Secondary Malnutrition in Children

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Children with primary protein-energy malnutrition (PEM) are generally found in underdeveloped countries as a result of an inadequate food supply due to socioecon-omic or political factors or occasionally due to natural disasters. In more advanced nations, primary PEM is an unusual event. Nonetheless, malnutrition in more advanced nations is not at all uncommon, although its prevalence and importance are often underappreciated. The vast majority of these children are undernourished in association with some underlying disorder and in this sense have secondary PEM.

Secondary PEM has clearly been demonstrated in American hospitals in both pediatric and adult patients (1,2). In a survey by Merritt and Suskind (1), one-third of children in a large metropolitan hospital had evidence of acute PEM, half of whom had second- or third-degree malnutrition. The number of disease entities associated with significant undernutrition is extensive (Table 1). The genesis of secondary malnutrition may be attributed in a broad sense to reduced food intake, abnormal nutrient losses, or increased energy expenditure acting independently or in concert with one another (Table 2).

Anorexia may accompany any severe medical or surgical illness. A reduced appetite may be directly disease-related, as in certain children with inflammatory bowel disease who experience abdominal pain or nausea while eating (3), or it may accompany specific therapies directed against the underlying disease, as with cancer chemotherapy (4).

Unfortunately, one of the more common causes of reduced intake is iatrogenic. Extensive or prolonged diagnostic testing often results in a nil per os status and an inadequate nutrient intake. Institutionalized patients such as children with severe mental retardation are frequently given an inadequate nutrient intake, either because of ignorance or due to difficulties in chewing, swallowing, or gastroesophageal reflux. Finally, food is often intentionally withheld in acute illnesses such as acute diarrheal disease, despite the clear demonstration that weight gain is appreciable if early refeeding is instituted (5).

PEM secondary to abnormal losses of nutrients is a feature of a variety of illnesses. Increased protein catabolism and a marked loss of plasma proteins and nutrients across damaged skin barriers occur in burn patients and in children suffering
TABLE 1. Disease states often associated with protein-energy malnutrition in children

- Low birth weight infants
- Short bowel syndrome
- Cystic fibrosis
- Mucosal disease
- Celiac disease
- Milk protein enteropathy
- Infectious enteritis
- Soy protein enteropathy
- Tropical sprue
- Allergic gastroenteritis
- Intractable diarrhea
- Immune deficiency disorders
- Chronic liver disease
- Inflammatory bowel disease
- Chronic renal disease
- Congenital heart disease
- Burns and trauma
- Anorexia nervosa
- Cancer

TABLE 2. Factors related to the genesis of protein-energy malnutrition

- Reduced nutrient intake
  - Latrogenic
    - Prolonged fasting for diagnostic tests
    - Staff ignorance of nutritional assessment and needs
    - *Perioperative nil per os*
    - Therapy-related anorexia
  - Anorexia
  - Intolerance
    - Disorders of the head and neck
    - Altered levels of consciousness
    - Vomiting
    - Gastrointestinal disorders (diarrhea, obstruction, etc.)
- Abnormal nutrient losses
  - Regurgitation
  - Malabsorption
    - Luminal disorders (mucosal disease, decreased bile acids, small bowel overgrowth, etc.)
    - Pancreatic insufficiency (cystic fibrosis, Schwachman syndrome, pancreatitis)
  - Damaged skin barrier
  - Burns and trauma
- Increased energy expenditure
  - Hypermetabolic states (burns, trauma, fever, sepsis, etc.)
  - Increased work (spasticity, labored respiration, etc.)
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major trauma. Nutrient malabsorption as a result of mucosal pathology in patients with Crohn's disease, pancreatic insufficiency of cystic fibrosis, or increased stool losses in children with surgical short gut are also potential causes of secondary PEM.

Increased nutrient requirements are particularly pronounced in patients suffering trauma and large surface area burns. Children with cystic fibrosis may expend more energy through the increased work of labored respiration. Although to a lesser extent, increased expenditure also contributes in diseases such as congenital heart disease and inflammatory diseases of the bowel.

The following discussion describes specific disease states that are representative of several such disorders and mechanisms associated with secondary PEM in children. While these forces of malnutrition are operative in patients of all ages, the accelerated rate of growth normally occurring in children and adolescents serves to accentuate existing nutritional deficiencies and needs. If permitted to develop undetected and untreated, secondary PEM will predictably lead to an increased risk of infection, impaired wound healing, perioperative complications, possibly a suboptimal response to therapy or a restriction of therapy of certain primary diseases, and ultimately, an increase in overall morbidity and mortality (6–8).

