Chapter 12. Protein-energy malnutrition

Protein-energy malnutrition (PEM) in young children is currently the most important nutritional problem in most countries in Asia, Latin America, the Near East and Africa. Energy deficiency is the major cause. No accurate figures exist on the world prevalence of PEM, but World Health Organization (WHO) estimates suggest that the prevalence of PEM in children under five years of age in developing countries has fallen progressively, from 42.6 percent in 1975 to 34.6 percent in 1995. However, in some regions this fall in percentage has not been as rapid as the rise in population; thus in some regions, such as Africa and South Asia, the number of malnourished children has in fact risen. In fact the number of underweight children worldwide has risen from 195 million in 1975 to an estimated 200 million at the end of 1994, which means that more than one-third of the world's under-five population is still malnourished.

Failure to grow adequately is the first and most important manifestation of PEM. It often results from consuming too little food, especially energy, and is frequently aggravated by infections. A child who manifests growth failure may be shorter in length or height or lighter in weight than expected for a child of his or her age, or may be thinner than expected for height.

The conceptual framework described in Chapter 1 suggests that there are three necessary conditions to prevent malnutrition or growth failure: adequate food availability and consumption; good health and access to medical care; and adequate care and feeding practices. If any one of these is absent, PEM is a likely outcome.

The term protein-energy malnutrition entered the medical literature fairly recently, but the condition has been known for many years. In earlier literature it was called by other names, including protein-calorie malnutrition (PCM) and protein-energy deficiency.

The term PEM is used to describe a broad array of clinical conditions ranging from the mild to the serious. At one end of the spectrum, mild PEM manifests itself mainly as poor physical growth in children; at the other end of the spectrum, kwashiorkor (characterized by the presence of oedema) and nutritional marasmus (characterized by severe wasting) have high case fatality rates.

It has been known for centuries that grossly inadequate food intake during famine and food shortages leads to weight loss and wasting and eventually to death from starvation. However, it was not until the 1930s that Cicely Williams, working in Ghana, described in detail the condition she termed "kwashiorkor" (using the local Ga word meaning "the disease of the displaced child"). In the 1950s kwashiorkor began to get a great deal of attention. It was often described as the most important form of malnutrition, and it was believed to be caused mainly by protein deficiency. The solution seemed to be to make more protein-rich foods available to children at risk. This stress on kwashiorkor and on protein led to a relative neglect of nutritional marasmus and adequate food and energy intakes for children.

The current view is that most PEM is the result of inadequate intake or poor utilization of food and energy, not a deficiency of one nutrient and not usually simply a lack of dietary protein. It has also been increasingly realized that infections contribute importantly to PEM. Nutritional marasmus is now recognized to be often more prevalent than kwashiorkor. It is unknown why a given child may develop one syndrome as opposed to the other, and it is now seen that these two serious clinical forms of PEM constitute only the small tip of the iceberg. In most populations studied in poor countries, the point prevalence rate for kwashiorkor and nutritional marasmus combined is 1 to 5 percent, whereas 30 to 70 percent of children up to five years of age manifest what is now termed mild or moderate PEM, diagnosed mainly on the basis of anthropometric measurements.

Causes and epidemiology
PEM, unlike the other important nutritional deficiency diseases, is a macronutrient deficiency, not a micronutrient deficiency. Although termed PEM, it is now generally accepted to stem in most cases from energy deficiency, often caused by insufficient food intake. Energy deficiency is more important and more common than protein deficiency. It is very often associated with infections and with micronutrient deficiencies. Inadequate care, for example infrequent feeding, may play a part.

The cause of PEM (and of some other deficiency diseases prevalent in developing countries) should not, however, be viewed simply in terms of inadequate intake of nutrients. For satisfactory nutrition, foods and the nutrients they contain must be available to the family in adequate quantity; the correct balance of foods and nutrients must be fed at the right intervals; the individual must have an appetite to consume the food; there must be proper digestion and absorption of the nutrients in the food; the metabolism of the person must be reasonably normal; and there should be no conditions that prevent body cells from utilizing the nutrients or that result in abnormal losses of nutrients. Factors that adversely influence any of these requisites can be causes of malnutrition, particularly PEM. The aetiology, therefore, can be complex. Certain factors that contribute to PEM, particularly in the young child, are related to the host, the agent (the diet) and the environment. The underlying causes could also be categorized as those related to the child's food security, health (including protection from infections and appropriate treatment of illness) and care, including maternal and family practices such as those related to frequency of feeding, breastfeeding and weaning.

Some examples of factors involved in the aetiology of PEM are:

- the young child's high needs for both energy and protein per kilogram relative to those of older family members;
- inappropriate weaning practices;
- inappropriate use of infant formula in place of breastfeeding for very young infants in poor families;
- staple diets that are often of low energy density (not infrequently bulky and unappetizing), low in protein and fat content and not fed frequently enough to children;
- inadequate or inappropriate child care because of, for example, time constraints for the mother or lack of knowledge regarding the importance of exclusive breastfeeding;
- inadequate availability of food for the family because of poverty, inequity or lack of sufficient arable land, and problems related to intrafamily food distribution;
- infections (viral, bacterial and parasitic) which may cause anorexia, reduce food intake, hinder nutrient absorption and utilization or result in nutrient losses;
- famine resulting from droughts, natural disasters, wars, civil disturbances, etc.

