and cooked in the home kitchen, and as befits the domestic economy. The facilities consist of a residential unit or a ward with a kitchen and a plot of land where the foods used in the daily cooking are grown. The objective is to demonstrate the cooking of multimixes which provide adequate calories and protein in manageable bulk. Whilst waiting for the child to recover rehabilitation is also to be thought of as cognitive recovery. Engaging the child in stimulating play activities is helpful for mental stimulation. Individual and group play help in the overall recovery process.

The development of the nutrition rehabilitation centres has been a useful step towards integration of treatment with prevention. After the initial period of acute illness, medical care is actively replaced by informed lay care as recovery proceeds. Moreover, there is cross-fertilization between medicine and several other related disciplines like community development, agriculture, education and social sciences. Such a unified team approach gives better results than the individual activities of the same disciplines based on rigid departmental lines.

A recent evaluation in Uganda, Peru, Haiti and Guatemala has proved that children attending these centres do show improved growth and nutrition recuperation in an impressive way.(See Figs 7.23 and 7.24).





Weights of children on admission and at the time of discharge from the nutrition rehabilitation centre

Figure 7.23 (left) Weights on admission

Figure 7.24 (right Weights at the time of discharge

The cost per child per day ranged from US$372 in Haiti to US$876 in Guatemala, and even though ten to forty times less than that of hospitalization, can still be called excessive. The effect of the nutrition rehabilitation centre on the knowledge of the mother was not always impressive. Thus there is still a great deal of room for improvement.

Daily diet in the nutrition rehabilitation centre

The catch up formula described in Table 7.7.is the basis of feeding and recovery. At the same time the child may be offered solids as required. Once the child is eating well the catch up formula feeds may be tapered off so that by the time of discharge the child is eating foods that the mother has learned to cook in the centre. A specimen diet is illustrated in Table 7.9. (See table 7.9)

Table 7.9 A specimen daily diet in the nutrition rehabilitation centre

Food	(g)	Cals	Prot. (g)	Fat (g)	CHO (g)	Ca (mg)	Fe (mg)	VitA (i.u.)	Thiamine (mg)	Riboflavin (mg)	Niacin (mg)	Vit C (mg)
Bread	30	74	2.1	0.2	16.0	3.40	0.30	-	0.2	0.1	0.2	-
Margarine	2	15	—	1.7	—	0.08	—	56				
Tea	212	119	4.5	2.1	20.0	159:00	0.13	91	0.055	0.198	0.17	2.3
Breakfast		**208**	**6.6**	**4.0**	**36.0**	**162.5**	**0.43**	**147**	**0.26**	**0.3**	**0.37**	**2.3**
Porridge with eggs	156	112	5.6	4.7	12.3	90.0	0.80	280	0.55	0.16	0.16	0.6
Paw paw	56	22	0.4		5.2	11.4	0.20	568	0.02	0.02	0.02	34.0
Sugar	10	40	—	—	10.0	—	—	--	—	—	—	—
Mid-morning		**174**	**6.0**	**4.7**	**27.5**	**101.4**	**1.0**	**848**	**0.57**	**0.18**	**0.36**	**34.6**
Rice	155	132	2.8	0.16	28.8	1.86	0.31		0.022	0.011	0.37	-
Mince	100	137	12.4	9.80	—	14.00	1.70		0.050	0.110	2.70	14-
Soup	80	9	0.7	0.08	1.4	34.00	0.56	530	0.016	0.040	0.24	
Stiff maize porridge	162	226	5.7	0.84	49.0	2.35	1.30		0.032	0.019	0.39	—
Mince	100	137	12.4	7.80	—	14.00	1.70		0.050	0.110	2.80	_
Soup	80	9	0.7	0.08	1.4	34.00	0.56	530	0.016	0.040	0.24	14
Lunch with Rice		**278**	**15.9.**	**10.04**	**30.2**	**49.9**	**2.6**	**530**	**0.09**	**0.16**	**3.4**	**14**
Lunch with Stiff porridge		**372**	**18.8**	**10.72**	**50.4**	**50.4**	**3.6**	**530**	**0.10**	**0.17**	**3.4**	**14**