PREMATURE NEWBORNS

With significant advances in the understanding and technology of the health care of preterm infants, many more very-low-birth-weight neonates are now surviving. Malnutrition has been identified as a significant problem in newborn nurseries. In a survey conducted by Cooper et al. in Philadelphia (9), 63% of all infants less than 3 months of age, including 93% of the premature infants, were malnourished, the majority with severe PEM. Premature newborns present a number of nutritional management difficulties. Since the fetus grows rapidly in utero, particularly in the last trimester, it is evident that if the fetus is born prematurely, the resultant very-low-birth-weight newborn will require a sufficient energy intake to satisfy the demand for this rapid growth. However, while the ability to provide adequate nutrition has reduced the morbidity and mortality of the premature infant, prompt resumption of an intrauterine growth rate may not be safe or necessary and is probably not possible in the majority of children (10).

Preterm infants usually have what is commonly referred to as "intestinal immaturity," as manifest by their poor ability to suck and swallow, increased gastroesophageal reflux due to a relatively incompetent gastroesophageal junction, delayed gastric emptying, and decreased intestinal motility (11,12). Similarly, digestion and absorption of fat, protein, and carbohydrate are markedly diminished in these infants as compared with term infants or older children (13). The enteral delivery of nutrients is further impaired because of the tendency of the very-low-birth-weight infant to develop necrotizing enterocolitis if early enteral feedings are overly aggressive (14). Other systemic disorders associated with prematurity such as respiratory distress, cardiovascular abnormalities, and renal immaturity may also limit enteral
intake. However, a trial of continuous feedings of human milk, isotonic formula, or hypotonic formula administered intragastrically or via the transpyloric route may be tolerated (15). Parenteral nutrition in conjunction with enteral tube feedings or alone may be necessary to deliver adequate nutrition.

**CONGENITAL HEART DISEASE**

It is well-established that children with uncorrected congenital heart disease (CHD) are often malnourished and fail to thrive, and they frequently have profound growth retardation. In a study of 890 children of both cyanotic and acyanotic CHD, more than 50% were below the 16th percentile and 27% below the 3rd percentile for height and weight (16). While growth retardation is more frequent and severe in cyanotic than acyanotic CHD, it is also pronounced in those children with acyanotic CHD who have concomitant pulmonary hypertension and large left to right shunts (17).

Genetic or congenital factors account for a small number of children with CHD and growth failure (18). Most of these children are usually born small-for-gestational-age and often fail to show catch-up growth when provided with a sufficient energy intake, suggesting an inherent abnormality of growth rather than growth failure related to cardiac disease and malnutrition (19). Prenatal infection such as rubella, as well as maternal ingestion of specific drugs including thalidomide, lithium, dilantin, and alcohol, have all been associated with cardiac defects and inherently impaired growth. CHD and growth failure may also be components of such genetic syndromes as trisomy 15 and trisomy 18 (20).

However, growth failure in the great majority of children with CHD is due to abnormalities of nutrition. There are several causes of growth failure in this group of children. Insufficient nutritional intake is the single most important factor in the genesis of malnutrition in children with CHD (18–21). Anorexia due to fatigue and shortness of breath results in decreased intake, while an increased respiratory effort, increased heart size, and recurrent pulmonary infection demand a greater energy intake. A role for hypoxia in the development of malnutrition has been inferred, since surgical repair of the cardiac lesion and supplemental oxygen can improve growth (22). Although growth failure is more severe in cyanotic CHD, the severity of cyanosis does not correlate with the severity of the growth impairment (17). Therefore, while hypoxia plays a role, it appears to be important only when associated with excessive lactic acid production as a result of anaerobic metabolism (18).

