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Marasmus–1985

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Introduction

Dr Wilfred J. Pearson writing for the first volume of this *Journal* in 1925 defined marasmus (Greek: *marasmos*, wasting) as 'a chronic state of malnutrition of a severe grade' but pointed out that 'we must retain the conception of cases varying in severity from those who simply show an insufficient gain or stationary weight with few systemic changes, to the most extreme form which is merely the end result of repeated nutritional or constitutional disturbances.' He described oedema in some cases as a complication of marasmus. The clinical picture of the syndrome, later described as kwashiorkor, was not generally recognized at this time, although a number of authors had described it (Czerney & Keller, 1928).

In 1933 Cicely D. Williams, a paediatrician working in the Gold Coast of Africa described a 'nutritional disease of childhood associated with a maize diet' and attributable to protein deficiency. She named it kwashiorkor (taken from the Ga language of Ghana, the disease of the deposed baby when the next one is born). Her classical description included oedema, chiefly of hands and feet, wasting, diarrhoea, irritability and dermatosis. This paper attracted much attention in the medical literature and clinical descriptions of similar disease subsequently appeared from many other countries. Paradoxically the more commonly encountered type of malnutrition, marasmus, was neglected and until recently did not attract detailed scientific investigation.

The original view that kwashiorkor resulted from protein lack in the presence of adequate, even excess carbohydrate, in contrast to marasmus which followed deficiency of energy alone, is not now accepted. Thus it has been shown that the protein: energy ratio of diets eaten by children developing washiorkor is adequate but total energy is deficient (Rutishauser & Frood, 1973; Goplan, 1968). However, it has become apparent that malnutrition produces a spectrum of syndromes ranging from simple growth failure alone to both pure and mixed syndromes of marasmus, marasmic kwashiorkor and kwashiorkor.

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In 1959 Jelliffe coined the term protein-calorie malnutrition (PCM) of early childhood to include mild, moderate and various types of severe degrees of malnutrition. The advent of the International System of Units (SI) resulted in the introduction of the term protein energy malnutrition (PEM). The World Health Organisation (WHO, 1973) defined PEM as follows: 'A range of pathological conditions arising from coincidental lack, in varying proportions, of protein and calories, occurring most frequently in infants and young children and commonly associated with infection'. This is not dissimilar from Wilfred Pearson's view of marasmus.

In order to treat and, more importantly, to prevent a condition effectively, evolution and causes must be understood. It is therefore necessary to consider marasmus within the context of PEM. The aim of this paper is to review the current state of knowledge concerning the pathophysiology, aetiology, clinical presentation and treatment of marasmus in the context of it being considered as one of a spectrum of syndromes produced by severe protein energy malnutrition (PEM).

Classification

Although a methodology for the assessment of PEM is needed, it is still not clear exactly what should be assessed, and there is a lack of agreement about classification among experts in this field. Until recently the method of classification used was that introduced in 1942 by Gomez and his colleagues (1956) based upon weight for age, in which the weight of the child was expressed as a percentage of the expected weight of a healthy child of the same age using a local or other appropriate standard (Table I).

A Wellcome working party (1969) considered the classification of PEM and agreed that the term marasmus should be applied to children who have less than 60% of the expected weight for age and no oedema i.e. Gomez's severe or 3rd degree malnutrition. However Gomez's method assumed that children of any given age should have the same weight and this assumption is incorrect. In 1972, Waterlow published

Table I Classification of PEM (Gomez, 1956)

Severity	*% of standard (Harvard)*
normal	>90%
mild (1st degree)	89–75%
moderate (2nd degree)	74–60%
severe (3rd degree)	<60%

Table II Classification of PEM according to wasting and stunting (after Waterlow *et al.*, 1972)

Stunting (height for age)	*Wasting (weight for height)*
0 = >95%	0 = >90%
1 = 95–90%	1 = 90–80%
2 = <90–85%	2 = 80–70%
3 = <85%	3 = <70%

0 denotes normal, and 1, 2, and 3, mild, moderate and severe PEM.

a classification based on both weight and height (Table II). He labelled present malnutrition 'wasting' measured by loss of weight related to height; and past malnutrition 'stunting' seen in retarded height for age. This system is now generally accepted and in 1977, WHO published recommendations primarily based upon it (Waterlow *et al.*, 1977). However, it needs to be translated into simpler terms for use in the busy routine clinic.

Other anthropometric measurements such as mid-arm circumference, skinfold thickness, mid-arm and head circumference ratio have been used but reproducibility is poor for the first two and there is an age limitation for the third.

