



---

## REVIEW ARTICLE

---

# *Malnutrition: a secular challenge to child nutrition*

Cristina M.G. Monte\*

### Abstract

**Objective:** to review current knowledge about child malnutrition, including the historical aspects of the problem, its dimension as a childhood public health problem, its natural history, physiopathology, clinical features, diagnosis and treatment, and strategies used by the health sector to control this disease.

**Methods:** information was collected by researching the Medline system, the Bireme library, internet sites of interest, catalogues of publications produced by Brazilian governmental organizations and international institutions dealing with child nutrition.

**Results:** the review pointed out that despite recent world prevalence reduction, child malnutrition is a major public health problem in developing countries. Malnutrition, in any of its forms, contributes for more than 50% of deaths among children under 5 years in those countries. Mortality rates of severely malnourished children treated as in patients have been unchanged for the last five decades. Guidelines for improving the treatment and reducing mortality rates of severely malnourished children treated in hospitals were recently defined by the World Health Organization. Even though some positive results have been achieved by the health sector in reducing child malnutrition prevalence, the effectivity of the interventions is often low. Lack of food might limit the success in treating and preventing malnutrition. Factors that may contribute to the effectiveness of interventions against malnutrition include approaches which reassure the confidence of health professionals about achieving positive results with the proper treatment of malnourished children, establishment of an effective relationship between health professionals and mothers, as well as practical support to mothers in recognizing them as valuable active agents for their children nutrition rehabilitation at the household level.

**Conclusions:** throughout the centuries, malnutrition has been the biggest challenge faced by developing countries in order to guarantee to children under five years of age their right of being well nourished and healthy. The current challenge is the proper use of the available scientific knowledge on child nutrition to further reduce the figures for all the types of child malnutrition.

*J. pediatr. (Rio J.). 2000; 76 (Supl.3): S285-S297: nutrition disorders, deficiency diseases, nutrition.*

### The problem of child deficiency-related malnutrition

Even though the inalienable right of any human being not to starve to death was reaffirmed over and over again at the meetings of UN member countries in 1948( Universal Declaration of Human Rights), in 1974(UN World Conference on Feeding) in 1978(International Pact for Economic Rights and World Health Organization

Declaration on "Health for All in the 21st century"), in 1989 (Convention on Children's Rights),<sup>1</sup> and recently regarded by the World Health Organization as a human right,<sup>2</sup> child malnutrition is still one of the major public health problems today due to its magnitude and disastrous consequences on children's development and survival. This article reviews the existing knowledge about child malnutrition and the strategies used for its control.

---

\* PhD in Human Nutrition, London School of Hygiene and Tropical Medicine.

The World Health Organization estimates that, every year, more than 20 million children present low weight at birth<sup>2</sup> and that approximately 150 million children younger than 5 years have low weight patterns for their age,<sup>3</sup> and that 182 million (32.5%) have low height.<sup>2</sup> Authors affirm that these values, however, may be underestimated as it is difficult to calculate world prevalence rates accurately.<sup>4</sup> Relatively small changes in the limit cut-off points of anthropometric indicators used for the classification of nutritional status, and consequently, for the definition of the disease, may imply variations amounting to millions of undernourished children who supposedly suffer from malnutrition.<sup>4,5</sup>

Malnutrition is the second most frequent cause of death in children under the age of 5 in developing countries.<sup>6</sup> Pelletier et al.,<sup>7</sup> attribute 56% of child mortality to malnutrition, due to the potential effects of mild and moderate forms of the disease.

Around 20 to 30% of children with severe malnutrition die when submitted to health services in developing countries.<sup>8</sup> These rates have remained unchanged for the last 5 decades, and correspond to a percentage 4 to 6 times higher than 5%, rate regarded as acceptable by the World Health Organization.<sup>9</sup>

A recent survey carried out in 79 hospitals around the world revealed that many health professionals have outdated ideas about or are totally unaware of the procedures involved in the treatment of children with severe malnutrition.<sup>8</sup>

Inadequate treatment often results from the incorrect perception of altered physiological state and of the reduced homeostatic mechanisms that characterize malnutrition.<sup>9,10</sup> Procedures include inadequate rehydration, causing heart strain and failure; imperception of infections that lead to septicemia; and lack of perception as to the vulnerability of children with severe malnutrition to hypothermia and hypoglycemia. Rehabilitation is too slow in most treatment centers or clinics.<sup>8</sup>

In some health centers, professionals have the necessary knowledge but the resources are limited.<sup>11</sup> In some others, the update on or the improvement of procedures through some slight changes may bring substantial benefits. This was observed in Bolivia, by adding zinc to the treatment of undernourished children.<sup>12</sup> In Brazil, decreased intake of lactose at the beginning of the treatment, increased energy intake during the growth enhancement stage, and supplementation with potassium, magnesium, zinc and copper reduced the incidence of diarrhea, allowed a fourfold increase in weight index, and considerably reduced treatment costs.<sup>13</sup> In South Africa, the simple use of micronutrients reduced mortality rate from 30 to 20%; however, when the intensity and quality of child care were enhanced, mortality rate dropped to 6%.<sup>14</sup>

In Brazil,<sup>15</sup> as in most developing countries,<sup>2</sup> the nutritional status of children under 5 years has changed for the better in the last few years due to extraordinary economic

development and the expansion of health services and programs.<sup>15</sup> Between 1975 and 1989, the prevalence of malnutrition fell approximately 60%, which is equivalent to one million children. Nevertheless, the fact that chronic child malnutrition nowadays is extremely frequent, shown mainly by reduced height for age standards, the percentage of children with severe malnutrition (although not very high), in addition to a high concentration of these children in the North and Northeast (country's poorest regions), indicates that the problem is not completely under control.<sup>16</sup> It is common knowledge that severe child malnutrition cases, even in small numbers, are just the tip of the iceberg. For each severe case, there are others that are less severe, and that sometimes show no clinical signs of malnutrition. Mild and moderate malnutrition is sometimes only observed in children who fail to thrive.<sup>5</sup>

### The secular challenge of malnutrition

The literature shows that physicians in the 19th and early 20th century already admitted that starvation, derived from low food intake, provoked growth retardation in children.<sup>4</sup> Several authors, also from Mexico and Chile, described different aspects of the disease from the 1800s onwards, and drew attention to the fact that extreme emaciation or nutritional edema caused by the disease could lead to child mortality.<sup>5</sup> However, the description of the disease as syndrome and the first denomination that configured its existence took place in the early 1930s. Williams<sup>17</sup> described Kwashiorkor, which was much more visible than child emaciation (marasmus). In Brazil, the disease was first registered in the 1950s.<sup>5</sup>

Once described, child malnutrition was recognized as a problem of medical nature that also included problems related to vitamin deficiency such as beriberi, pellagra, xerophthalmia and scurvy. The cure for the disease, found in the 1950s, widespread in Africa, Central America and Brazil, was attributed to the intake of high-protein foods.<sup>18</sup> Protein intake requirements were increased by FAO experts in 1973,<sup>19</sup> and the production of complementary high-protein foods was then stimulated. This was the "Age of Protein Science"

On the other hand, still in the 70's, the so-called "great protein fiasco" took place.<sup>20</sup> Nutritional surveys extensively carried out by FAO showed that, in all studied countries, the diet met protein requirements but did not provide the energy needs that had been preconized by consultants from the same organization, in 1973.<sup>21</sup> Since then, it is known that protein-energy malnutrition results mainly from energy deficiency. Distribution, access of poor people to food, was identified as the crucial factor. Poverty was considered to be the major cause of malnutrition.<sup>22,23</sup> The subsequent discovery of physiopathological aspects of the disease and the discovery of the synergistic relationship and association between malnutrition and infection<sup>24</sup> added to previous findings.