Abnormal intestinal losses of energy as a result of fat malabsorption and protein-losing enteropathy are present in many children with CHD (23). Energy metabolism in infants with CHD, as measured by the basal metabolic rate, is generally increased, although the data are somewhat contradictory (19). This hypermetabolic state is likely to be due to the increased work of breathing that accompanies severe congestive heart failure. Because of abnormal intestinal losses and increased energy expenditure, the energy requirements of infants with CHD and growth failure may be increased to 175 to 180 kcal/kg-day (24). With the provision of sufficient energy, these infants show weight gain and a positive nitrogen balance (25,26).
Enteral intake of energy can be increased by increasing the concentration of formula and by the addition of medium-chain triglycerides and/or glucose polymers. However, an increased intake of energy-dense formula occurs at the expense of a decreased free water intake and a potential increased solute load, thereby limiting the extent of concentration. In children with significant malabsorption of fat and protein, the use of a semielemental formula such as Pregestimil may be beneficial. Unfortunately, voluntary oral intake is often insufficient, particularly in infants and younger children. In these children, nasogastric feedings have been shown to reverse as well as actually to prevent growth failure (25,27). Despite concerns of volume overload, newborns and infants generally tolerate the increased volume necessary to deliver the required energy, particularly when provided as slow continuous nasogastric feedings.

INFLAMMATORY BOWEL DISEASE

Inflammatory bowel disease (IBD) consists of two related but distinct idiopathic intestinal disorders, Crohn’s disease and ulcerative colitis. The etiology of IBD is unknown, and the diagnosis is made by a combination of clinical, pathologic, endoscopic, and radiologic features. One-fourth to one-third of all patients with Crohn’s disease and 15% of patients with ulcerative colitis present before age 20 years. 30 to 40% of them develop PEM and multiple vitamin and mineral deficiencies (28). Growth arrest and delay in maturation may predate the onset of other clinical features by months to years. While both nutritional and disease-related factors contribute to the genesis of malnutrition, an inadequate intake of nutrients is the most important factor, as shown by reversal of malnutrition and growth failure with the provision of supplemental nutrition (29,30).

Children with IBD often have a decreased intake, primarily because of anorexia related to intestinal inflammation or altered taste sensation. Eating may precipitate noxious symptoms including abdominal pain, diarrhea, or nausea and vomiting. A poorly understood but characteristic early satiety may also contribute to poor energy intake, which, together with the unpleasant eating-evoked symptoms, may produce a conditioned food aversion.

Excessive fecal loss of nutrients, particularly nitrogen, may occur secondary to mucosal inflammation or diarrhea. Chronic intestinal inflammation forms the basis for malabsorption of fats and carbohydrates, as well as a protein-losing enteropathy (31,32). Vitamins and mineral insufficiencies occur secondary to defects in absorption and excessive urinary excretion (33). Small bowel overgrowth, present in up to 30% of individuals with Crohn’s disease, and surgical small bowel resections may precipitate or enhance absorption abnormalities (34).

Inadequate energy intake and increased nutrient losses occur against a background of the need for additional energy for normal growth of the child and adolescent and may further magnify the deficiencies. The primary goal of medical treatment of IBD is the diminution of the inflammatory process, controlling symptoms through the appropriate use of certain pharmacologic agents and through nutritional repletion and maintenance. Steroids and/or sulfasalazine historically have
proved to be the most useful agents to achieve remission of intestinal disease. Nutritional therapy, delivered as total parenteral nutrition or liquid elemental diets, has been equally efficacious in inducing remission of Crohn's disease; however, relapses occur sooner and more often (35). Nonetheless, a role for nutrition as adjuvant therapy for inducing disease remission has been clearly established.

There are several ways to deliver supplemental nutrition in patients with IBD. In mild disease or disease in remission, the most practical method is to increase the intake of a normal diet. In this regard, avoidance of restrictive diets is crucial (36). While this approach is often effective, certain children will require additional supplements with commercially prepared liquid formulas. These can usually be taken as in-between meal snacks. Because of problems with anorexia or postprandial symptoms, nighttime continuous nasogastric or nasojejunal tube feedings may be preferable in selected patients (37). Peripheral or central parenteral nutrition has proved useful during periods when patients cannot tolerate or absorb enteral feedings and if necessary can be administered chronically at home. In all cases, specific vitamins and minerals should also be provided. Nutritional support given enterally or parenterally not only prevents and treats PEM in children with IBD, but also stimulates catch-up growth and the onset of puberty (29,38).