Prematurity or low birth weight may predispose the child to the development of nutritional marasmus. Failure of breastfeeding because of death of the mother, separation from the mother or lack of or insufficient breastmilk may be causes in poor societies where breastfeeding is often the only feasible way for mothers to feed their babies adequately. An underlying cause of PEM is any influence that prevents mothers from breastfeeding their newborn infants when they live in households where proper bottle-feeding may be difficult or hazardous. Therefore promotion of infant formula and insufficient support of breastfeeding by the medical profession and health services may be factors in the aetiology of marasmus. Prolonged exclusive breastfeeding without the introduction of other foods after six months of age may also contribute to growth faltering, PEM and eventually nutritional marasmus.
The view that kwashiorkor is the result of protein deficiency and nutritional marasmus the result of energy deficiency is an oversimplification, as the causes of both conditions are complex. Both endogenous and exogenous causes are likely to influence whether a child develops nutritional marasmus, kwashiorkor or the intermediate form known as marasmic kwashiorkor. In a child who consumes much less food than required for his or her energy needs, energy is mobilized from both body fat and muscle. Gluconeogenesis in the liver is enhanced, and there is loss of subcutaneous fat and wasting of muscles. It has been suggested that under these circumstances, especially when protein intake is very low relative to carbohydrate intake (with the situation perhaps aggravated by nitrogen losses from infections), various metabolic changes take place which contribute to the development of oedema. More sodium and more water are retained, and much of the water collects outside the cardiovascular system in the tissues, which results in pitting oedema. The actual role of infection has not been adequately explained, but certain infections cause major increases in urinary nitrogen, which derives from amino acids in muscle tissue.

There is not yet broad agreement on the actual cause of the oedema that is the hallmark of kwashiorkor. Most researchers agree that potassium deficiency and sodium retention are important in the pathogenesis of oedema. Some evidence supports the classical argument that oedematous malnutrition is a sign of inadequate protein intake. For example, oedema, fatty liver and a kwashiorkor-like condition can be induced in pigs and baboons on a protein-deficient diet. Epidemiological evidence also shows higher rates of kwashiorkor in Uganda, where the staple diet is plantain, which is very low in protein, than in neighbouring areas where the staple food is a cereal.

Recently two new theories have been advanced to explain the cause of kwashiorkor. The first is that kwashiorkor is due to aflatoxin poisoning. The second is that free radicals are important in the pathogenesis of kwashiorkor; it has been hypothesized that most of the clinical features of kwashiorkor could be caused by an excess free radical stress. This new, relatively untested theory also suggests, however, that kwashiorkor, even if produced by free radicals, is likely to occur only in children who have inadequate food intake and are subjected to infection. Thus even if this theory were to be proved correct, it would merely explain a mechanism for the pathogenesis of kwashiorkor. It would not change the fact that improving diet and reducing infection lead to significant reduction in both kwashiorkor and nutritional marasmus. Neither the aflatoxin nor the free radical theory has been proved experimentally, nor is there adequate convincing research to uphold the view of individual dysadaptation as the cause of severe PEM. Surprisingly, no studies have been able to give conclusive proof of either similarities or differences in dietary consumption between children who develop kwashiorkor with oedema and those who show clinical signs of nutritional marasmus without any oedema.

In severe PEM there is usually biochemical evidence, and often clinical evidence, of micronutrient deficiencies, which is not surprising in a child or adult who consumes a grossly inadequate diet. In both nutritional marasmus and kwashiorkor (and also in moderate PEM), clinical examinations or biochemical tests often give clear evidence of, for example, vitamin A deficiency, nutritional anaemia and/or zinc deficiency. However, there is little indication that any one micronutrient deficiency is the main cause of PEM or is by itself responsible for the oedema of kwashiorkor.

Irrespective of which theory of aetiology may be proved correct, improving the quantity of food consumed, taking steps to ensure that diets are nutritionally well balanced and controlling infection all help to prevent PEM.

Manifestations and clinical picture

Mild and moderate PEM

The condition of PEM is often likened to an iceberg, of which 20 percent is visible above the water and about 80 percent submerged. The severe forms of PEM - kwashiorkor, nutritional marasmus and marasmic kwashiorkor - constitute the top, exposed part of the iceberg: they are relatively easy for a doctor or health
worker to diagnose simply from their clinical manifestations, described below. On the other hand, children with moderate or mild malnutrition often do not have clear clinical manifestations of malnutrition; rather, they are shorter and/or thinner than would be expected for their age, and they may have deficits in psychological development and perhaps other signs not easy to detect. Mild and moderate PEM are diagnosed mainly on the basis of anthropometry, especially using measurements of weight and height and sometimes other measurements such as arm circumference or skinfold thickness.

As shown by the iceberg diagram (Figure 5), the prevalence of highly visible, serious PEM (kwashiorkor, marasmic kwashiorkor and nutritional marasmus) is usually between about 1 and 5 percent, except in famine areas. In contrast, moderate and mild malnutrition in many countries of sub-Saharan Africa and South Asia add up to 30 to 70 percent. In these areas often only 15 to 50 percent of young children between six months and 60 months of age do not have evidence of PEM. The diagram illustrates that both energy deficiency and protein deficiency play a part, but that energy deficiency is more important. It suggests that protein deficiency plays a greater part in kwashiorkor and energy deficiency in nutritional marasmus.

The percentage of children classified as having severe, moderate and mild PEM depends on how these terms are defined. The two severe forms of malnutrition, kwashiorkor and nutritional marasmus, have very different appearances and clinical features as described below. It is generally agreed that the hallmark of kwashiorkor is pitting oedema, and the overriding feature of nutritional marasmus is severe underweight. Children who have both oedema and severe underweight are diagnosed as having marasmic kwashiorkor.

FIGURE 5. PEM iceberg

The so-called Wellcome classification of severe forms of PEM has been widely used for over 20 years (see Table 19). It has the advantage of simplicity because it is based on only two measures, namely the percentage of standard weight for age and the presence or absence of oedema. The category "undernourished" includes children who have moderate or moderately severe PEM but no oedema and whose weight is above 60 percent of the standard. Today a cut-off point using standard deviations (SD) is considered more appropriate than percentage of standard, but not many children would be reclassified.
In the 1950s and 1960s the degree of malnutrition was almost always based on the child's percentage of standard weight for age. In Latin America and elsewhere the Gomez classification was very widely used (Table 20).