Malnutrition, due to its multifactorial nature, began to be regarded as a social problem and not a merely public health issue. This way, malnutrition, once within the realms of medical and health sciences, moved into the domain of technical and bureaucratic planning. This was the time of the so-called “theory of intersectorial nutrition planning”, which took for granted that the nutritional problems in underdeveloped countries would be overcome through planning and rational resource allocation. However, the theory failed, getting in the way of the implementation of nutrition policies and discouraging the effective action of health professionals.<sup>25</sup>

Nutrition planning can hardly benefit poor people as an anonymous Hindu<sup>10</sup> wittily remarked in his complaint about how discouraged he felt with the food donation policy:

*“I was hungry and you set up a committee to investigate my hunger;*

*I was homeless and you filled out a form with my complaint;*

*I was sick and you organized a debate on poor people’s nutrition;*

*You looked into all the aspects of my complaint and, still, I am hungry, homeless and sick”.*

In the 80’s, the participation of the health sector was clearly involved in the fight against child malnutrition once again, through the policy of “health for all the people of the world by the year 2000”, set up by Alma-Ata International Conference.<sup>26</sup>

### **The natural history of child malnutrition**

The challenge of maintaining good nutrition standards is present throughout life.<sup>2</sup> Malnutrition usually begins in the mother’s womb, affects children, and by means of girls and women, extends into adulthood and then from a generation to the next, through its negative cumulative effect on low infant weight. Low-weight babies who have intrauterine growth retardation are born with malnutrition and are at greater risk of death than normal babies. If they survive, it is unlikely that they have catch-up growth, and they will probably present a series of growth deficiencies. In addition, current evidence indicates that low weight at birth is associated with a greater risk for chronic diseases in adulthood.<sup>2</sup>

In childhood, frequent and prolonged infections, and inadequate intake of nutrients, especially energy, protein, vitamin A, zinc and iron, exacerbate the effects of intrauterine growth retardation. Fail-to-thrive occurs quickly up to the age of 2, resulting in low weight and height. It is known that the major causes of inadequate growth today in developing countries are nutritional deficiencies and infections, usually combined.<sup>2</sup>

Presently, from the point of view of natural history of the disease, there are two types of child growth deficiency: emaciation and low height.<sup>5</sup> Emaciation is defined by the weight for height, and 1 level of thinness below -2DP of the NCHS/WHO average reference standard is regarded as pathological. Low height is defined by the height for age, and when below -2SD of the NCHS/WHO average reference standard, it is called “stunting”. These two types of deficiency represent distinct biological processes. Although they can be found in the same child, they are, actually, statistically independent and their respective prevalence rates, as recommended by the World Health Organization, are presented all over the world in a separate way.<sup>5</sup>

The peak of emaciation prevalence occurs in the second year of life, coinciding with the initiation of complementary feeding and a high incidence of diarrhea.<sup>21</sup> The cause seems to be extremely direct: inadequate diet (quantitatively and qualitatively) and a high incidence of diarrheal diseases. Once the infectious episode is under control, and there is the possibility of feeding, children recover weight. The necessary amount of food necessary for growth enhancement is not very high and the diet needs to be rich in energy and proteins.<sup>21,27</sup>

Low height or “stunting” has a natural history that is different from that of emaciation. Typically, decreased linear growth has its onset around the third month of life and continues for 2 or 3 years, as demonstrated by studies carried out in Guatemala and India. The growth rate is just restored around the fifth year of life, when height deficiency may be approximately 15 cm in comparison with a normal child; and kept during adult life. Between 5 and 18 years, the growth rate is normal, but there is no growth enhancement.<sup>5</sup>

Recent research suggests that stunting originates from a reduction in the frequency of growth events in children or from a reduction in growth intervals when an event occurs.<sup>28,29</sup> If children affected with stunting continue to live in the same conditions they were living at the time they developed the disease, they will not have enough growth enhancement to measure up to their genetic potential. However, some studies show that it is possible to reverse stunting when living conditions are changed for the better, as observed in South Africa, Jamaica and Peru.<sup>29</sup> Therefore, the presence of stunting is a “proxy” indicator of a multifaceted deprivation and its prevalence is currently considered an appropriate quality-of-life indicator of a given population.<sup>28</sup> The important aspect of low height is its relation to the development of mental retardation in young children, which is not observed in emaciation alone. Even if children are able to perfectly recover their rate of retarded linear physical growth, the effects on mental development are more long-lasting.<sup>5,29,31</sup>

The role of infection is very important for the development and survival of children who suffer from malnutrition. Pelletier<sup>7</sup> convincingly argues that the effects of malnutrition and infection, even in its mild and moderate

forms, are not cumulative but multiplicative. In this analysis, there is no distinction between emaciation and stunting.

### **Child malnutrition and its causes**

*Child malnutrition* is a multicausal, complex disease whose roots are established in poverty-stricken communities. Its onset occurs when the body does not receive enough nutrients for its physiological metabolism due to the lack of adequate structure or waste of nutrients. Thus, in most cases, malnutrition is the result of insufficient feeding or hunger, and diseases.<sup>2</sup>

There are a considerable number of studies and publications in the literature related to malnutrition, its causes and effects.<sup>10,30,36</sup>

Several causal models have been proposed by a varied number of authors and organizations to explain the origin of malnutrition.<sup>10,37,38</sup> Causal schemes have also been proposed in an attempt to hierarchize the importance of causal factors,<sup>10</sup> by presenting a more comprehensive scheme that includes child care and preoccupation with his/her living conditions,<sup>38</sup> and by introducing the relevance of mother-child relationship as vital element in the genesis of malnutrition.<sup>39</sup> The World Health Organization<sup>2</sup> suggests that the "food-health-care" scheme, presented by UNICEF<sup>38</sup> be used as a tool to analyze the interaction of the several determinants of malnutrition in distinct social levels. In this scheme, malnutrition is shown as the result of an inadequate diet and diseases that originate from the lack of nutritional security, mother's improper care, and inefficient health services. The basic causes that contribute to these factors are social structures and organizations, political and ideological systems, distribution of richness and potential resources.<sup>38</sup>

Among the factors that contribute to malnutrition in young children we find relatively higher energy and protein requirements in comparison with other family members; low energy content of supplemental foods which are used and insufficiently fed to the children; inappropriate availability of foods because of poverty, social inequality, landlessness, and intrafamilial distribution problems; recurrent viral, bacterial and parasitic infections that could cause anorexia and reduce the intake, absorption and use of nutrients, or cause their loss; hunger caused by droughts or other natural disasters or wars; inadequate child care such as feeding of foods that are too diluted and/or not hygienically prepared.<sup>40,41</sup>

In marasmus, the frequent clinical form of malnutrition in most developing countries, especially in children under 18 months, all the factors described above may be present. There is no doubt that inadequate food intake, mainly insufficient energy to fulfill metabolic and normal growth needs, plays an important role in malnutrition.<sup>4</sup>

Prematurity and low weight at birth are predispositional causes.<sup>2</sup> Other factors that contribute to the disease include

non-breast-feeding and delayed supplementation of appropriate foods, and lack of adequate support to health professionals when counseling mothers. Mothers, due to scarce financial resources and/or little knowledge about health and child nutrition, often use hyperdiluted formulas, prepared in non-hygienic conditions, and sometimes stored at room temperature for a long time.<sup>40,41</sup>

The causes of Kwashiorkor are complex. A child who suffers from kwashiorkor usually has a diet that is poor in energy and proteins.<sup>5</sup> Infections play an important role. Some authors argue that when protein intake is too low, compared to the intake of carbohydrates, which could be aggravated by the loss of nitrogen caused by infections, several metabolic changes occur and can lead to edema.<sup>4,5</sup> Other authors state that kwashiorkor is a hormonal disadaptation to protein deficiency. Some others attribute the formation of edema to endogenous mechanisms related to free radicals.<sup>32</sup> Therefore, there is not a common ground on the etiology of kwashiorkor.