Prevalence

Despite considerable research, UN resolutions and development programmes, there is more malnutrition in the world now than existed 30 years ago (Latham, 1982). There is an increasing gap between the people of the rich and poor nations and there has been a failure to reduce malnutrition and hunger due to poverty. The reasons for the failure of development and for increased inequality are beyond the scope of this paper.

Point prevalence figures for PEM collected by WHO (Bengoa, 1974) based on 77 surveys suggested that about 100 million children throughout the world are suffering from moderate or severe PEM at any one time. The relative prevalence of marasmus and kwashiorkor worldwide is not known but marasmus ranged up to 95% or more of cases of severe malnutrition in Beirut (McLaren, 1966) and Santiago (Monckeberg, 1966).

In the UK 3rd degree PEM is rare and is usually secondary to some underlying organic disease but may occasionally be due to child abuse.

Clinical presentation

This will vary with the degree and duration of PEM, the age of the child and the presence of associated vitamin, mineral and trace element deficiencies. In order to prevent marasmus it is important that these early changes are understood and recognized. PEM of all degrees is most common in the post-weaning phase (9 to 24 months of age) but can occur at any age. Failure of breast feeding and inadequate artificial feeding can lead to marasmus in the first 9 months of life.

Mild PEM

The main clinical presentation of mild PEM is that of failure to thrive with retarded physical growth and development revealed by anthropometric abnormalities. There is also an increased risk of infection and retardation of mental development. Anaemia may be present; activity is diminished and the hair around the temples may be sparse.

Anthropometric abnormalities include: (1) cessation or slowing of weight gain or weight loss; (2) slowing or cessation of height gain; (3) normal or diminished weight/height ratio; (4) decreased mid-arm circumference; (5) delayed bone maturation and (6) normal or diminished skin fold thickness.

Weight and height are the most useful indices providing that the age of the child is known. Patterns of growth failure, however, vary considerably and chronic disease, apart from nutritional deficiency, may also contribute to the problem.

Children with mild PEM have a high rate of infection particularly with gastroenteritis, measles and pneumonia. In tropical areas the risk of contracting malaria, hookworm and schistosomiasis is also increased and it is therefore important to assess the nutritional status of any child presenting with an infection or infestation. Both the morbidity and mortality due to infestations are considerably increased in children with PEM (Scrimshaw & Behar, 1961; Chandra, 1983).

Severe form of PEM – marasmus

The presenting features are gross failure to thrive accompanied by irritable crying or apathy. The child has a shrunken, wasted and stark appearance due to loss of subcutaneous fat. The fontanelle, if present, may be sunken due to dehydration and the eyes appear large. There is marked wasting of the buttocks and the

abdomen is usually distended. The degree of wasting is extreme and, by definition, the child is less than 60% of expected weight for age and may be well under 40–50% of expected weight. In chronic cases length may also be markedly affected so that the weight/height ratio may be unaltered. In acute cases the child is grossly underweight for height; skinfold thickness, mid-arm circumference and chest/head circumference are all markedly reduced. The child may be anorexic but may be ravenously hungry. There is usually chronic, watery diarrhoea but the stools may be semi-solid; vomiting may be a feature in some cases. The child is listless and physical and mental development is retarded. The child may be anaemic and may have signs of rickets.

In pure marasmus, the signs of kwashiorkor such as oedema, dermatosis, scarcity and dyspigmentation of hair, angular stomatitis, and cheilosis are not present. Children with marasmic kwashiorkor weigh less than 60% of expected weight but, in addition, have one or more signs of kwashiorkor.

Pathophysiology

There are marked changes in amount and distribution of body water, fat, minerals and total body protein.

Total body water (TBW)

There is gradual increase in TBW as a percentage of body weight. There is a direct relationship between weight deficit and total body water content (Hansen *et al.*, 1968) and hence children with most marked tissue wasting have the greatest TBW. Increase in TBW is associated with a proportionate rise in extracellular fluid (ECF) but children with oedema have more ECF than those without.

Minerals

There is a decrease in total body potassium of the order of 10–33% (Garrow *et al.*, 1965). In addition to potassium loss with nitrogen, potassium is lost in the diarrhoeal stools resulting in a cellular deficit. Mann *et al.* (1972) using a ^{40}K counting technique have shown that total body potassium is of the order of 39 mmol/kg in marasmus, 31 mmol/kg in kwashiorkor and 45–55 mmol/kg in normal controls. There is depletion of other minerals. Total body sodium, calcium and phosphorus are depleted to 93%, 77%, and 79% of expected values (Garrow *et al.*, 1965).