In the early 1970s a number of nutrition workers began to suggest that judging the degree of malnutrition only on the basis of weight for age had many disadvantages. A method was suggested that distinguished three categories of mild to moderate PEM based on weight and height measurements of children. Subsequently these categories came to be known as follows:

- **wasting**: acute current, short-duration malnutrition, where weight for age and weight for height are low but height for age is normal;

- **stunting**: past chronic malnutrition, where weight for age and height for age are low but weight for height is normal;

- **wasting and stunting**: acute and chronic or current long-duration malnutrition, where weight for age, height for age and weight for height are all low.

### TABLE 19

**Wellcome classification of severe forms of protein-energy malnutrition**

<table>
<thead>
<tr>
<th>Percentage of standard weight for age</th>
<th>Oedema present</th>
<th>Oedema absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>60-80</td>
<td>Kwashiorkor</td>
<td>Undernourishment</td>
</tr>
<tr>
<td>&lt;60</td>
<td>Marasmic kwashiorkor</td>
<td>Nutritional marasmus</td>
</tr>
</tbody>
</table>

### TABLE 20

**The Gomez classification of malnutrition based on weight-for-age standards**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Percentage of standard weight for age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&gt;90</td>
</tr>
<tr>
<td>Grade I (mild malnutrition)</td>
<td>75-89.9</td>
</tr>
<tr>
<td>Grade II (moderate malnutrition)</td>
<td>60-74.9</td>
</tr>
<tr>
<td>Grade III&lt;sup&gt;a&lt;/sup&gt; (severe malnutrition)</td>
<td>&lt;=60</td>
</tr>
</tbody>
</table>

<sup>a</sup> J. Bengoa of WHO suggested that all children with oedema be placed in Grade III. This became known as the Bengoa modification.

This classification makes a distinction between current and past influences on nutritional status. It helps the examiner assess the likelihood that supplementary feeding will markedly improve the nutritional status of the child, and it gives the clinician some clue as to the history of the malnutrition in the patient. It also has advantages for nutritional surveys and surveillance. In general, stunting is more prevalent than wasting worldwide.

As is discussed in Chapter 33, which deals with assessment of nutritional status, it is now generally recommended that malnutrition be judged on the basis of SD below the growth standards of the United States National Center for Health Statistics (NCHS) as published by WHO. In country reports published based on
weight for age alone, "underweight" is commonly used to denote weight below 2 SD of the NCHS standards in children up to five years of age. In a normal distribution it is expected that 2 to 3 percent of children will fall below the -2 SD cut-off point. Prevalence above that level suggests that there is a nutritional problem in the population assessed. If measurements are also taken of length or height, then the children can be further divided into those who are wasted, stunted, or wasted and stunted.

Policy-makers and health workers need to decide which growth standards to use as a yardstick for judging malnutrition and for surveys, monitoring and surveillance. In recent years the WHO/NCHS growth standards (which do not differ very much from previously used standards, such as the Harvard and Denver growth standards) have gained increasing acceptance. The international growth standards have been found to be applicable for developing countries, as evidence shows that the growth of privileged children in developing countries does not differ importantly from these standards, and that the poorer growth seen among the underprivileged results from social factors, including the malnutrition-infection complex, rather than from ethnic or geographic differences.

The functional significance of mild or moderate PEM is still not fully known. Studies from several countries show that the risk of mortality increases rather steadily with worsening nutritional status as indicated by anthropometric measures. Recent investigations in Guatemala indicated that teenagers who had manifested poor growth when examined in early childhood were smaller in stature, did less well at school, had poorer physical fitness and had lower scores on psychological development tests than children from the same villages who grew better as young children. These results suggest long-term consequences of PEM in early childhood.

The attempt to control the extent and severity of PEM using many different strategies and actions is at the heart of nutritional programmes and policies in most developing countries. The reduction and eventual prevention of mild or moderate malnutrition will automatically reduce severe malnutrition. Thus, although it may be tempting (particularly for doctors and other health workers) to put major emphasis on the control of nutritional marasmus and kwashiorkor, resources are often better spent on controlling mild and moderate PEM, which will in turn reduce severe PEM.

**Kwashiorkor**

Kwashiorkor is one of the serious forms of PEM. It is seen most frequently in children one to three years of age, but it may occur at any age. It is found in children who have a diet that is usually insufficient in energy and protein and often in other nutrients. Often the food provided to the child is mainly carbohydrate; it may be very bulky, and it may not be provided very frequently.

Kwashiorkor is often associated with, or even precipitated by, infectious diseases. Diarrhoea, respiratory infections, measles, whooping cough, intestinal parasites and other infections are common underlying causes of PEM and may precipitate children into either kwashiorkor or nutritional marasmus. These infections often result in loss of appetite, which is important as a cause of serious PEM. Infections, especially those resulting in fever, lead to an increased loss of nitrogen from the body which can only be replaced by protein in the diet.

**Clinical signs of kwashiorkor**

Kwashiorkor is relatively easy to diagnose based on the child's history, the symptoms reported and the clinical signs observed (Figure 6). Laboratory tests are not essential but do throw more light on each case. All cases of kwashiorkor have oedema to some degree, poor growth, wasting of muscles and fatty infiltration of the liver. Other signs include mental changes, abnormal hair, a typical dermatosis, anaemia, diarrhoea and often evidence of other micronutrient deficiencies.

**Oedema.** The accumulation of fluid in the tissues causes swelling; in kwashiorkor this condition is always present to some degree. It usually starts with a slight swelling of the feet and often spreads up the legs. Later,
the hands and face may also swell. To diagnose the presence of oedema the medical attendant presses with a finger or thumb above the ankle. If oedema is present the pit formed takes a few seconds to return to the level of the surrounding skin.

**Poor growth.** Growth failure always occurs. If the child's precise age is known, the child will be found to be shorter than normal and, except in cases of gross oedema, lighter in weight than normal (usually 60 to 80 percent of standard or below 2 SD). These signs may be obscured by oedema or ignorance of the child's age.