CYSTIC FIBROSIS

Cystic fibrosis (CF) is the most common lethal or semilethal disease in the Caucasian population, with a prevalence of about one per 2,000 live births in the United States (39). CF is also the most common cause of exocrine pancreatic insufficiency in infants and children in North America and Western Europe. Clinically, CF is a generalized disease of the exocrine glands associated with exocrine pancreatic insufficiency and malabsorption as well as chronic obstructive lung disease with recurrent infections leading to respiratory failure and death. Hallmarks of pancreatic involvement in CF include fat and protein maldigestion through fecal losses. Impetus for aggressive nutritional support of children with CF is the result of careful clinical studies, which have shown that PEM is common and that a relative weight deficit is a major factor adversely affecting survival (40-42). Pulmonary function tests and overall prognosis are significantly better in patients with normal fat absorption as compared with patients who have steatorrhea (43). Therefore, reversible nutritional factors probably have an influence on the course of pulmonary disease in patients with CF and deteriorating lung function.

In addition to stool losses, patients with CF have an increase in energy requirements that occurs with recurrent respiratory tract infections. Increased respiratory effort and work in advanced pulmonary disease lead to an increased energy expenditure, which accentuates the need for additional energy intake (44). Observations by Shepherd et al. (45) are suggestive of an inherently increased energy expenditure in children with CF, which is independent of chronic lung disease or infections. The net result is that excluding stool losses, children with CF have energy requirements of 120 to 150% of normal children, depending on the severity of their respiratory in-
sufficiency. Yet, most children do not meet these needs unassisted. This is complicated by the anorexia that generally accompanies their compromised pulmonary function and respiratory tract infections. Vomiting induced by paroxysmal cough may further reduce energy intake.

Therapy is directed at enhancing intestinal absorption with the use of pancreatic enzyme supplements. Fat-soluble vitamins are also provided, and serum levels should be periodically monitored. The use of restricted diets should be avoided, as they too often lead to meals that are unpleasant in taste and appearance and are, therefore, avoided by the younger patients. As pancreatic enzyme supplements may not entirely resolve the steatorrhea no matter how high the dose, nutritional supplementation beyond the spontaneous normal intake may be required. This is usually done in a stepwise fashion starting with the oral ingestion of commercially prepared high-energy liquid supplements. In the event that the child is unable to comply with the initial regimen, consideration should be given to nighttime nasogastric tube feedings or nighttime gastrostomy tube feedings, preferably with an elemental or semielemental formula to optimize energy absorption without the need for pancreatic enzyme supplements (42,46). Additionally, aggressive antibiotic treatment of pulmonary infections improves nutritional intake over the course of many weeks and months.

The long-term goal in the management of nutritional deficiencies in children with CF is to stabilize pulmonary function and reduce the frequency of respiratory tract infections (47). Although aggressive nutritional support clearly results in improvement, the underlying disease process is such that a cycle of pulmonary destruction, chronic infection, and further tissue damage is established. The combined effect ultimately results in the progressive deterioration of pulmonary function and eventual death of all patients. Long-term mortality rates are, therefore, unaffected, and the benefits of nutritional therapy are probably to reduce "premature" mortality and morbidity and to improve the quality of life.

CHRONIC LIVER DISEASE

Nutritional disorders in children with liver disease result from the combined effects of hepatocellular damage, hepatic fibrosis and cirrhosis, and obstruction of bile flow. The development of cirrhosis after liver injury is the common end result of many hepatic diseases. Childhood liver disease of this nature is generally associated with mild to severe PEM (48).

Synthesis of proteins such as albumin, clotting factors, and complement among others is impaired with the loss of functioning hepatocytes (49,50). These "vital" proteins are critically involved in mechanisms of hemostasis and immunity, so it is not unexpected that hemorrhage and sepsis constitute the most common final pathways leading to death in children with end-stage liver disease. With the loss of urea production, ammonia generated by intestinal bacteria and body tissue accumulates, contributing to the development of hepatic encephalopathy. A key component of therapy for hepatic encephalopathy is the restriction of dietary protein. Somewhat
paradoxically, sufficient protein must be supplied in order to prevent muscle protein catabolism, a process that may actually augment the hyperammonemia. Nutritional therapy in this instance is designed to be protein-sparing, with lipids and carbohydrates satisfying most of the energy requirements. A trial of branched-chain amino acids can be used in an attempt to compete with aromatic amino acids, thereby reducing cerebrospinal fluid (CSF) concentrations of false neurotransmitters contributing to the development of hepatic encephalopathy (51).