Food	(g)	Cals.	Prot (g)	Fat (g)	CHO (g)	Ca (mg)	Fe (mg)	Vit A (i.u.)	Thiamin (mg)	Ribo .(mg)	Niacin (mg)	Vit C (mg)
Afternoon tea												
Bread	2	80	2.2	0.2	17.1	3.60	0.35		0.019	0.013	0.22	0
Margarine	3	23	-	2.5	-0.12	-	85	-	-	-	-	-
Milky Tea with sugar	212	119	4.5	2.1	20.0	159.0	0.13	91	0.055	0.198	0.17	2.3
Orange (1/2)	70	37	0.6	-	9.1	21.0	35	21	0.060	0.020	0.14	31.0
Tea time total		**259**	**7.3**	**4.8**	**46.2**	**183.7**	**0.83**	**197**	**0.13**	**0.23**	**0.5**	**33.3**
Supper												
Rice	110	94	2.0	0.11	20.4	1.3	0.22	-	0.015	0.008	0.26	-
Mince	113	155	14.0	11.10	-	15.8	1.92	-	0.057	0.124	3.16	-
Soup	46	-	-	-	-	-	-	-	-	-	-	-
Supper total		**249**	**16.0**	**11.2**	**20.4**	**17.1**	**2.14**	**-**	**0.07**	**0.13**	**3.4**	**-**
Average Daily total		**1215**	**53.3**	**35.1**	**170.4**	**514.8**	**7.5**	**1722**	**1.13**	**1.01**	**8.03**	**84.2**
R.D.A.	1-3 yrs. 3-6 yrs.	1130 1510	40 50			400 400	7 8	2000 2500	0.6 0.8	1.0 1.2	6 8	35 50

R.D.A. = Recommended daily Allowance

Several shortcomings of the nutrition rehabilitation centre are obvious, even though it is a significant advance compared to the hospital ward for the treatment of malnutrition. Only a small proportion of all children suffering from malnutrition in the community are brought to a health facility for treatment, and only a proportion of these will be eventually admitted to the nutrition rehabilitation centre. Thus a centre can provide only limited coverage. It is physically impossible to provide one centre for each village in a country.
For these reasons, health workers have experimented with several types of cost-effective approaches.

Outpatient care for severely malnourished children.

As experience has been gained with regard to the care and management of severely malnourished children a number of modifications have been attempted in different countries to suit the local situation. In Indonesia, health workers treated severely malnourished children as out-patients and reported a lower case fatality rate of 16.6% compared with 50% for hospital based care. In a controlled trial in Bangladesh researchers compared home based care with day care and in-patient management of severe malnutrition. They found home based care to be most cost effective being five times cheaper than in-patient care. As the patho-physiology of severe malnutrition has come to be better understood its management is getting rationalized and is increasingly undertaken at five different levels depending upon available resources viz. in hospitals; in nutrition rehabilitation units; in health centers; in community out-reach clinics; and at home with regular follow-up. It is the initial intensive phase which requires careful search and treatment of life threatening complications like septiceamia, hypoglycaemia, hypothermia and electrolyte imbalance. In this phase of stabilization it is also necessary to avoid metabolic stress by offering feeds with relatively low concentration of energy (100kcal/kg/day) and protein (1 to 1.5 g/kg/day). When recovery sets in and appetite returns energy content may be increased to 150-220 kcal/kg/day and protein to 4 to 6 g/kg/day. It is also important to bear in mind that severe malnutrition is a global deficiency in which a large number of nutrients including vitamins and minerals need to be replenished. These are then the principles of treatment which can be followed wherever care is undertaken.

In emergency situations where a large number of children (and adults) have to be cared for with limited resources researchers have suggested classifying acute malnutrition for treatment purposes as follows:

Acute malnutrition complicated by infection (<80% of median weight for height; or presence of pitting oedema in both legs; or mid-arm circumference <110 mm; with presence of any of the following: anorexia; diarrhoea; hypothermia; clinical signs of respiratory infection; anaemia; excessive drowsiness); Group 1 Acute uncomplicated malnutrition (<70% median weight for height; or oedema; or mid-upper arm circumference <110 mm; appetite good; clinically well; alert); Group 2 Moderate uncomplicated malnutrition (70 – 80% median weight for height; no oedema; mid-upper arm circumference 110-125 mm; appetite good; clinically well; alert); Group 3.