Body fat

Normally fat constitutes around 19% of body weight. In marasmus this may fall to around 5% and a high proportion of the residual fat (up to 50%) may be in the liver (fatty liver) although there is no relationship between liver and subcutaneous fat. In the liver the fat, mainly triglycerides, first appears in the periportal area and then spreads to the central vein area and distends the liver cells. Fatty liver is thought to result from a combination of increased flux of fatty acids from adipose tissue together with decreased hepatic synthesis of beta-lipo-proteins which normally transport triglycerides from the liver. The lipo-protein synthesis is especially sensitive to lack of protein and the liver changes are more marked in kwashiorkor than marasmus. On recovery the excess fat disappears from the liver over about 3 weeks and usually there is no residual damage unless infection or hepatic toxins are involved.

Absorption of fat and fat soluble vitamins is usually impaired, although steatorrhea does not usually occur in PEM. The following play a role in the pathogenesis of fat malabsorption: (a) reduced intestinal transit time; (b) impaired micellar solubilization of lipids due to abnormalities in bile acid metabolism caused by changes in the bacterial flora of the upper gut (Schneider & Viteri, 1974); (c) impaired intraluminal digestion due to low pancreatic lipase; (d) abnormal transport of digested lipids due to decreased beta-lipoprotein. In contrast to marasmus, the body fat in kwashiorkor may be well preserved.

Body protein

Children with PEM have a greater deficit of total body protein than body weight compared to normal children of the same height. In severe cases total body protein may be reduced to around 50%; muscle mass is particularly reduced and may be as low as 30% of normal (Montgomery, 1962) with a relative increase in interstitial collagen and reduction in cellular protein/DNA ratio.

Protein metabolism

In severe PEM there is marked hypoalbuminaemia and total albumin mass may be reduced by 50%. This occurs despite reduction in albumin catabolism (reduced by half) and is due to the extreme sensitivity of albumin synthesis to protein intake and particularly lack of the branched chain amino acids, leucine, isoleucine and valine (Waterlow & Alleyne, 1971). There is net movement of albumin from the extravascular to the intravascular pool so that the former is relatively more depleted.

Gamma globulins are not affected by PEM. Protein digestion, although slightly reduced, is adequate despite depression of pancreatic exocrine secretion; pancreatic β cell function may also be depressed although α cell function and glucagon secretion are normal.

Absorption of nitrogen from a milk diet may be reduced by 10–20% compared to normal (from 90% to 70%–80%). Nitrogen retention, however, is much more efficient and remains high during treatment until almost normal growth rates have been restored.

Changes in the gastrointestinal tract

Diarrhoea is a major problem in malnutrition and the two together have been estimated to cause over 5 million deaths among children every year (Rohde & Northrup, 1976). A vicious cycle is established whereby a child who is malnourished and more prone to infection gets repeated attacks of gasteroenteritis or protracted diarrhoea which result in further malnutrition. The pathogens responsible for the diarrhoea include rotavirus, enteropathogenic and enterotoxigenic *E.coli*, salmonella, shigellae, *Giardia lamblia*, *Entamoeba histolytica* and campylobacter. The pathogenesis of the protracted diarrhoea is rather complex and in addition to enteric infection other factors may play a part including the following.

(a) *Lactose intolerance* Many studies have shown that, in PEM, disaccharidase levels are reduced, lactase being the most severely affected, and may be low or absent in two thirds of the children. The pathogenesis of enzyme changes is not fully understood but may represent mucosal damage or decreased production due to protein deficiency. In severe cases sucrose and glucose intolerance may occur. After clinical recovery lactase levels may not recover fully although the child may tolerate milk feeds.

(b) *Abnormal small bowel mucosa* The mucosal changes may range from almost normal villi to severe villous atrophy with only convolutions or ridges being visible on microscopy. Shiner *et al.* (1973) found that in PEM there are gross epithelial cell changes with a brush border which is sparse and has markedly shortened microvilli. The nuclei were found to be irregular, the mitochondria disrupted and cytoplasmic organelles showed a lack of organization. Biopsies often show intense mucosal and submucosal infiltration by lymphocytes and plasma cells. On treatment, recovery of the jejunal mucosa may be slow and take months before it is complete. It is now established that in the small intestine villi are the site of absorption of solute and water and the crypts are the site of secretion. In children with PEM who have a protracted secretory diarrhoea the altered villus to crypt ratio may play a role.