*FIGURE 6. Characteristics of kwashiorkor*

*Wasting.* Wasting of muscles is also typical but may not be evident because of oedema. The child's arms and legs are thin because of muscle wasting.

*Fatty infiltration of the liver.* This condition is always found in post-mortem examination of kwashiorkor cases. It may cause palpable enlargement of the liver (hepatomegaly).
**Mental changes.** Mental changes are common but not invariably noticed. The child is usually apathetic about his or her surroundings and irritable when moved or disturbed. The child prefers to remain in one position and is nearly always miserable and unsmiling. Appetite is nearly always poor.

**Hair changes.** The hair of a normal Asian, African or Latin American child is usually dark black and coarse in texture and has a healthy sheen that reflects light. In kwashiorkor, the hair becomes silkier and thinner. African hair loses its tight curl. At the same time it lacks lustre, is dull and lifeless and may change in colour to brown or reddish brown. Sometimes small tufts can be easily and almost painlessly plucked out. On examination under a microscope, plucked hair exhibits root changes and a narrower diameter than normal hair. The tensile strength of the hair is also reduced. In Latin America bands of discoloured hair are reported as a sign of kwashiorkor. These reddish-brown stripes have been termed the "flag sign" or "signa bandera".

**Skin changes.** Dermatosis develops in some but not all cases of kwashiorkor. It tends to occur first in areas of friction or of pressure such as the groin, behind the knees and at the elbow. Darkly pigmented patches appear, which may peel off or desquamate. The similarity of these patches to old sun-baked, blistered paint has given rise to the term "flaky-paint dermatosis". Underneath the flaking skin are atrophic depigmented areas which may resemble a healing burn.

**Anaemia.** Most cases have some degree of anaemia because of lack of the protein required to synthesize blood cells. Anaemia may be complicated by iron deficiency, malaria, hookworm, etc.

**Diarrhoea.** Stools are frequently loose and contain undigested particles of food. Sometimes they have an offensive smell or are watery or tinged with blood.

**Moonface.** The cheeks may appear to be swollen with either fatty tissue or fluid, giving the characteristic appearance known as "moonface".

**Signs of other deficiencies.** In kwashiorkor some subcutaneous fat is usually palpable, and the amount gives an indication of the degree of energy deficiency. Mouth and lip changes characteristic of vitamin B deficiency are common. Xerosis or xerophthalmia resulting from vitamin A deficiency may be seen. Deficiencies of zinc and other micronutrients may occur.

**Differential diagnosis**

**Nephrosis.** Oedema is also a feature of nephrosis, which may therefore be confused with kwashiorkor. In nephrosis, however, the urine contains much albumin as well as casts and cells. In kwashiorkor, there is usually only a trace of albumin. If flaky-paint dermatosis or other signs of kwashiorkor are present, the diagnosis is established. Ascites is frequently seen in nephrosis, but only rarely in kwashiorkor. In most developing countries kwashiorkor is a much more common cause of oedema than nephrosis.

**Severe hookworm anaemia.** Oedema may result from this cause alone. In young children kwashiorkor is often also present. In pure hookworm anaemia there are no skin changes other than pallor. In all cases the stools should be examined.

**Chronic dysentery.** In this disease oedema is not a feature.

**Pellagra.** Pellagra is rare in young children. The skin lesions are sometimes similar to those of kwashiorkor, but in pellagra they tend to be on areas exposed to sunlight(not the groin, for example). There may frequently be diarrhoea and weight loss, but no oedema or hair changes.

TABLE 21
Comparison of the features of kwashiorkor and marasmus

<table>
<thead>
<tr>
<th>Feature</th>
<th>Kwashiorkor</th>
<th>Marasmus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Growth failure</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Wasting</td>
<td>Present</td>
<td>Present, marked</td>
</tr>
<tr>
<td>Oedema</td>
<td>Present (sometimes mild)</td>
<td>Absent</td>
</tr>
<tr>
<td>Hair changes</td>
<td>Common</td>
<td>Less common</td>
</tr>
<tr>
<td>Mental changes</td>
<td>Very common</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Dermatosis, flaky-paint</td>
<td>Common</td>
<td>Does not occur</td>
</tr>
<tr>
<td>Appetite</td>
<td>Poor</td>
<td>Good</td>
</tr>
<tr>
<td>Anaemia</td>
<td>Severe (sometimes)</td>
<td>Present, less severe</td>
</tr>
<tr>
<td>Subcutaneous fat</td>
<td>Reduced but present</td>
<td>Absent</td>
</tr>
<tr>
<td>Face</td>
<td>May be oedematous</td>
<td>Drawn in, monkey-like</td>
</tr>
<tr>
<td>Fatty infiltration of liver</td>
<td>Present</td>
<td>Absent</td>
</tr>
</tbody>
</table>

Nutritional marasmus

In most countries marasmus, the other severe form of PEM, is now much more prevalent than kwashiorkor. In marasmus the main deficiency is one of food in general, and therefore also of energy. It may occur at any age, most commonly up to about three and a half years, but in contrast to kwashiorkor it is more common during the first year of life. Nutritional marasmus is in fact a form of starvation, and the possible underlying causes are numerous. For whatever reason, the child does not get adequate supplies of breastmilk or of any alternative food.

Perhaps the most important precipitating causes of marasmus are infectious and parasitic diseases of childhood. These include measles, whooping cough, diarrhoea, malaria and other parasitic diseases. Chronic infections such as tuberculosis may also lead to marasmus. Other common causes of marasmus are premature birth, mental deficiency and digestive upsets such as malabsorption or vomiting. A very common cause is early cessation of breastfeeding.

Clinical features of nutritional marasmus

The important features of kwashiorkor and nutritional marasmus are compared in Table 21. The following are the main signs of marasmus.