Hunger and diseases may be caused by different factors, or a long sequence of interconnected events. This way, it is difficult to draw conclusions on the determinant factors of malnutrition in a given population.<sup>10</sup> Most recent conclusions on the causes of child malnutrition result from cross-studies and evidence obtained from health interventions.<sup>10</sup> Randomly-controlled studies are not suitable for investigating the origins of malnutrition.<sup>37</sup> Poverty is not homogenous and socioeconomic variables, for instance, cannot be tested through randomly-controlled studies.<sup>37</sup> Another difficulty is the nature of the variables themselves. Family income and education, for example, considered as determinant factors in child nutritional status, may vary with time. In addition, these data are not easily collected. Therefore, it is difficult to investigate the relationship with nutritional status in time either through long-term cross-studies or prospective studies.<sup>37</sup>

### **Physiopathology of child severe malnutrition**

Malnutrition affects children's whole systems and organs. None of the functions studied so far have been considered to be normal.<sup>18</sup> It is suggested that all processes in the body enter a phase of adaptive functional reduction as a way to ensure survival.<sup>4,5,10</sup>

Details on the physiopathology of malnutrition can be found in several publications.<sup>4,5,9-11,14,30,32,36,42-49</sup> It is important to get acquainted with details that serve as basis for the treatment of children with severe malnutrition.<sup>9</sup> See the following table.

### **Edematous malnutrition**

The cause of edematous malnutrition is still a controversial topic in protein-energy malnutrition. Classical theory<sup>55,56</sup> affirms that deficient protein supply leads to

**Table 1** - Major physiopathological aspects of child severe malnutrition

| <b>Organ/System</b>       | <b>Alteration</b>  | <b>Effect on functions</b>  |
|---------------------------|--|---|
| <b>Digestive tract</b>    | - Flattening and atrophy of intestinal villi   | - Reduction in digestive enzyme levels<br>- Maldigestion, malabsorption, and diarrhea<br>- Micronutrient deficiency |
| <b>Liver</b>              | - Steatosis<br>- Hepatocyte injury<br>- Severe alteration of all liver functions<br>- Reduction of protein synthesis and gluconeogenesis | - Hypoproteinemia<br>- Edema<br>- Hypoglycemia  |
| <b>Muscles</b>            | - Reduction/loss of skeletal and smooth muscle mass  | - Severe emaciation<br>- Weak movement of trunk and limbs<br>- Myocardial alterations                               |
| <b>Immune system</b>      | - Atrophy of the thymus, tonsils and lymph nodes<br>- Depressed immunity   | - Subclinical infections<br>- Septicemia  |
| <b>Metabolism</b>         | - Alterations in basal metabolism and sodium-potassium pump activity   | - Hypoglycemia<br>- Hypothermia/hyperthermia<br>- Electrolyte disorders   |
| <b>Circulatory system</b> | - Altered kidney function<br>- Cardiac output and reduced circulatory volume   | - Risk of death due to cardiac overload   |
| <b>Hormone system</b>     | - High levels of insulin and growth factor 1<br>- High levels of growth hormone and cortisol   | - Reduced intolerance of lactose and insulin  |
| <b>Kidney</b>             | - Reduced glomerular filtration, reduced sodium and phosphate excretion  | - Risk of death due to sodium administration<br>- Regular urinary infections  |

reduced albumin synthesis. Fat liver, with a fat content 50% higher than its structure, a ratio that is higher than that observed in animal experiments or in any other human condition, is statistically associated with edematous malnutrition.<sup>5</sup> The hypothesis that this disease results from a flaw in the transport of fat out of the liver, and that this is caused, in its turn, by the reduced apolipoprotein synthesis in parallel with the reduced albumin synthesis, has not been proved yet. There is also epidemiological evidence in support to this classical theory: edematous malnutrition usually affects populations where the staple diet is poor in protein, including foods such as cassava, or containing poor quality protein as corn-based diets.

An antagonistic theory denies the relationship between edema and hypoalbuminemia and suggests that the edema originates from the lesion caused by free radicals on the walls of capillary vessels and cell membranes.<sup>32</sup>

### **Malnutrition clinical status**

The term protein-energy malnutrition comprises a wide variety of clinical situations whose severity ranges from extremely severe to mild cases. At one end of this spectrum, we find kwashiorkor and marasmus, with high mortality rates, and at the other end, mild PED (Protein-energy deficiency), whose main manifestation in children is growth retardation. The clinical status of protein-energy malnutrition has been described in detail in classic literature.<sup>4,5,10,17,35,39,44</sup>

Kwashiorkor and marasmus have distinct clinical manifestations. The major characteristics of Kwashiorkor are growth retardation; muscle loss and subcutaneous fat loss with less intensity than in marasmus; pitting edema, located especially on the legs of toddlers but which could spread all over the body; accentuated hepatomegaly due to hepatic steatosis; and mental and humor behavioral changes.

Generalized or localized (flag-shaped) hair lesions (texture, color, brightness, hairfall), and also skin lesions (depigmentation, frictional dermatosis, desquamation) may also occur. Anorexia, diarrhea, infections and micronutrient deficiency (vitamin A, zinc, iron) are frequent. The presence of a significantly high weight loss rate and the presence of edema are the essential aspects that help diagnose kwashiorkor.<sup>9</sup>

A child with advanced marasmus presents an unmistakable appearance: too thin, with observable loss of muscle mass; extremely thin sides and, sometimes, protuberant abdomen; face with an old or Siamese cat-like aspect; and loose skinfolds on the buttocks (mainly). The major clinical signs are low weight (weight/age ratio less than 60% of the weight expected for the age); growth retardation (low height for the age); and scarce or inexistent skin fat. Normally, diarrhea, respiratory infection, parasite infections, and tuberculosis are present as well as signs of micronutrient deficiency such as xerophthalmia, vitamin B deficiency, iron deficiency anemia and others. The state of mind may be characterized by anxiety rather than apathy. Body temperature tends towards hypothermia.

A percentage of undernourished children may present a mixed form of malnutrition, marasmatic kwashiorkor, with mingled characteristics in relation to other clinical forms. Usually, when the edema disappears with treatment, it is possible to observe that these children are affected by marasmus.

In children with malnutrition, the signs of dehydration are not reliable. The children may present sunken eyes due to subcutaneous fat loss by the eye orbit. Several glands such as sweat, lacrimal and salivary glands present atrophy. The children have dry mouth and dry eyes, and reduced sweating. Respiratory muscles are easily fatigable. The children lack energy.

Children with severe malnutrition are usually apathetic when admitted to hospital, do not respond well to social stimuli, cry very often, are extremely thin, unproportionate, and/or present edema restricted to the dorsum of the foot or hand or generalized edema. These children also have overall retarded development, being unable to captivate people, as usually occurs with healthy children. When on their mothers' lap, they resemble a parcel that is being carried, and not human beings.

Children who do not present the clinical features of marasmus, but who present growth deficiency, are considered to have moderate or mild malnutrition.

At present, the height/age, weight/height and weight/age indicators are used to determine nutritional status, according to the World Health Organization recommendation.<sup>51,52</sup> The cutoff points for determining the nutritional status (severe if less than -3 DP, moderate between -2 and -3 DP, and mild between -1 and -2DP) are

based on statistical relations between anthropometric indicators on the one side, and functional restrictions, increased risks of morbidity and mortality and other evidences of risk factor consequences related to food and non-food risk factors, on the other side. Other publications<sup>51-54</sup> contain a detailed description on anthropometric indicators, including their construction, application and interpretation.