(c) *Unconjugated bile acids* Infestation or contamination of upper gastrointestinal tract (GIT) with bacteria may lead to deconjugation of bile salts. Jejunal aspirates of children with PEM have been found to contain unconjugated bile acids (Gracey *et al.*, 1973). Unconjugated bile acids are known to be small intestinal secretagogues and may play a role in the pathogenesis of protracted diarrhoea in these children.

Immunological changes

Infection, particularly with Gram negative organisms, is very common in severe PEM, particularly of the gastrointestinal, respiratory and urinary tracts and may lead to septicaemia. The patients may not exhibit clinical signs of infection such as fever. Children with PEM have been found to have evidence of depressed cell mediated immunity (CMI) as measured by skin tests and lymphocyte transformation and may also show thymic atrophy (Smythe *et al.*, 1971; Lomnitzer *et al.*, 1976). Secretory IGA may be reduced and may play a role in infections of gastrointestinal and respiratory tracts (Reddy *et al.*, 1976).

Serum immunoglobulins are usually normal but antibody response to specific viral or bacterial antigen may be impaired (Faulk *et al.*, 1974), with obvious implications for immunization programmes. Live bacterial or viral vaccines may be more immunogenic (and hence effective) than killed ones. It has been suggested that for optimum response, immunization programmes should be preceded by a food supplementation programme.

The total white cell count is usually normal but neutrophil function abnormalities have been described (Rosen *et al.*, 1974; Sbarra *et al.*, 1974).

All components of serum complement except C4 may be reduced.

All these abnormalities are reversed with treatment and recovery.

Central nervous system changes

Apathy and irritability are well known features of severe PEM and may be related to low brain potassium but the effect on long term intellectual development has not been established. Animal experiments suggest that inadequate nutrition in early life can have a permanent effect on brain size and cell number. In humans the adult number of neurones are already present at mid-pregnancy and in order to have any effect PEM would have to be present from early pregnancy or for a long time in order to affect glial multiplication and myelinization (Dobbing, 1974). However the brains of children dying in the first year of life with severe PEM have shown reduction in the number of brain cells in the cerebrum (WHO, 1974). Clinical studies also give conflicting results as social factors cannot be completely excluded (Grantham-McGregor *et al.*, 1980; Lopez *et al.*, 1985).

Endocrine changes

The endocrine glands, particularly the adrenals and the pituitary, appear to atrophy in PEM (Trowell *et al.*, 1954) although there is no significant reduction in production of hormones. Thyroid function remains normal although thyroid function tests may be altered by the alteration in plasma protein binding. Thyrotrophin response is normal. Growth hormone level is normal and may even be supranormal in marasmus.

Circadian oscillations are abolished but plasma cortisol level may be elevated and as hypoalbuminaemia leads to reduced binding of cortisol, the level of free cortisol in the plasma may be particularly high. This may contribute to the abnormal glucose tolerance found in patients with marasmus. After an oral glucose load insulin secretion may be low but returns to normal after 3 to 6 weeks of therapy.

The raised plasma cortisol diverts free amino acids from tissue turnover to muscle rather than liver and a differential rise in plasma cortisol may be an important difference between marasmus and kwashiorkor (Lunn *et al.*, 1976).

Aetiology

Severe malnutrition leading to marasmus may stem from inadequate food intake, malabsorption, or poor utilization. However, more than one factor may be responsible and infection, particularly of the gastrointestinal tract, often plays an important role in the pathophysiology of the condition.

PEM is much less common in adults because they do not need protein for growth. In most normal adults dietary protein provides only 10% of the energy requirements.

Inadequate food intake

Marasmus, together with other forms of PEM, is common in areas of the world with insufficient food, inadequate knowledge of feeding techniques and poor hygiene. It has been estimated that 100 million children under four years of age suffer from moderate or severe malnutrition (Bengoa, 1974). A recently growing problem has been the importation into developing countries of inappropriate western models such as feeding with manufactured infant milks which have tended to replace the traditional practice of prolonged breast feeding. However, family incomes may be insufficient to buy adequate supplies of milk and facilities to prepare uncontaminated feeds may be inadequate. This is thought to have resulted in increased infant morbidity, malnutrition and mortality.