**Poor growth.** In all cases the child fails to grow properly. If the age is known, the weight will be found to be extremely low by normal standards (below 60 percent or -3 SD of the standard). In severe cases the loss of flesh is obvious: the ribs are prominent; the belly, in contrast to the rest of the body, may be protuberant; the face has a characteristic simian (monkey-like) appearance; and the limbs are very emaciated. The child appears to be skin and bones. An advanced case of the disease is unmistakable, and once seen is never forgotten.

**Wasting.** The muscles are always extremely wasted. There is little if any subcutaneous fat left. The skin hangs in wrinkles, especially around the buttocks and thighs. When the skin is taken between forefinger and thumb, the usual layer of adipose tissue is found to be absent.
**Alertness.** Children with marasmus are quite often not disinterested like those with kwashiorkor. Instead the deep sunken eyes have a rather wide-awake appearance. Similarly, the child may be less miserable and less irritable.

**Appetite.** The child often has a good appetite. In fact, like any starving being, the child may be ravenous. Children with marasmus often violently suck their hands or clothing or anything else available. Sometimes they make sucking noises.

**Anorexia.** Some children are anorexic.

**Diarrhoea.** Stools may be loose, but this is not a constant feature of the disease. Diarrhoea of an infective nature, as mentioned above, may commonly have been a precipitating factor.

**Anaemia.** Anaemia is usually present.

**Skin sores.** There may be pressure sores, but these are usually over bony prominences, not in areas of friction. In contrast to kwashiorkor, there is no oedema and no flaky-paint dermatosis in marasmus.

**Hair changes.** Changes similar to those in kwashiorkor can occur. There is more frequently a change of texture than of colour.

**Dehydration.** Although not a feature of the disease itself, dehydration is a frequent accompaniment of the disease; it results from severe diarrhoea (and sometimes vomiting).

**Marasmic kwashiorkor**

Children with features of both nutritional marasmus and kwashiorkor are diagnosed as having marasmic kwashiorkor. In the Wellcome classification (see above) this diagnosis is given for a child with severe malnutrition who is found to have both oedema and a weight for age below 60 percent of that expected for his or her age. Children with marasmic kwashiorkor have all the features of nutritional marasmus including severe wasting, lack of subcutaneous fat and poor growth, and in addition to oedema, which is always present, they may also have any of the features of kwashiorkor described above. There may be skin changes including flaky-paint dermatosis, hair changes, mental changes and hepatomegaly. Many of these children have diarrhoea.

**Laboratory tests**

Laboratory tests have a limited usefulness for the diagnosis or evaluation of PEM. Some biochemical estimations are used, and give different results for children with kwashiorkor and nutritional marasmus than for normal children or those with moderate PEM.

In kwashiorkor there is a reduction in total serum proteins, and especially in the albumin fraction. In nutritional marasmus the reduction is usually much less marked. Often, because of infections, the globulin fraction in the serum is normal or even raised. Serum albumin drops to low or very low levels usually only in clinically evident kwashiorkor. Serum albumin levels are not useful in predicting imminent kwashiorkor development in moderate PEM cases. It is often true that the more severe the kwashiorkor, the lower the serum albumin, but serum albumin levels are not useful in evaluating less severe PEM.

There is general agreement that serum albumin concentrations below 3 g/dl are low and that those below 2.5 g/dl are seriously deficient (see Table 22). It has also been suggested that serum albumin levels below 2.8 g/dl should be considered deficient and indicate a high risk.
TABLE 22

Levels of serum albumin concentrations in malnourished children

<table>
<thead>
<tr>
<th>Concentration (g/dl)</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥ 3.5</td>
<td>Normal</td>
</tr>
<tr>
<td>3.4-3.0</td>
<td>Subnormal</td>
</tr>
<tr>
<td>2.5-2.9</td>
<td>Low</td>
</tr>
<tr>
<td>&lt; 2.5</td>
<td>Pathological</td>
</tr>
</tbody>
</table>

Source: Alleyne et al., 1977.

Serum albumin determinations are relatively easy and cheap to perform, and unlike the other biochemical tests mentioned below, they can be done in modest laboratories in many developing countries.

Levels of two other serum proteins, pre-albumin and serum transferrin, are also of use and not too difficult to determine. Levels of both are reduced in kwashiorkor and may be useful in judging its severity. However, serum transferrin levels are also influenced by iron status, which reduces their usefulness as an indicator of kwashiorkor.

Levels of retinol binding protein (RBP), which is the carrier protein for retinol, also tend to be reduced in kwashiorkor and to a lesser degree in nutritional marasmus. However, other diseases, such as liver disease, vitamin A and zinc deficiencies and hyperthyroidism, may also influence RBP levels.

Other biochemical tests that have been used or recommended for diagnosing or evaluating PEM have limited usefulness. These include tests for:

- fasting serum insulin levels, which are elevated in kwashiorkor and low in marasmus;
- ratio of serum essential amino acids to non-essential amino acids, which is low in kwashiorkor but not much influenced by nutritional marasmus;
- hydroxyproline and creatinine levels in urine, which if low may indicate current growth deficits and nutritional marasmus.

These tests are not specific, and most cannot be performed in ordinary hospital laboratories.

Treatment of severe pem

Hospitalization

All children with severe kwashiorkor, nutritional marasmus or marasmic kwashiorkor should, if possible, be admitted to hospital with the mother. The child should be given a thorough clinical examination, including careful examination for any infection and a special search for respiratory infection such as pneumonia or tuberculosis. Stool, urine and blood tests (for haemoglobin and malaria parasites) should be performed. The child should be weighed and measured.
Often hospital treatment is not possible. In that case the best possible medical treatment available at a health centre, dispensary or other medical facility is necessary. If the child is still being breastfed, breastfeeding should continue.