The results of nutritional, physiological processes for children are physical growth, activity, morbidity and mortality (related to immunocompetence and tissue integrity and psychological development.<sup>55</sup> Among these, growth is the one that can be measured more easily: malnutrition leads to early growth curve horizontalization and/or growth curve reduction<sup>48</sup> and is the cause of most anthropometric deficiencies observed in children from developing countries.<sup>1</sup>

Currently, there are three forms of malnutrition according to anthropometric classification: *low height*, *nutritional stunting* and *weight loss* (emaciation), which were already described in the natural history of the disease, and low weight. Low weight is detected by the weight/age indicator, which represents the body mass in relation to age; reflects linear growth and accumulated weight in the prenatal and postnatal period (long term) as well as the accumulation of weight on the short term. Low weight/age may thus reflect a normal growth variation or growth deficiency. A child is considered to have low weight/age when his/her weight is below -2DP if compared to the international growth reference standard.<sup>16</sup> This indicator may be influenced by the addition of child's rate of stunting and thinness, and is then difficult to be interpreted. Therefore, it is not the ideal indicator for either defining the kind of intervention that should be used or identifying the target group.<sup>54</sup>

By definition, the average Z-score of the reference population is zero for any of the indicators. The negative Z-score indicates that the studied child and/or population is below the nutritional status standard desired. If the Z-score equals or is less than -3, malnutrition is severe; between 2 and -2.9, malnutrition is moderate. Conventionally, a typical healthy population presents less than 1% in severe deficiencies and around 2.3% in moderate deficiencies.<sup>16,54,51</sup>

Another way to classify nutritional status is a percentile-based method of comparison. If the anthropometric indicator value is below the 3rd percentile, malnutrition is considered moderate or severe, and if it is between the 3rd and 10th percentiles, malnutrition is mild.<sup>16</sup> This method is commonly used in Brazil by health programs and the System for Food and Nutrition Surveillance.

For a more detailed description of anthropometric indicators, including their construction, use and interpretation, see the references at the end of this article.<sup>51-54</sup>

## Diagnosis

The diagnosis of malnutrition is made through the child's clinical history, clinical examination and determination of his/her nutritional status. To diagnose the disease, define and control its treatment, it is extremely important that the child be carefully assessed and followed up. The child's clinical history is efficient throughout the treatment, even in situations in which lab exams cannot be carried out or easily interpreted. When there are available resources, lab exams may be used to help with the treatment. However, it is worth remembering that, in the case of children with severe malnutrition, exam results must be carefully interpreted, as they may be altered by malnutrition itself, establishing confusion among less experienced health professionals.

The biochemical and metabolic modifications are similar in children with marasmus, kwashiorkor, and marasmatic kwashiorkor.<sup>9,45</sup> Lab exams may be confusing or difficult to interpret, due to the fact that modifications are severe and complex. The total serum protein in kwashiorkor is low due to albumin reduction, as a result of the hepatic synthesis alterations. In marasmus, this concentration is normal. The content of essential aminoacids may be low while, especially in kwashiorkor, the content of nonessential aminoacids is normal or high. The concentration of serum immunoglobulin G may be high because of infections. The concentration of retinol-binding protein may be low. The concentrations of hemoglobin and hematocrit are usually low. The concentrations of creatinine and urinary hydroxyproline are low, especially in patients who are too emaciated.

Biochemical signs of vitamin A, riboflavin, thiamin, niacin and ascorbic acid may be found in addition to deficiency of minerals such as iron, zinc and magnesium. There might also be biochemical signs of hydroelectrolytic imbalance secondary to dehydration caused by diarrhea.

## Treatment

Several aspects in the treatment of children with malnutrition have been reviewed by several authors.<sup>4,8-12,9-11,14,48</sup> In Brazil, the necessity to improve the assistance to these children has given rise to debates on ideas and proposals for improved and systematized actions,<sup>57-59</sup> including a possible adaptation of the new World Health Organization<sup>9</sup> guidelines for the treatment of children with severe malnutrition to Brazilian reality.

Depending on how severe the case is, children who suffer from malnutrition may be treated at a hospital, nutrition centers, clinics and in his/her community/home.

*Hospitalization is mandatory for the treatment of children with clinical manifestations of kwashiorkor, marasmus or marasmatic kwashiorkor, whose weight/age ratio is less than - 3DP or less than 70% of the NCHS average reference values, associated with increased inappetence; and/or diarrhea and/or vomiting; and/or any associated infection. Children with severe protein-energy*

*malnutrition (W/A < -3DP) must also be admitted to hospital (they should not be referred to treatment in clinics, nutrition rehabilitation centers and others.<sup>60</sup> When it is possible to measure height, the weight/age indicator should be used; severe malnutrition is characterized by the presence of edema, severe emaciation (less than 70% of weight/age ratio or - 3 DP) or clinical signs of severe malnutrition.<sup>9</sup>*

The *hospital approach* is also used for the treatment of children with kwashiorkor, marasmus and marasmatic kwashiorkor. The treatment should be initiated at the time of medical assistance; the child must be handled as little as possible. To facilitate understanding, the treatment for children with severe malnutrition can be divided into 3 stages, which are described next. The minimum total duration for the treatment is 26 weeks, so that rehabilitation is achieved and relapses can be prevented.

In the *stabilization stage*, from the 1<sup>st</sup> to the 7<sup>th</sup> day of treatment, life-threatening problems are identified and treated, and specific deficiencies and metabolic abnormalities are corrected; after that, feeding is initiated. In the *rehabilitation stage*, from the 2<sup>nd</sup> up to the 6<sup>th</sup> week, the child must be intensively fed so that he/she can recover most of the weight that was lost. Emotional and physical motivation is enhanced, the mother, or anyone who takes care of the child, must be trained in order to proceed with the treatment at home; and, finally, the child is ready to leave the hospital. If hospitalization lasts less than 6 weeks, adequate support must be provided so that the rehabilitation treatment can be concluded at a nutrition center, clinic or at home.

The *follow-up stage*, from the 7<sup>th</sup> up to the 26<sup>th</sup> week, starts right after the child leaves the hospital, especially if the child was released before the rehabilitation stage was complete. The child and his/her family are followed up to make sure there is no relapse and guarantee the child's emotional, physical and mental development be continued.

When children finish the first stage of treatment, if they do not present any complications, and are feeding normally and gaining weight satisfactorily (usually 2-3 weeks after admission), they can proceed with the treatment at a nutrition rehabilitation center, without the need for hospitalization. A *nutrition rehabilitation center*<sup>61</sup> is a day hospital, a health center or similar institution that provides day care through a team specialized in child malnutrition. Children spend the night at home, are brought in every morning, and go back home at the end of the day. There must be a close collaboration between the hospital and the health center to ensure that child continue to be cared for and to provide their immediate referral to the hospital in the event of any serious problems. In urban areas, the nutrition centers must be preferably located near hospitals. In areas where there are no specialized centers, the hospital must take care of children until they are ready to be released from the treatment.

*Children with severe malnutrition are normally seriously ill and at risk of death when brought in for treatment. They*

usually require emergency treatment. These children must be kept warm, properly dressed and covered, and away from air drafts, especially at night. The ideal room temperature should be kept at 25-30 °C because children with severe malnutrition, especially the younger ones, are prone to hypothermia. In hot regions, extra care should be taken to prevent children from being overly warm during the hottest hours of the day.