According to this classification group 1 needs intensive inpatient stabilization care; Group 2 can be treated in the outpatients with therapeutic care (i.e. catch-up formula with gradual introduction of solid food and nutritional rehabilitation); and Group 3 can be helped with just supplementary feeding.

Community Centred approaches

As with all severe illnesses the best approach is prevention. A fresh look at the problem of childhood malnutrition and our methods of dealing with it is necessary. Traditionally we tend to think of malnutrition as any other disease in which the problem is at the family level and the management is also aimed at the level of the patient and his family. But malnutrition is a problem at the level of the community, and is the consequence of the prevailing state of cultural, social and technical development. A community which is able to feed its children is in a healthier state of development compared to one which cannot. One must therefore search for solutions within the community on the principle that "the state of nutrition is determined by what people do and not by what they get'. Amongst the community-based programmes are those for increasing productivity at the village level, preparation of weaning diets from local foods in the form of multimixes, child feeding programmes supported by community effort, identification of beneficiaries for community assistance by village health workers using simple techniques, and other similar activities. Many of these activities supplement the effects of services like the under-5s clinics described earlier, and together prepare the community for programmes of integrated development. Thus, in Hyderabad (India) the village community was mobilized to create farmers' groups who raised crops on communal plots; the women in turn processed these foods into a nourishing multimix, the village health workers identified children in need of food supplementation and all these activities were strongly supported by a network of community health activities. In Jamkhed (India) the community increases its agricultural potential by building earth dams across streams, and young farmers' clubs raise crops for a village feeding programme which is supervised by village health committees and operated by the village health workers.

These are some of the new approaches. But they are all in experimental stages and operating within defined boundaries. For them to spread both nationally and internationally a new breed of professional are needed who are capable of identifying the social, ecological and other root causes of nutritional disorders, and of working with the community to find solutions.

Monitoring the nutrition of the community

The health worker usually operates from the district hospital or health and the community to be served is defined administratively or by geographical boundaries. The first need is to get to know one's community well. It is important to obtain an ordnance map of the area and the latest census data. Other government departments like agriculture, community development, water supply, education and so on may have useful information about the area and the people. A study of annual reports of these departments supplemented with reports of any surveys carried out can provide information on land availability, agricultural productivity, literacy, rates, employment, local industry as well as local beliefs and practices. Meetings and discussions with local leaders and officials may also help to add further details about the community and its problems. The best source of information, however, can come through familiarizing oneself with the area and its people during community rounds.

Is malnutrition a problem in the community? Hospital admission figures and out-patient registers provide a useful clue to its presence in the community. When malnutrition is being commonly diagnosed in the health institutions it is important that the extent of the problem be measured in the community, always bearing in mind that most health institutions mainly serve the community in their immediate neighbourhood. Many cases of malnutrition, and especially the mild-moderate forms, may not be brought to the notice of the health workers, and hence it is useful to measure their prevalence in the community.

All health workers are not in the fortunate position of having the resources to carry out extensive surveys, but this should not prevent them from measuring the effectiveness of their activities at regular intervals. Several techniques of rapid rural assessment have been described recently and can be used to advantage by health workers in isolated rural areas with poor facilities and support.

Making a community diagnosis of malnutrition

Depending upon the resources available, weight-for-age, height-for-age, arm circumference and arm circumference-for-height (Quac Stick) may be used as parameters for measuring the growth and the nutritional status of children. Clearly it is not possible to measure all the children in the area and it may only be possible to measure the children in one or two villages at a time. However, if these villages are selected with forethought, a useful picture of the whole district can be put together over a couple of years. Many villages have a population of about 1000 persons, and often villages and hamlets are in clusters. The desirable minimum for statistical purposes is about 50 children under the age of 3 years per village. For truly useful information about the dietary pattern, the recommended number is 300 children under the age of 3 years. Depending upon the size of the village or of the residential area (e.g. squatter settlement), the number of households to be visited is indicated in Table 6.4. The information obtained through such surveys can be

Table 7.10 Number of children to be included in a survey of households for assessing Nutritional Status

No. of people in the settlement	No. of children in the settlement	No. of children for measuring	No. of houses in the settlement	Homes to visit
100	20	20	5-7	All houses
500	100	100	25-30	All houses
1000	200	200	50-70	All houses
2000	400	200	50-70	Every 2nd house
5000	1000	200	50-70	Every 5th house

augmented through lay reporting. Various members of the community, such as village health workers, school teachers, or school children can gather information using the arm band and/or the Quac Stick, as well as a simple questionnaire on infant feeding. In the case of the latter, as well as in general discussions with community leaders, it is useful to follow four areas of enquiry, viz:

(1) What do informants say people ought to do? (Assesses level of knowledge).