**Diet.** Treatment is often based on dried skimmed milk (DSM) powder. DSM may most simply be reconstituted in hospital by adding one teaspoonful of DSM powder to 25 ml of boiled water and mixing thoroughly. The child should receive 150 ml of this mixture per kilogram of body weight per day, given in six feeds at approximately four-hour intervals. For example, a 5-kg child should receive $5 \times 150$ ml per day = 750 ml per day, divided into six feeds = 125 ml per feed. Each feed is made by adding five teaspoonfuls of DSM powder to 125 ml of water.

1 There is a risk if non-vitaminized DSM is used. Attention to providing all micronutrients is important.

The milk mixture should be fed to the child with a feeding cup or a spoon. If cup or spoon-feeding is difficult - which is possible if the child does not have sufficient appetite and is unable to cooperate or if the child is seriously ill the same mixture is best given through an intragastric tube. The tube should be made of polyethylene; it should be about 50 cm long and should have an internal diameter of 1 mm. It is passed through one nostril into the stomach. The protruding end should be secured to the cheek either with sticky tape or zinc oxide plaster. The tube can safely be left in position for five days. The milk mixture is best given as a continuous drip, as for a transfusion. Alternatively, the mixture can be administered intermittently using a large syringe and a needle that fits the tube. The milk mixture is then given in feeds at four-hour intervals. Before and after each feed, 5 ml of warm, previously boiled water should be injected through the lumen of the tube to prevent blockage.

There are better mixtures than plain DSM. They can all be administered in exactly the same way (by spoon, feeding cup or intragastric tube). Most of these mixtures contain a vegetable oil (e.g. sesame, cottonseed), casein (pure milk protein), DSM and sugar. The vegetable oil increases the energy content and energy density of the mixture and appears to be tolerated better than the fat of full cream milk. Casein increases the cost of the mixture, but as it often serves to reduce the length of the hospital stay, the money is well spent. A good and easily remembered formula for the sugar/casein/oil/milk (SCOM) mixture is: one part sugar, one part casein, one part oil and one part DSM, with water added to make 20 parts. A stock of the dry SCOM mixture can be stored for up to one month in a sealed tin. To make a feeding, the desired quantity of the mixture is placed in a measuring jug, and water is added to the correct level. Stirring or, better still, whisking will ensure an even mixture. As with the plain DSM mixture, 150 ml of liquid SCOM mixture should be given per kilogram of body weight per day; a 5-kg child should receive 750 ml per day in six 125-ml feeds, each made by adding five teaspoonfuls of SCOM mixture to 125 ml of boiled water. A 30-ml portion of made-up liquid feed provides about 28 kcal, 1 g protein and 12 mg potassium.

**Rehydration.** Children with kwashiorkor or nutritional marasmus who have severe diarrhoea or diarrhoea with vomiting may be dehydrated. Intravenous feeding is not necessary unless the vomiting is severe or the child refuses to take fluids orally. Rehydration should be achieved using standard oral rehydration solution (ORS), as is described for the treatment of diarrhoea (see Chapter 37). For severely malnourished children, unusually dilute ORS often provides some therapeutic advantage. Thus if standard ORS packets are used which are normally added to 1 litre of boiled water, in a serious case a packet might be added to 1.5 litres of water.

**Treatment of hypothermia.** Even in tropical areas temperatures at night often drop markedly in hospital wards and elsewhere. The seriously malnourished child has difficulty maintaining his or her temperature and may easily develop a lower than normal body temperature, termed hypothermia. Untreated hypothermia is a common cause of death in malnourished children. At home the child may have been kept warm sleeping in bed with the mother, or the windows of the house may have been kept closed. In the hospital ward the child may sleep alone, and the staff may keep the windows open. If the child's temperature is below 36°C, efforts must be made to warm the child. He or she must be kept in warm clothes and must be kept covered with warm bedding, and
there must be an effort to ensure that the room is adequately warm. Sometimes hot-water bottles in the bed are used. The child's temperature should be checked frequently.

**Medication.** Although it is useful to establish standard procedures for treating kwashiorkor and nutritional marasmus in any hospital or other health unit, each case should nevertheless be treated on its own merits. No two children have identical needs.

Infections are so common in severely malnourished children that antibiotics are often routinely recommended. Benzyl-penicillin by intramuscular injection, 1 million units per day in divided doses for five days, is often used. Ampicillin, 250 mg in tablet form four times a day by mouth, or amoxycillin, 125 mg three times a day by mouth, can also be given. Gentamycin and chloramphenicol are alternative options but are less often used.

In areas where malaria is present an antimalarial is desirable, e.g. half a tablet (125 mg) of chloroquine daily for three days, then half a tablet weekly. In severe cases and when vomiting is present, chloroquine should be given by injection.

If anaemia is very severe it should be treated by blood transfusion, which should be followed by ferrous sulphate mixture or tablets given three times daily.

If a stool examination reveals the presence of hookworm, roundworm or other intestinal parasites, then an appropriate anthelmintic drug such as albendazole should be given after the general condition of the child has improved.

Severely malnourished children not infrequently have tuberculosis and should be examined for it. If the disease is found to be present, specific treatment is needed.

**Recovery**

On the above regime, a child with serious kwashiorkor would usually begin to lose oedema during the first three to seven days, with consequent loss in weight. During this period, the diarrhoea should ease or cease, the child should become more cheerful and alert, and skin lesions should begin to clear.

When the diarrhoea has stopped, the oedema has disappeared and the appetite has returned, it is desirable to stop tube-feeding if this method has been used. The same SCOM or plain DSM mixture can be continued with a cup and spoon or feeding bowl. A bottle and teat should not be used. If anaemia is still present, the child should now start a course of iron by mouth, and half a tablet (125 mg) of chloroquine should be given weekly.

Children with severe nutritional marasmus may consume very high amounts of energy, and weight gain may be quite rapid. However, the length of time needed in hospital or for full recovery may be longer than for children with kwashiorkor.

In both conditions, as recovery continues, usually during the second week in hospital, the patient gains weight. While feeding of milk is continued, a mixed diet should gradually be introduced, aimed at providing the energy, protein, minerals and vitamins needed by the child.