*All children with severe malnutrition are at risk for hypoglycemia* (blood glucose concentration <54mg/100ml or <3mmol/l), an important cause of death during the first two days of treatment. Hypoglycemia may result from a severe systemic infection or from the lack of feeding during the last 4-6 hours. If there is suspicion of hypoglycemia, *the treatment must be started immediately, without the need for laboratory confirmation*; there will be no harm even if the diagnosis is not correct. Usually, the only sign before hypoglycemia-associated death is drowsiness. If it is not possible to dose glycemia, it should be taken for granted that all children with severe malnutrition have hypoglycemia. If children are able to drink, they should be given 50ml glucose or saccharosis at 10%, or fed a preparation that is suitable for this stage. If the child is unconscious, administer 5ml/kg of body weight of a sterilized glucose solution at 10% intravenous (IV). If children have convulsions caused by hypoglycemia, keep intravenous infusion of glucose at a speed between 4 and 6 mg/kg/hour, until the patient's status is stabilized. When the status is stabilized, administer 50ml of glucose at 10% or saccharosis through a nasogastric tube. When children recover consciousness, start to administer the diet or solution of glucose diluted in water (60g/l). Continue oral feeding or nasogastric tube feeding every 2 hours, day and night, at least during the first day, to avoid recurrence.<sup>9</sup> *All children with severe malnutrition suspected of/ diagnosed with hypoglycemia* must be treated with broad-spectrum antibiotics for systemic infections.

*Hypothermia is associated with increased mortality.* All hypothermic children must be treated for hypoglycemia and for severe systemic infection. Hypothermia is common in children with severe malnutrition, younger than 12 months; with marasmus, with a large part of the skin presenting lesions; or severe infections. If axillary temperature is below 35 °C or cannot be read on the available thermometer, take for granted that children are hypothermic. Keep children warm, feed them, and treat existing infections.

*It is hard to make a difference between dehydration and septic shock in children with severe malnutrition.* In many septic shock cases, there is a history of diarrhea and a mild or moderate level of dehydration, producing a mixed clinical status. Many of the signs commonly used to assess dehydration are not reliable in children with severe malnutrition, which does not allow the detection of dehydration and its severity in a reliable way. In addition, many signs of dehydration are also found in septic shock, causing dehydration to be overdiagnosed and its severity to

be overestimated. It is very common to treat children for dehydration and septic shock simultaneously.

*When treating dehydration*, oral administration is preferred; IV administration is reserved for cases in which there are definite signs of shock, as it usually causes hyperhydration and cardiac insufficiency. The oral rehydration solution should have less sodium and more potassium than the standard solution recommended by the World Health Organization. Magnesium, zinc and copper should also be used to correct the insufficiency of these minerals. The oral dehydration solution suggested by the World Health Organization for children with severe malnutrition contains approximately 45mmol of sodium, 36mmol of potassium and 3mmol of magnesium per liter.<sup>9</sup>

*When treating septic shock*, use intravenous hydration with an initial volume of 15 to 20ml/kg during the first hour, with a 0.45% saline solution (half the physiological solution) with 5%<sup>2</sup> glucose (glucosate solution at 5% -1:1.) or Ringer's solution lactate with glucose at 5%. If possible, add sterilized potassium chloride (20mmol/l). Monitor children rigorously to avoid hyperhydration, adapting the subsequent steps to the response obtained. Treat the infection vigorously. Resume feeding and start oral rehydration as soon as possible. For further details on rehydration, see the references at the end of this article.<sup>9</sup>

*Congestive heart failure is a usual complication caused by hyperhydration* (especially when IV infusion is carried out or the standard rehydration solution is used), severe anemia, by blood or plasma transfusion, or by high-sodium diet. If there are signs of heart failure, all oral intake and IV fluids must be interrupted until the failure no longer exists. In this case, the use of an IV diuretic is recommended, preferably furosemide (1mg/kg). Digitalin should not be administered unless the heart failure is unequivocal and the levels of plasma potassium are normal. In this case, 5mg/kg of body weight of digoxin (single dose) should be IV administered, or orally if the preparation for IV infusion is not readily available.

All undernourished children present potassium and magnesium deficiency, which could take 2 or more weeks to be corrected. The edema is partially resultant from these deficiencies. Total body sodium is too high, although plasma sodium may be low. High-sodium administration may lead to child's death. Extra potassium (2-4mmol/kg/day) and magnesium (0.3-0.6mmol/kg/day) may be added to food during its preparation. Low-sodium fluids must be used for rehydration. Existing edematous malnutrition cannot be treated with diuretics.

*Almost all the children with severe malnutrition have bacterial infections at the time they are admitted for hospital treatment*, and it is right to assume that, usually, these infections are subclinical and have to be treated immediately. The administration of antibiotics soon after admission, until the results of lab exams are known, may save many children. The antibiotic therapy may be changed later, if necessary, according to the results of lab exams.

*All children with severe malnutrition have vitamin and mineral deficiencies.* The recommended treatment is : iron-free multivitamin supplementation for at least 2 weeks, every day; folic acid ( 5mg on the first day and after that 1mg/day), zinc (2mg/kg/day), copper (0.2mg/kg/day). *When children start to gain weight*, which usually occurs at the beginning of the second week of treatment, start using ferrous sulphate (3mg of Fe/kg/day). Children with severe malnutrition are at high risk for blindness due to vitamin A deficiency, and should be given vitamin A orally, on the first day (infants younger than 6 months: 50,000IU; 6-12 months: 100,000IU; older children: 200,000 IU). The age-specific dose should be given on the second day and should be repeated for at least 2 weeks after that.

*In case of severe anemia* (Hb < 4g/dl or Hb between 4 and 6g/dl ) and/or difficult breathing, infants should be fed a blood meal (slow administration of 10ml/kg during 3 hours and furosemide 1mg/kg IV, at the beginning of transfusion). Children need to be monitored every five minutes for checking heart failure.

*Children must be fed light meals*, with low osmolarity and low lactose, every 2, 3 or 4 hours, night and day, right after admission. Oral administration is preferred; if that is not possible, a nasogastric tube should be used. The nutritional goal in this stage is to reach the maximum intake of 100kcal/kg/day (minimum acceptable 80kcal/kg/day ) and 1-1.5g protein/kg/day. A total of 130ml/kg/d of fluid is recommended (100ml/kg/day if children present important edema) . If children are being breast-fed, breast-feeding must be discontinued, but adequate formula-feeding should be provided so that calorie requirements are met. IV administration is exceptionally used in primary malnutrition.

The amount of food ingested by children has to be rigidly measured and recorded. If the minimal goal is not achieved, children will have to be fed through a nasogastric tube, after having been orally fed. The nasogastric tube must be removed when children start to orally ingest  $\frac{3}{4}$  of the total daily diet, or the total volume in 2 consecutive meals. If intake in the next 24 hours does not reach the minimum of 80kcal/kg/day, the nasogastric tube should be reintroduced.

The stabilization stage, for children who have good appetite and who do not have edema, may be completed within 2 to 3 days.

*Appetite response indicates that children have entered the rehabilitation stage*, usually one week after admission. In this stage, it is necessary to have a high rate of intake to allow fast growth to take place, >10g/kg/day. A gradual transition between the formula used in the initial stage and the formula used for growth enhancement is recommended in order to avoid the risk for heart failure, which could occur when children ingest a large amount of food in the initial stage. The initial formula (75 kcal/100ml and 0.9g protein/100ml) must be replaced *during 48 hours*, with the same volume of milk-based formula for growth enhancement (containing 100kcal and 2.9g of protein/100ml). Modified

milk porridge or complementary foods may be used provided that they supply comparable amounts of energy and protein content. After 48 hours, if children adapt well to the formula, an increase of 10ml at each successive feeding should be applied until food is left over. This usually happens when intake reaches 200ml/kg/day.