The nutritional consequences of conditions causing biliary cirrhosis may be severe, with extrahepatic biliary atresia and congenital or acquired strictures of the biliary system being representative of this group of disorders. Decreased or absent excretion of bile acids into the proximal small intestine leads to malabsorption of fats and fat-soluble vitamins (52). Since long-chain triglycerides are poorly absorbed when intraluminal bile salts are diminished, nutritional maintenance and repletion in children with cholestasis are best accomplished through the use of medium-chain triglycerides (MCT) as the source of dietary fat (53). MCT are "elemental" in the sense that they do not require further solubilization by bile acids for absorption.

It is of note that fat-soluble vitamin absorption is generally far more impaired than absorption of dietary fat. Monitoring fat-soluble vitamins (A, D, E, and K) and supplementation with their water-miscible forms when available are essential. Vitamin E in particular may be completely malabsorbed, despite massive oral doses. Vitamin E deficiency in children with chronic liver disease is associated with a potentially reversible degenerative neurologic syndrome characterized by a defined pattern of progressive neurologic symptoms (54). Intramuscular vitamin E or an experimental vitamin E preparation, d-alpha-tocopherol polyethylene glycol-1000 succinate (TPGS), may ultimately be required.

Other factors resulting in malnutrition in children with hepatic disease include anorexia, early satiety, vomiting, and gastroesophageal reflux as the result of infection or compression of abdominal and thoracic viscera by ascites and organomegaly. The presence of portal hypertension may lead to increased pressure of the intra-abdominal lymphatics and reduced absorption of chylomicrons.

Calcium, zinc, and iron status should be closely monitored, since abnormal losses of these elements may occur from fat malabsorption, urinary losses, deficiency of transport proteins, or gastrointestinal bleeding.

With the advent of liver transplantation as a therapeutic option and potential cure for many otherwise lethal liver diseases, the relevance of optimal nutritional management is immediately recognized. The child with end-stage liver disease who is the benefactor of good nutritional management may be able to survive the waiting period required by the lack of suitable donor organs for transplantation and be better able to survive perioperative complications associated with poor nutritional status.

CHRONIC RENAL DISEASE

Growth retardation is the rule in children with chronic renal failure (CRF) and is the product of multiple factors, including hormonal, metabolic, and nutritional ab-
normalities. Linear growth is decreased in 35 to 65% of children with renal insufficiency and more than half of children with end-stage renal disease (55,56). It is well-established that undernutrition is particularly widely prevalent in these children and that PEM is associated with more severe growth failure (57,58), independently or in combination with other factors. PEM develops in children with CRF primarily due to a very low oral energy intake, below the daily minimum requirement for maintenance and much below that needed for growth (59).

Children with CRF commonly develop signs and symptoms of PEM, such as an increased serum growth hormone, decreased somatomedin-C, low weight-for-height, stunted growth, decreased muscle mass, decreased skin fold thickness, and other evidence of protein depletion including low serum albumin concentration (57,60). Food intake is reduced as a result of anorexia due to "uremic toxins," altered taste sensation, and other problems associated with CRF such as hypertension or drug therapies. Mental depression, food aversions, or manipulative behavior contributes in certain children.

Even so, a consistent relationship between energy intake and altered growth has not been identified, an observation that reflects the complex pathogenesis of growth failure in CRF (61). Nonetheless, most of the published data suggest that additional energy intake will improve growth and restore tissue mass, especially in those children who are receiving less than 70 to 80% of the recommended daily allowance (RDA) for height (62). Careful control of acidosis, other metabolic abnormalities, and renal osteodystrophy is obviously important as well (63).

Growth retardation is more prevalent in children with congenital than acquired renal disease, and in large part represents the stunting of chronic malnutrition (62). As in children with primary PEM, hypercaloric intakes in these children rarely result in catch-up growth, although growth velocity may remain normal. The first several months of life in children with congenital renal insufficiency or failure have been identified as critical periods for the child’s ultimate height. Perhaps of significance, therefore, is the observation that growth rate was improved over the first year of life in a group of infants with end-stage renal disease whose energy intakes were adjusted to allow normal or catch-up weight gain for height (64).