During the nineteenth century in the industrial towns of Europe and North America, marasmus resulting from poor diets and numerous infections contributed to high infant death rates which were comparable to those now encountered in many Asian, African and South American towns today. Urban influences, rapid successive pregnancies, early weaning, unsound artificial feeding, poor hygiene and repeated infections all play a role in the pathogenesis of marasmus. The reason for early weaning may be due to another pregnancy or to the belief that the milk of a pregnant mother is harmful for the child.

The reasons for inadequate food intake in the UK are given in Table III. Anorexia and feeding difficulties may complicate any illness, operation or injury in children. There may be associated vomiting and diarrhoea. In addition, metabolic expenditure may be considerably increased with negative nitrogen balance particularly in post-operative cases. The three factors of impaired intake, malabsorption and increased loss of dietary nutrients often coincide in gastrointestinal tract disease.

Malabsorption

Arateus, in the second century B.C. noted that undigested food might appear in the stools and that

Table III Causes of inadequate nutrition in the UK

	Example
Systemic diseases	renal or hepatic failure, chronic infections, congenital heart disease
Structural or physiological abnormalities of the gastrointestinal tract	severe gastroesophageal reflux, pyloric stenosis, Pierre-Robin syndrome
Non-accidental injury	maternal or environmental deprivation, emotional deprivation and/or physical injury
Psychosomatic disorders	such as anorexia nervosa, bulimia
Food fads	
Economic deprivation	
Neurological disorders	cerebral palsy
Others	drugs such as digoxin and diuretics may cause vomiting or anorexia

malnutrition could thus occur. He called this the 'coeliac disease of chronic nature' (Adams, 1856).

In 1888 Samuel Gee described the clinical features of coeliac disease and recommended withdrawal of flour from the diet: a good example of a gut disorder leading to malnutrition and on occasions marasmus which is reversible on withdrawal of dietary gluten.

Table IV Causes of malabsorption

	Example
Surgical	short gut syndrome, post gastrectomy, Hirschprung's disease
Pancreatic insufficiency	cystic fibrosis, Schwachmann-Diamond syndrome, chronic pancreatitis
Mucosal small intestinal damage	coeliac disease, acquired lactose intolerance, cows' milk protein intolerance
Intestinal infections	persistent bacterial or viral infection, parasitic infestation, e.g. *Giardia lamblia*, enterocolitis
Extraintestinal infections	urinary tract infections, recurrent respiratory tract infections, congenital infections, e.g. cytomegalovirus
Immune deficiency states	severe combined imuno-deficiency
Abnormal bile acid metabolism	ileal disease or resection, bacterial colonization of small intestine
Post-enteritis syndrome	following episode of acute gastroenteritis
Inflammation and infiltration	Crohn's disease, ulcerative colitis, amyloidosis
Malignancy	lymphomas
Venous or lymphatic obstruction	constrictive pericarditis
Selective inborn errors of absorption and digestion	glucose-galactose malabsorption, congenital chloridorrhoea
Miscellaneous	lethal familial protracted diarrhoea laxative abuse

Other causes of malabsorption, defined as the failure of the normal digestive and absorptive functions of the small intestine for at least one nutrient (Burke & Anderson, 1975), are given in Table IV.

Children presenting with protracted diarrhoea pose particular problems in diagnosis and management. The diarrhoea may be devastating and the child severely marasmic. The diarrhoea may result from a wide variety of aetiologies but in a small proportion diagnosis cannot be made despite extensive investigations (Larcher *et al.*, 1977; Sandhu & Milla, 1984).

Treatment

The present day treatment of marasmus has evolved over the years and as our understanding of the pathophysiology of PEM has improved the treatment has become more scientifically based.

Treatment of severe PEM can be divided into three phases:– resuscitation, stabilization and rehabilitation. During stabilization, if the underlying aetiology is not apparent, investigations may need to be undertaken to establish a diagnosis. In the developing countries the exact treatment used will depend on the local facilities and resources available and various regimes incorporating locally available food sources have been described (De Meyer *et al.*, 1981; Harland, 1983; Vis, 1985).

Resuscitation

The immediate causes of death in severe PEM are dehydration and electrolyte imbalance, infection, hypoglycaemia and hypothermia and these need urgent attention. Dehydration usually needs to be corrected with an intravenous isotonic solution. If the child has hypovolaemia the ideal initial fluid may be plasma or blood (20 ml/kg). If intravenous therapy is not available rehydration may be carried out using an oral glucose-electrolyte solution.

The child needs to be nursed in a warm room as marasmic children are susceptible to hypothermia. Infection may be present without pyrexia and if the child appears septicaemic or critically ill, antibiotics need to be given after blood cultures and other bacteriological specimens have been obtained.