*After gradual transition*, provide frequent feedings, according to acceptance (unlimited amount), in order to achieve the nutritional goal of 150-220kcal/kg/day and 4-6g of protein/kg/day. Family food may be fed to infants older than 24 months, to guarantee nutrition requirements. The progress of rehabilitation should be assessed through weight gain. Heart failure is unlikely to occur if gradual transition is followed. However, to be on the safe side, the presence of early signs of heart failure should be monitored. If there are signs suggesting heart failure, the administered volume should be reduced to 100ml/kg/24 hours and be gradually increased to 115ml/kg/day during the next 24 hours, 130ml/kg/day for the next 48 hours, and finally increased to 10 ml at each feeding, as previously described.

If intolerance to lactose with effects on infant growth is diagnosed, the treatment must be carried out with lactose-free formulas, which should be modified in order to meet the adequate goals required for rehabilitation. Whole milk meals should be reintroduced before children leave the hospital to determine whether intolerance was eliminated.

*As there is behavioral and mental development retardation in children with severe malnutrition*, it is important that basic stimuli, recreation, affection, and mother-child relationship be enhanced. Mothers should participate in child care tasks and should be taught to provide basic stimuli.

During hospitalization, *children and mothers should be prepared to leave the hospital*. Children who present 85% -90% of the weight required for their age or height (equivalent to - 1DP) may be regarded as rehabilitated. These children may still have low weight for their age due to stunting. Good feeding practices and psychological stimuli should be continued at home. Parents, or whoever is taking care of these children, should be taught how to feed them foods that are rich in energy and nutrients and how to provide them with structured recreational therapy/basic stimuli.

During follow-up, children should be weighed every week. If these children do not lose or gain weight within 2 weeks, they should be referred to hospital for re-evaluation. If these children respond well, the frequency of treatment may be progressively changed to 1 time every 15 days during 3 months, every 30 days during 3 months, every 60 days during 6 months and every 6 months after that, until these children turn 3 years.

*Nutritional rehabilitation in clinics and community* is possible, although it takes longer. In this case, parents have heavier responsibility for the successful treatment of their children. Therefore, it is crucial that parents get the necessary guidance and practical support from health agents and

professionals.<sup>58</sup> In general, orientation is similar to that given before the children leave the hospital and throughout the follow-up of those children who were hospitalized.

Mothers should be instructed on how to prepare meals with adequate nutritional value and be informed that their children need to be fed at least 5 times a day. Usual family food should be prepared so as to contain approximately 100kcal and 2-3 g of protein per 100g of food. Vitamin, iron and electrolyte/mineral supplements should be provided.

A follow-up plan has to be followed and an effective treatment routine<sup>58</sup> should be adopted, at least until children are totally rehabilitated. It is essential that community health workers give their support. If children were discharged from hospital early, supervision must be reinforced in the clinic or at home, as they have a very high risk for relapse and death. Children should be carefully assessed before leaving the hospital and some kind of community support must be available in order to prevent a relapse. Home treatment, in addition to having a reduced cost for the health sector, is mothers' favorite.<sup>62</sup>

#### **Possible alternatives for child malnutrition control**

The attempts to control malnutrition as a public health problem in developing countries began in the postwar period and used to include increased production of protein-rich foods and the promotion of nutrition programs and education.<sup>63</sup> The results were disappointing. Nutritional assistance, which was provided on a large scale but independently from other social and developmental efforts, consisted of a symptomatic instead of causal treatment.

The effective prevention of protein-energy malnutrition cannot have a distant objective from that of general measures that aim at meeting poor people's basic needs.<sup>23</sup> This fact has triggered international decisions on population's economic improvement through strategies and programs especially designed for groups at higher risk for malnutrition.<sup>23</sup> Even though history shows that the solution to poverty is an ambition that is too difficult to be fulfilled on the short-term, there have been well-planned interventions by the health sector aimed at preventing child malnutrition.<sup>64</sup>

Unfortunately, as malnutrition is considered to have multiple causes, it has often been a preoccupation to many people, but no one has taken the onus on themselves to combat it.<sup>10</sup> Several health professionals label child malnutrition as a "social problem" and approach it with listlessness, unimportance or defeatism regarding children with or at risk for malnutrition; and contempt for any child nutrition campaigns. These workers act fragmentally and often keep a distance from updated information that could be helpful in controlling malnutrition.<sup>51,52,65</sup> Others, taking for granted that there are fewer children with severe malnutrition today, underestimate the importance of such problem and even ignore the existence of a silent hidden starvation and malnutrition epidemics which is expressed

through the number of children with moderate and mild malnutrition, although less evident.<sup>66</sup>

The strategies adopted by the health sector as to the prevention of malnutrition were clearly defined in 1978, when, in Alma-Ata, the focus on primary attention to health for all in the year 2000,<sup>26</sup> and the promotion for adequate nutrition were designed. According to the World Health Organization, the health sector holds the following responsibilities as far as nationwide nutrition and feeding are concerned: a) definition and analysis of nutrition problems; b) promotion and participation in multisectorial feeding/nutrition strategies and programs; and c) implementation of a feeding and nutrition surveillance system.<sup>67</sup>

In several countries,<sup>64</sup> and also in Brazil, well-planned programs for primary attention to health have made a difference, especially if the instructive information is not vertically defined and is culturally appropriate and viable for mothers.<sup>41,61,68</sup>

The introduction of the nutritional aspect in children's routine treatment plays a vital role in the prevention of malnutrition. Recent studies show that, as far as infants younger than 3 years are concerned, stunting begins a few months after birth and goes up to the 2nd year of life, coinciding with the age at which foods complementary to breast-feeding are introduced. Thus, it is possible that innovative program approaches aimed at promoting complementary feeding for infants aged under 2 years have better cost-effectiveness than those approaches targeted on preschool children and may have historically unprecedented success in reducing child malnutrition.<sup>40</sup> Nutrition guides for the promotion of complementary feeding for Brazilian infants under 2 years, as an initiative by Pan-American Health Organization (PAHO/WHO) and Ministry of Health, have been recently elaborated with the solid participation of health professionals from all over the country and are just about to be printed. After that, these guides will be ready for distribution.<sup>69</sup>

In Brazil, in agreement with the governmental objective of reducing the prevalence of moderate and severe malnutrition to 50% until the year 2000,<sup>16</sup> which was endorsed at the World Summit on Child Care, the Ministry of Health has earnestly endeavored to promote nutrition and reduce infant mortality. In an attempt to allow population's access to these actions, some programs were successively implemented in the 1980s. Namely, these programs are: Community Health Agents Program, Family Health Program<sup>70</sup> and, in partnership with WHO/PHO, the initiative called Full Attention to Prevalent Childhood Diseases.<sup>60</sup> It is believed that Community Health Agents may be effective if they are well-trained and supervised, and if they have enough level of judgment and accuracy for the reference of cases that cannot be treated at home.<sup>71</sup> The Full Attention to Prevalent Childhood Diseases<sup>60</sup> currently includes exclusive actions for the promotion of clinic-based child nutrition treatment.

At clinics or at home, the Family Health Program teams, with health agents and professionals, may act on critical aspects,<sup>38</sup> providing nutritional counseling to pregnant women, information on infections, advice on programs that handle shortage of food, and warning against the apparent negligence towards child care caused by mothers' misinformation.<sup>57,66</sup>

Although the shortage of food at home is an important factor in malnutrition, a possibility for effective intervention by the health sector, working together with mothers in order to reduce malnutrition, was found by health professionals as Nobrega and Campos,<sup>44</sup> throughout 20 years of work at a nutrition rehabilitation center, and was recently confirmed by Muniz.<sup>67</sup> These authors found that around 30% of mothers whose children suffered from malnutrition were paradoxically eutrophic, were overweight or even obese. An in-depth investigation showed that child care practices were inadequate. In children assisted by Nobrega and Campos,<sup>72</sup> this was due to a withered mother-child relationship. Their multiprofessional team has enabled mothers to rehabilitate their children and avoid the recurrence of malnutrition through the support and enhancement of a positive relationship with mothers.