(2) What do informants say most people do? (Provides a measure of beliefs and practices).

(3) What do informants say they themselves do? (Gives a measure of common d dietary patterns).

(4) What do informants and others actually do? (This information is obtained through actual observations and recall type of enquiry).

The traditional birth attendants in the community are usually knowledgeable and can provide valuable information on family life patterns, dietary practices and attitudes, details of pregnancies as well as birth weights and birth intervals.

When all the information discussed above is assembled, it is possible to construct a nutrition profile of the community. The proportion of infants with low birth weight, percentage low weight for age, percentage with low arm circumference, percentage with low arm circumference for height, percentage low weight for height, birth intervals, high-risk pregnancies, family stability, customs and taboos, use of fats and oils in the dietaries and similar other information can be obtained about life patterns in the different settlements and communities of the district. Malnutrition is common in communities who do not receive adequate health and other services. Hence indicators of health coverage provide useful contributory information. Availability and acceptance of prenatal care, attendance at the under-5s clinics as judged by the presence of a weight chart at home and the regularity of weighing, coverage by immunization, local facilities for oral rehydration to treat diarrhoea and acceptance of family planning services serve as measures of the quality of health coverage of the community. Malnutrition is also the consequence of the prevailing state of cultural, social and technical development of the community as well as the sharing of resources. Hence socioeconomic indicators such as availability of safe water in adequate amounts, facilities for disposal of human waste, literacy rates, employment or availability of land, type of dwellings and so on help to assess the determinants of malnutrition in the community.

Malnutrition is said to be prevalent when community surveys reveal the following:

(1) the number of children with marasmus and those with oedema is greater than 20 per cent;

(2) the number of children with severe anaemia is greater than 5 per cent;

(3) the number of children under-weight exceeds 20 per cent;

(4) the number of children dying is greater than 20 per cent of total children born to mothers in the community.

Alleviation and eradication of hunger requires community action. Hence community awareness and interest are important. The sharing of all information with the community helps to create rapport and exchange of ideas. Several of the community programmes mentioned in the preceding section have active community participation as the primary objective. I n the sharing of information and exchange of ideas one may discover the beginnings of community-based interventions.

In all surveys and activities for monitoring the impact of services it is important to be aware of certain pitfalls. Those who are in greatest need are usually out of sight. It is the remote village away from the main road where the need is most acute. It is the disadvantaged in the community, such as for example, the lower castes or the single-parent families, whose voice is rarely heard in the dialogue with the community. Subjects like low wages, often below the statutory minimum wage, the iron grip of the big landlords, high interest rates and consequent indebtedness, and unemployment rarely get discussed in the open and need to be looked for. Interviews with the disadvantaged in small groups may provide useful information. The effects of the seasons must also be taken into account. If one has established a regular pattern of community or village rounds, then visiting the same place just before and soon after the wet season may provide useful insights. Finally, all surveys are snapshots and not trends. Hence one should be careful about drawing firm conclusions and making generalizations.

The objective of monitoring services and programmes is to bring about improvements in their effectiveness and efficiency. Technical innovations, policy decisions and administrative changes are all part of the process. A question often asked is, "How can health and nutrition interventions alter destitution and poverty? Surely political decisions and socioeconomic development are the answers!' This is true to a large extent, but health workers can contribute a great deal by slanting the services to seek out and help the neediest. Health services, like other national programmes in most countries, are largely monopolized by the elite. To be able to identify those at greatest risk and ensure their health and welfare will be one way to alleviate poverty; for to ensure freedom from illness and incapacity is to ensure availability for employment. To create self-ufficiency through locally available foods is to save scarce family resources. Community cohesion will ensure community support for the disadvantaged. To help promote functional literacy is to enable the disadvantaged to fend for themselves in a hostile world. Health and nutrition interventions hold the promise of becoming springboards for community upliftment.

FURTHER READING

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