If the disease is not to recur, it is important that the mother or guardian participate in the feeding at this stage. She must be told what the child is being fed and why. Her cooperation with and follow-up of this regime is much more likely if the hospital diet of the child is based mainly on products that are used at home and that are likely to be available to the family. This is not feasible in every case in a large hospital, but the diet should at least be based on locally available foods. Thus in a maize-eating area, for example, the child would now receive maize gruel with DSM added. For an older child, crushed groundnuts can be added twice a day, or, if preferred by custom, roasted groundnuts can be eaten. A few teaspoonfuls of ripe papaya, mango, orange or other fruit
can be given. At one or two meals per day, a small portion of the green vegetable and the beans, fish or meat that the mother eats can be fed to the child, after having been well chopped. Protein-rich foods (e.g. beans, peas, groundnuts, meat, sour milk or eggs) can be given. If eggs are available and custom allows their consumption, an egg can be boiled or scrambled for the child; the mother can watch as it is prepared. Alternatively, a raw egg can be broken into some simmering gruel. Protein-rich foods of animal origin are often relatively expensive. They are not essential; a good mixture of cereals, legumes and vegetables serves just as well. If suitable vitamin-containing foods are not available, then a vitamin mixture should be given, because the DSM and SCOM mixtures are not rich in vitamins.

The above maize-based diet is just an example. If the diet of the area is based on rice or wheat, these can be used instead of maize. If the staple food is plantain or cassava, then protein-rich supplements are important.

After discharge, or if a moderate case of kwashiorkor has been treated at home and not in the hospital, the child should be followed if possible in the out-patient department or a clinic. It is much better if such cases can visit separately from other patients (i.e. on a particular afternoon or at a child welfare or growth monitoring clinic) to avoid the tumult of most out patient sessions. A relaxed atmosphere is desirable, and the medical attendant should have time to explain matters to the mother and to see that she understands what is expected of her. It is useless just to hand over a bag of milk powder or other supplement, or simply to weigh the child but not provide simple guidance.

Satisfactory weight gain is a good measure of progress. At each visit the child should be weighed. Weight is plotted on a chart to provide a picture for the health worker and the mother.

Out-patient treatment should be based on the provision of a suitable dietary supplement, but in most cases it is best that this supplement be given as part of the diet. The mother should be shown a teaspoon and told how many teaspoonfuls to give per day based on the child's weight. Many supplements, especially DSM, are best provided by adding them to the child's usual food (such as cereal gruel) rather than by making a separate preparation. The mother should be asked how many times a day she feeds the child. If he or she is fed only at family mealtimes and the family eats only twice a day, then the mother should be told to feed the child two extra times.

If facilities exist and it is feasible, the SCOM mixture can be used for out-patient treatment. It is best provided ready mixed in sealed polyethylene bags.

**Prognosis**

Most deaths in children hospitalized for kwashiorkor or nutritional marasmus occur in the first three days after admission. Case fatality rates depend on many factors including the seriousness of the child's illness at the time of admission and the adequacy of the treatment given. In some societies sick children are taken to hospital very late in the disease, when they are almost moribund. In this situation fatality rates are high.

The cause and the severity of the disease determine the prognosis. A child with severe marasmus and lungs grossly damaged by tuberculous infection obviously has poor prospects. The prospects of a child with mild marasmus and no other infection are better. Response to treatment is likely to be slower with marasmus than with kwashiorkor.

It is often difficult to know what to do when the child is cured, especially if the child is under one year of age. There may be no mother or she may be ill, or she may have insufficient or no breastmilk. Instruction and nutrition education are vital for the person who will be responsible for the child. If the child has been brought by the father, then some female relative should spend a few days in the hospital before the child is discharged. She should be instructed in feeding with a spoon or cup and told not to feed the child from a bottle unless he or she is under three months of age. The best procedure is usually to provide a thin gruel made from the local
staple food plus two teaspoonfuls of DSM (or some other protein-rich supplement) and two teaspoonfuls of oil per kilogram of body weight per day. Instruction regarding other items in the diet must be given if the child is over six months old. The mother or guardian should be advised to attend the hospital or clinic at weekly intervals if the family lives near enough (within about 10 km) or at monthly intervals if the distance is greater. Supplies of a suitable supplement to last for slightly longer than the interval between visits should be given at each visit. The child can be put on other foods, as mentioned in the discussion of infant feeding in Chapter 6.

It is essential that the diet provide adequate energy and protein. Usually 120 kcal and 3 g of protein per kilogram of body weight per day are sufficient for long-term treatment. Thus a 10-kg child should receive about 1200 kcal and 30 g of protein daily. It should be noted that a marasmic child during the early part of recovery may be capable of consuming and utilizing 150 to 200 kcal and 4 to 5 g of protein per kilogram of body weight per day.

**Protein-energy malnutrition in adults**

**Adult kwashiorkor**

There is little doubt that a disorder due mainly to energy deficiency does occur in adults; it is more common in communities suffering from chronic protein deficiency. The patient is markedly underweight for his or her height (unless grossly oedematous), the muscles are wasted, and subcutaneous fat is reduced. Mental changes are common: the patient is usually disinterested and appears to be in a dream world. It is difficult to attract the patient's attention and equally hard to keep it. Appetite is reduced, and the patient is very weak.

Some degree of oedema is nearly always present, and this may mask the weight loss, wasting and lack of subcutaneous fat. Oedema is most common in the legs, and in male patients also in the scrotum, but any part of the body may be affected. The face is often puffy. This condition has been termed "famine oedema" because it occurs where there is starvation resulting from famine or other causes. It was commonly reported in famines in Indonesia and Papua New Guinea.