Muniz,<sup>66</sup> found an unfavorable pattern of child care practices. She used an approach chosen together with mothers with the aim of restoring their self-esteem, in addition to providing them with practical knowledge and instructions on how to take care of children with malnutrition. This education-based intervention carried out by the Family Health Program in Vitória, state of Espírito Santo, managed to change mothers' practice to a more favorable pattern, and rehabilitated 70% of the children with malnutrition through home treatment and without any additional food supplements.

One of the main characteristics of nutrition enhancement actions is the potential for prevention and management of infectious diseases.<sup>4,7,8</sup> The appropriate management of children's diet favors the reduction in the frequency and severity of infections. During an infection, dietary management is aimed at changing the course and outcomes of the disease, through adequate food intake during the infection and rehabilitation period, especially in younger children.<sup>38</sup> Dietary management actions may be promoted by health professionals by directly counseling mothers on breast-feeding; complementary feeding for infants aged 6 months or older, including foods that are rich in vitamin A, iron, zinc, vitamin B6; feeding of diseased children; and oral rehydration in case of diarrhea. For further practical information on these procedures, see the references at the end of this article.<sup>39,40,68,73</sup>

Reducing the prevalence of child malnutrition requires focused and systematized health sector and nutrition safety actions, in special regard to mothers, so that they can take proper care of their children. The key elements of such actions include access to education, health care, good quality water, protection against diseases, and adequate

intake of micronutrients. In addition, an appropriate community system for following up and supporting children with mild, moderate and severe malnutrition should be implemented. If this is done in an effective way, it is possible to quickly reduce malnutrition rates as in Oman, Thailand, Uruguay, Vietnam, and Zimbabwe.<sup>5</sup> This quick reduction in malnutrition rates is an urgent need since it is part of each and every prospective effort into reducing poverty.<sup>28</sup>

## Conclusion

Child malnutrition is still the most important public health problem in developing countries. Its effective reduction depends on integrated interventions that can reduce poverty and improve the quality of life of unprivileged families. This implies wide-scope strategies developed by the government and involves intense community participation. Health professionals are not in charge of changing society's political and economic structure, but they have to understand the inequalities and limitations experienced by the population they assist and be able to apply currently available scientific knowledge. The challenge is to gradually reduce the number of children affected by malnutrition no matter how severe the disease is. There is much to be done, and there are several opportunities and ways by which health professionals who are updated and work in an efficient and correct way may contribute to the nutrition and health of Brazilian children. Some possibilities were presented in this article.

Thanks to Helenice Muniz, Roseli Sarni, Ana Augusta Cavalcante and Virginia Costa for their support and productive exchange of ideas during this review.

## References

1. ACC/SCN - First Report on the World Nutrition Situation. Geneva: ACC/SCN; 1988.
2. ACC/SCN. Nutrition throughout life. 4<sup>th</sup> Report on the world nutrition situation. Geneva; ACC/SCN./World Health Organization: 2000.
3. WHO. Global database on child growth and malnutrition. <http://www.who.int/nutgrowthdh>. Geneva: World Health Organization; 1997.
4. Latham MC. Malnutrición proteico-energética In: OPS/ILSI- Conocimientos actuales sobre nutrición. Sexta edición. Publicación Científica n° 532. Washington, DC: Organización Panamericana de La Salud; 1991. p. 47-55.
5. Waterlow JC. Protein-energy malnutrition: the nature and extent of the problem. *Clin Nut* 1997; 16: 3-9.
6. WHO. The world health report 1995: Bridging the Gaps. Geneva: World Health Organization; 1995.
7. Pelletier DL. Potentiating effects of malnutrition on child mortality: epidemiologic evidence and policy implications. *Food Nutr Bull* 1995; 16: 206-13.