Stabilization

During this phase of initiation of recovery, maintenance amounts of energy and protein are gradually introduced and a steady state achieved (0.6 g of protein and 95 cal/kg/day). Too rapid an introduction of protein may produce liver failure and coma, too high an energy or sodium intake may lead to cardiac failure. Oral feeding is usually started as frequent

small boluses of milk, the first few feeds may be diluted 1:1 with water. If there is lactose malabsorption, a lactose-free formula is usually necessary. The marasmic infant is prone to prolonged non-specific diarrhoea. In children who have severe protracted diarrhoea and temporary multiple food intolerance, the usual practice in the UK is to use an oligoallergenic formula consisting of comminuted chicken with carbohydrate added as a glucose polymer (Francis, 1974). Gradually long chain fatty acids, triglycerides, vitamins and mineral supplements are added. Alternatively a formula based on a casein hydrolysate, may be used or introduced after the child has been stabilized on a comminuted chicken mix.

If there is not a rapid response to dietary manipulation and the child continues to lose weight, parenteral nutrition is normally started at an early stage and may be life saving. Otherwise a vicious cycle with persistent diarrhoea, and failure to replace depleted tissues will predispose to overwhelming infection, septicaemia and death. The only way to interrupt this cycle is to provide adequate energy and protein intravenously. The intravenous solutions contain amino acids, dextrose, electrolytes, minerals and vitamins. The amount given is gradually increased and a fat emulsion can be added to the regime.

In the less developed countries peripheral alimentation has been found to be more successful than the central venous route because of the high incidence of septicaemia associated with central venous catheters in malnourished children nursed in poorly staffed units unfamiliar with parenteral feeding. Peripheral alimentation may be continued for two to three weeks while oral feeds are gradually being introduced. If the diarrhoea still poses a serious problem, particularly if the small intestine is in a secretory state, pharmacological agents with antisecretory properties may be helpful (Sandhu *et al.*, 1981, 1983).

Rehabilitation

After stabilization the child is ready to start the process of repair and growth. This demands not only adequate provision of energy and protein but also vitamins (A, B, D, C and folic acid), essential minerals and trace elements. The energy needs for growth are approximately 5 cal/g of tissue. A child weighing 6 kg and a weight of 60 per cent of the expected weight for his age and height needs $4,000 \times 5 = 20,000$ cal in addition to maintenance for catch-up growth. Rapid rehabilitation requires 150 to 200 cal/kg (Ashworth, 1980). This can be accomplished using milk or milk substitute with added vegetable oil and a diet planned with the help of a dietician with the child feeding according to appetite. The voluntary intake falls to normal values as the expected weight for height is approached. During rehabilitation mother–child interaction needs to be encouraged and improved. Developmental delay and apathy characteristic of the malnourished child can be reversed if stimulation and play are provided (Grantham-McGregor *et al.*, 1980).

Prognosis

The mortality figures from various units around the world vary considerably but it is apparent that over the last 30 years the mortality rate from severe PEM has decreased from approximately 50% to as low as 1%. The long term morbidity will depend on the underlying cause and the environment to which the child returns. In the majority of cases if the environmental circumstances are favourable, normal stature and health can be obtained (Keet *et al.*, 1971). Although it is often stated that malnutrition in early life permanently impairs intellectual development, there is little evidence to support this proposition (WHO, 1974) and environmental factors probably play a considerable role in the ultimate intellectual achievement of each child.

Prevention

Severe PEM is closely associated with infection and is more prevalent where poverty, social inequality, poor hygiene and ignorance coexist. Marasmus has almost been eliminated in the more developed countries over the last century due to improvements in living standards, health education and improved hygiene. Similar improvements are needed elsewhere in the world. Practical strategies to alleviate malnutrition and infection include adequate food supply, uncontaminated drinking water, improved waste disposal, primary health care, control of communicable diseases, and the promotion of breast feeding. There is also a need for the detection of mild and moderate PEM, whether due to malnutrition or underlying organic disease, so that the treatment can be initiated before a life threatening condition has developed.

Our understanding of the pathophysiology of marasmus and other forms of PEM has increased considerably over the last 50 years and the mortality due to severe PEM has decreased markedly. However, many fundamental questions, such as why a child with severe PEM may present with marasmus and another from the same family with kwashiorkor, and what is the precise pathogenesis of the so called post-enteritis syndrome, remain unanswered.

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