Frequent, loose, offensive stools may be passed. The abdomen is often slightly distended, and on palpation the organs can be very easily felt through the thin abdominal wall. During palpation there is nearly always a gurgling noise from the abdomen, and peristaltic movements can often be detected with the fingertips. It is not uncommon for adult kwashiorkor patients to regard their physical state as a consequence of abdominal upset. For this reason, strong purgatives, either proprietary or herbal, and peppery enemas are sometimes used by these patients before they reach hospital, which may greatly aggravate the condition.

The hair frequently shows changes. The skin is often dry and scaly, and may have a crazy-pavement appearance, especially over the tibia. Swelling of both parotid glands is frequent. On palpation the glands are found to be firm and rubbery.

Anaemia is nearly always present and may be severe. The blood pressure is low. There is usually only a trace of albumin in the urine.

Oedema may also be caused by severe anaemia. In adult PEM there is less dyspnoea than in anaemia and usually no cardiomegaly. Other features such as hair changes and parotid swelling are common in adult PEM but not in anaemia. However, the two conditions are closely related.

**Nutritional marasmus in adults**

In contrast to adult kwashiorkor or famine oedema, which is not very prevalent, the adult equivalent of nutritional marasmus is very common. There are five major causes.
Insufficient food. Any older child or adult whose diet is grossly deficient in energy will develop signs almost exactly like those of nutritional marasmus, and if the condition progresses it may often be fatal. In the case of famines, the condition may be termed starvation (see Chapter 24). Famines and severe food shortages resulting from war, civil disturbance or natural disasters such as droughts, floods and earthquakes may result in nutritional marasmus in children and a similar condition in adults, who suffer from weight loss, wasting, diarrhoea, infectious diseases, etc.

Infections. The second major cause of severe wasting or severe PEM in adults is infections, especially chronic, untreated or untreatable infections. The most common of these now is acquired immunodeficiency syndrome (AIDS) resulting from infection with the human immunodeficiency virus (HIV). As the disease progresses there is marked weight loss and severe wasting. As mentioned in Chapter 3, in Uganda the name "slim disease" is given to AIDS because of the thinness of its victims. Advanced tuberculosis and many other long-term chronic infections also lead to wasting and weight loss.

Malabsorption. A number of malabsorption conditions cause PEM in adults and children. These diseases, of which some are hereditary, result in the inabillity of the body to digest or absorb certain foods or nutrients. Examples are cystic fibrosis, coeliac disease and adult sprue.

Malignancies. Another cause of wasting in people of any age is malignancy or cancer of any organ once it progresses to a stage not treatable by surgical excision. Cachexia is a feature of many advanced cancers.

Eating disorders. A group of eating disorders cause weight loss leading to the equivalent of PEM. The most widely described is anorexia nervosa, which occurs much more commonly in females than males, in adolescents or younger adults rather than older persons and in affluent rather than poor societies. Other psychological conditions may also result in poor food intake and lead to PEM.

Treatment

Treatment of adult PEM includes therapy related to the underlying cause of the condition and therapy related to feeding and rehabilitation, when the cause makes that feasible. Thus infections such as tuberculosis or chronic amoebiasis require specific therapy which when effective will eliminate the cause of the weight loss and wasting. In contrast, curative treatment is not applicable in advanced AIDS or cancer.

Dietary treatment for adult PEM should be based on principles similar to those described for the treatment of severe PEM in children, including those recovering from kwashiorkor or marasmus. Emergency feeding and the rehabilitation of famine victims (described in Chapter 24) have relevance to adult PEM.

Prevention and control of PEM

The prevention of PEM in Asia, Africa and the Americas presents a huge challenge. It is much more difficult than controlling, for example, iodine deficiency disorders (IDD) and vitamin A deficiency, because the underlying and basic causes, as described above, are often numerous and complex, and because there is no single, universal, cheap, sustainable strategy that can be applied everywhere to reduce the prevalence or severity of PEM.

Part V of this book includes various strategies to reduce the prevalence of PEM. Appropriate nutrition policies and programmes are suggested, and separate chapters deal with, for example, improving food security, protection and promotion of good health, and appropriate care practices to ensure good nutrition. These chapters provide guidance on how to deal with the three underlying causes of malnutrition, namely inadequate food, health and care, which in Chapter 1 were included in the conceptual framework for malnutrition. Other chapters in Part V discuss solutions to particular aspects of the problem, including improving the quality and safety of foods, promoting appropriate diets and healthy lifestyles, procuring food in different ways and incorporating
nutrition objectives into development policies and programmes. Throughout Part V there is an emphasis on improving the quality of life of people, especially by reducing poverty, improving diets and promoting good health. Improving the energy intakes of those at risk of PEM is vital.

In the late 1950s and 1960s it was thought that most PEM was caused mainly by inadequate intake of protein. A great deal of emphasis was placed on protein-rich foods as a major solution to the huge problem of malnutrition in the world. This inappropriate strategy diverted attention from the first need, which is adequate food intake by children. There is now much less emphasis on high-protein weaning foods and on nutrition education efforts to ensure greater consumption of meat, fish and eggs, which are economically out of the reach of many families who have children with PEM.

Protein is an essential nutrient, but PEM is more often associated with deficient food intake than with deficient protein intake. In general, when commonly consumed cereal-based diets meet energy needs, they usually also meet protein needs, especially if the diet also provides modest amounts of legumes and vegetables. Primary attention needs to be given to increasing total food intake and reducing infection.

Sensible efforts are needed to protect and promote breastfeeding and sound weaning; to increase the consumption by young children of cereals, legumes and other locally produced weaning foods; to prevent and control infection and parasitic disease; to increase meal frequency for children; and, where appropriate, to encourage higher consumption of oil, fat and other items that reduce bulk and increase the energy density of foods fed to children at risk. These measures are likely to have more impact if accompanied by growth monitoring, immunization, oral rehydration therapy for diarrhoea, early treatment of common diseases, regular deworming and attention to the underlying causes of PEM such as poverty and inequity. Some of these measures can be implemented as part of primary health care. Readers planning strategies to control PEM should consult Part V of this book.