8. Schofield C, Asworth A. Why have mortality rates for severe malnutrition remained so high? *Bull World Health Org* 1996; 74:223-9.
9. OMS. Manejo da desnutrição grave: um manual para profissionais de saúde de nível superior (médicos, enfermeiros, nutricionistas e outros) e seus auxiliares. Genebra/Brasília: Organização Mundial da Saúde/Organização Panamericana da Saúde; 2000.
10. Waterlow JC. Protein and energy malnutrition. London: Edward Arnold; 1992.
11. Brewster D, Manary M. Treatment of severe malnutrition. *Lancet* 1995; 345:1046-47.
12. Chevalier P. Zinc and duration of treatment of severe malnutrition. *Lancet* 1995; 345:1046-7.
13. Cavalcante AAM, Pinheiro LMP, Monte CMG, Guimarães ARP, Ashworth A. Treatment of malnutrition in Brazil: simple solutions to common problems. *Trop Doctor* 1998; 28: 95-7.
14. Chopra M, Wilkinson D. Treatment of severe malnutrition. *Lancet* 1995; 345: 788-9.
15. Monteiro CA. Velhos e novos males da saúde pública no Brasil. São Paulo: Hucitec; 1995.
16. Ministério da Saúde. Metas governamentais para o ano 2000. Brasília: Ministério da Saúde; 1996.
17. Williams CD. A nutritional disease of childhood associated with a maize diet. *Arch Dis Child* 1933;1: 8:423-33.
18. Trowell HC, Davies JNP, Dean RFA. Kwashiorkor. London: Edward Arnold; 1954.
19. WHO. Energy and protein requirements. World Health Organization Technical Report Series Nº 522. Geneva: WHO; 1973.
20. Mc Laren DS. The protein fiasco. *Lancet* 1975; ii: 93-6.
21. Dewey KG, Beaton G, Fjeld C, Lonnerdal B, Reeds P. Protein requirements of infants and children. *Eur J Clin Nutr* 1996; 50: Suppl 1: 5139-49.
22. FAO. Fourth World Food Survey. Rome: Food and Agricultural Organization; 1977.
23. World Bank. Poverty and hunger: issues and options for food security in developing countries. Washington: World Bank; 1986.
24. Scrimshaw NS, Taylor CE, Gordon JE. Interaction of nutrition and infection. WHO Monograph Series nº 57. Geneva: World Health Organization; 1957. p.1-52.
25. Monte CMG. Monitoring/evaluating health and nutrition programmes in developing countries: Information to whom and for what purpose? [thesis] Londres: London School of Hygiene and Tropical Medicine; 1988.
26. WHO. Health for all in the 21<sup>st</sup> Century. EB101/8. Geneva: World Health Organization, 1998.
27. Ashworth A, Bell R, James WPT, Waterlow JC. Calorie requirements of children recovering from protein calorie malnutrition. *Lancet* 1968;ii: 2600-3.
28. ACC/SCN - Third World Report on the World Nutrition Situation. Geneva: ACC/SCN; 1997.
29. Martorell R, Kettel K, Schroeder DG. Reversibility of stunting: epidemiological findings in children from developing countries. In: Waterlow JC & Schurch B, eds. Causes and mechanisms of linear growth. I/D/E/C/G/ workshop held in London, January 15-18, 1993. *Eur Jour Clin Nutr* 1994; 48: 45-57.
30. Bengoa JM. The problem of malnutrition. WHO chrono 1974; 28:3-7.
31. Bruiya A, Zimicki SD, Souza S. Socioeconomic differentials in child nutrition and morbidity in rural area of Bangladesh. *J Trop Ped* 1986, 32:17-23.
32. Golden MHN, Ramdath DD. Free radicals in the pathogenesis of kwashiorkor. *Proc Nut Soc* 1987; 46:53-68.
33. Gabr M. Undernutrition and quality of life. *Wld Ver Nutr Diet*. 1987; 49: 1-21.
34. Waterston T, Nhembe M. Causes of malnutrition in Harare. A hospital case control. *Centr Afr J Med* 1984, 30: 97-102.
35. Golden MHN. Severe malnutrition. In: Weatherall DJ, Ledingham JGG, Warrell DA. *Oxford textbook of Medicine*. Vol 1. 3rd edition. Oxford: Oxford University Press; 1996. p.1278-96.
36. Jelliffe D B. Protein-calorie malnutrition: a review of the recent literature. *J Pediatr* 1959; 54:227-56.
37. Schrimpton R. Ecologia da desnutrição na infância: análise das evidências das relações entre variáveis socio-econômicas e estado nutricional. Brasília: CNRH/IPEA/UNICEF; 1986. p.104.
38. UNICEF - The State of the World's children 1998. Oxford: United Nations Children Fund/Oxford University Press; 1997.
39. Nobrega FJ, Campos ARL. Fraco vínculo mãe-filho como fator de risco. In: Nobrega FJ, org. *Distúrbios da Nutrição*. Rio de Janeiro: Revinter; 1998. p.88-93.
40. WHO - Complementary feeding of young children in developing countries. Geneva: World Health Organization; 1998.
41. Monte CMG, Sá MLB, eds. Guias alimentares para as crianças de 6-23 meses do Nordeste do Brasil: da teoria à prática. 1<sup>a</sup> ed. Fortaleza: The British Council Northeast Brazil/OPS/MS; 1998.
42. Golden MH, Briend A. Treatment of severe child malnutrition in refugee camps. *Eur J Clin Nutr* 1992; 46: 607-706.
43. Jackson AA. Nutritional adaptation in disease and recovery. In: Blaxter K, Waterlow JC, eds. *Nutritional adaptation in man*. London: John Libbey; 1984. p.11-126.
44. Sawaya AL. Transição: Desnutrição Energético Protéica e Obesidade. In: *Desnutrição urbana no Brasil: em um período de transição*. São Paulo: Revinter; 1997.
45. Ashworth A, Schofield C. Latest developments in the treatment of severe malnutrition in children. *Nutr* 1998; 14:244-5.
46. Sherry TM. The effect of the inflammatory response on bone growth. *Eur Jour Clin Nut* 1989;49:646-53.
47. Lesoud B M, Mazari. Immune response during recovery from protein-energy malnutrition. *Clin Nutr* 1997; 16: 37-46.
48. Scherbaum V, Furst P. New concepts on nutritional management of severe malnutrition: the role of protein. *Curr Opin Clin Nut Metab Care* 2000; 3:31-8.
49. Golden MHN. Protein energy interactions in the management of severe malnutrition. *Clin Nut* 1997; 16:19-23.
50. Jelliffe DB. The assessment of the nutritional status of the community with special reference to field surveys in developing regions of the world. Geneva: World Health Organization; 1966.
51. WHO. Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. WHO Technical Report Series 854. Geneva: World Health Organization; 1995.
52. WHO. Working group on infant growth: an evaluation of infant growth: the use and interpretation of anthropometry in infants. *Bull World Health Org* 1995; 73:165-74.
53. WHO. Working group on the growth reference protocol and WHO task force on methods for the natural regulation of fertility growth patterns of breastfed infants in seven groups. *Acta Paediatrica* 2000; 89: 215-22.
54. Beaton GH, Kelly A, Kevany J, Martorell R, Mason J. Appropriate uses of anthropometric indices in children. ACC/SCN State-of-Art series. Nutrition Policy Discussion Paper no 7. Geneva: ACC/SCN; 1990.
55. ACC/SCN. Second world report on the world nutrition situation. Geneva: ACC/SCN; 1992.
56. SESA-Ce/SeSA-Pi/UNICEF. Alimentação da criança nos 3 primeiros anos de vida. Manual para auxiliares de enfermagem, agentes de saúde e supervisores. Fortaleza: UNICEF; 1994.

57. Monte CMG, Sarni RS. Tratamento da criança gravemente desnutrida a nível hospitalar. São Paulo: Departamento de Nutrição da Sociedade Brasileira de Pediatria. Mimeo 38p. Fevereiro 2000. (Documento preliminar, atualmente em fase final de revisão pelo Departamento).
58. Monte CMG. Atendimento à criança desnutrida em ambulatório e comunidade. São Paulo: Departamento de Nutrição da Sociedade Brasileira de Pediatria.; 1999. Mimeo 15p.
59. Monte CMG. Normas de atenção à criança desnutrida nos diversos níveis dos serviços de saúde. Subsídio para discussão do grupo consultor de desnutrição infantil para o Ministério da Saúde. Fortaleza: UFC; 2000. Mimeo, 53 pg.
60. MS-OPAS/OMS-UNICEF/Tacro. Aconselhar a mãe ou ao acompanhante- Curso de capacitação- Atenção integrada às doenças prevalentes da infância. OPS/ HCT/ ARI/ CDD/ 96.4.(original inglês) versão preliminar Brasília: OPS/MS; 1997, 79p.
61. Monte CMG, Ashworth A, Sá MLB, Diniz RLP. Effectiveness of nutrition centres in Ceará state, northeast Brazil. *Pan Am J Public Health* 1998; 4: 375-82.
62. Khanun S, Ashworth A, Huttly SRA. Controlled trial of three approaches to the treatment of severe malnutrition. *Lancet* 1994;344:1728-32.
63. FAO. Basic texts of the Food and Agriculture Organization of the United Nations. Rome: FAO;1980.
64. Gwatkin DR, Wilcox JR, Wray JD. Overseas Development and Council. Monograph n° 13. Washington: ODC; 1980.
65. Golden MHN. Is complete catch-up possible for stunted malnourished children? In: Waterlow JC. Causes and mechanisms of linear growth retardation. I/D/E/C/G/ Workshop held in London January 15-18, 1993. *Eur Jour Clin Nutr* 1994;48:58-71.
66. Muniz HF. Práticas sociais de cuidados infantis: uma proposta de intervenção em domicílio de crianças desnutridas [thesis]. Vitória: Universidade Federal do Espírito Santo; 2000.
67. Monte CMG, Ashworth A, Nations MK, Lima AAAM, Barreto A, Huttly SR. Designing educational messages to improve weaning food hygiene practices of families living in poverty. *Soc Sci Med* 1997; 44:1453-64.
68. WHO. The role of the health sector in food and nutrition. Report of a WHO Expert Committee. Technical Report Series n° 667. Geneva: World Health Organization; 1981.
69. OPS/OMS/MS. Guias alimentares para a criança brasileira menor de 2 anos. Brasília:Ministério da Saúde/OPS; 2000. (in press).
70. MS. Saúde da família: uma estratégia para a reorientação do modelo assistencial. Brasília: Ministério da Saúde; 1997.
71. WHO. Strengthening the performance of community health workers in primary health care. Report of a WHO Expert Committee. Technical Report Series n° 780. Geneva: World Health Organization;1989.
72. Nobrega FJ, Campos ARL. Distúrbios nutricionais e fraco vínculo mãe-filho. Rio de Janeiro: Revinter; 1996.

**Correspondence:**

Dra. Cristina Monte

Rua Frei Mansueto, 150 – apto 1201

CEP 60175-070 – Fortaleza, Ceará, Brazil

Phone +55 8526.33526 – Fax +55 85 2815212

E-mail: cristina@ivia.com.br