Because of the multiple therapeutic options for children with CRF, including conservative management, hemodialysis, intermittent or continuous peritoneal dialysis, and transplantation, a universal "renal failure" diet is nonexistent (59). However, as reduced food intake is so common in children with CRF, nutritional therapy is directed at supplementation of the spontaneous oral intake with additional palatable energy, often as nasogastric feedings (64). Through high-energy supplementation in both nondialyzed and dialyzed children with chronic renal insufficiency or chronic renal failure, many investigators have induced improved growth rates (65).

High-quality protein should be provided in an amount that minimizes uremia as a metabolic end product, yet in sufficient quantities to prevent endogenous muscle catabolism and the subsequent production of nitrogenous wastes (64).

It is reasonable to conclude that while some studies suggest growth and weight gain are not solely related to food intake and may be related to the metabolic abnormalities of CRF, it is clear that a trial of energy supplementation is warranted in
children with CRF and growth failure. Resolution of malnutrition is not only associated with improved growth in most children with CRF, but an improved sense of well-being, activity level, and perhaps cognitive development (66).

**CANCER**

The importance of the relationship between cancer and nutrition is clear, although not without its complexities. Surveys have shown that PEM exists in 10 to 17% of children with newly diagnosed tumors and in as many as 38% of all inpatient pediatric cancer patients with advanced disease (67,68). The presence of malnutrition in childhood cancer is associated with the type of tumor, extent of the disease, the form of therapy, as well as the age of the child (69).

Certain malignancies predispose to the development of malnutrition as a result of the tumor itself, its anatomic location, or type of therapy specific for tumor type. Patients with advanced solid tumors, lymphomas, and leukemias may have an accelerated basal metabolic rate (70). Malignancies of the oropharynx interfere with normal feeding because of mechanical obstruction or as a result of head and neck surgery. Specific antitumor therapy as a determinant of PEM is exemplified by the treatment of osteosarcoma. With the advent of cis-platinum and high-dose methotrexate as the chemotherapy for this tumor, osteosarcoma has become one of the tumors often associated with significant malnutrition (71).

A marked reduction in nutrient intake occurs within the context of the notable "cancer cachexia" syndrome of progressive weakness, weight loss, and wasting (72). The precise mechanism(s) of this phenomenon is unknown. However, many investigators suspect a humorally mediated cause such as tumor metabolites or peptides, or an abnormal production of serotonergic neurotransmitters (73,74). The cytokines cachectin (tumor necrosis factor) and interleukin I are also potential candidates, among others, as mediators of cancer cachexia (75).

Anorexia related to surgery, chemotherapy, or radiation therapy is common (76). Depending on the regimen, nausea and vomiting can be severe (71,77). Infectious, chemical, or radiation-induced mucositis or esophagitis may precipitate odynophagia or dysphagia (78). There is also considerable concern that these adverse reactions may evoke feeding-aversion behavior in certain children as a conditioned response to multiple intermittent courses of antitumor therapy (79). Finally, anorexia due to a reactive depression most certainly plays a role in many children.

Malabsorption of ingested fat, protein, and carbohydrate, as well as losses from exudative protein-losing enteropathy, also contributes significantly to the development of PEM in many children with cancer (78,80,81). This disordered nutrient absorption is generally iatrogenic, in that it is caused by surgery, chemotherapy, radiation therapy, or graft-versus-host disease and to a lesser extent by the underlying malignancy (76).

Clinical studies have produced evidence for an increased energy requirement in cancer patients. Increased resting energy expenditure is associated with certain tumors, although most studies to date are confounded by multiple variables
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(70,81,82). Infectious complications, which are so very common in these children, extract a high metabolic cost as well.

Vigorous attention to nutritional support is a prerequisite. This can be accomplished via enteral feedings, with elemental diets (Vivonex, Vital, etc.), if necessary. However, because of the many gastrointestinal abnormalities, total parenteral nutrition is often required. Despite concerns about serious catheter-related infections in children with cancer, intravenous nutrition is usually well-tolerated, even in the presence of neutropenia (83). While there is no evidence that nutritional factors have a direct etiologic role in any type of malignancy, it is clear that a good nutritional state enables a more aggressive and, therefore, potentially more efficacious treatment regimen, improves the response to chemotherapy, and may actually reduce the rate of relapse (8,84). Furthermore, the direct relationship of malnutrition to infection, a leading cause of death in children with cancer, demands active attention to nutritional therapy and support.

SUMMARY

Thus, children with primary organ failure such as congenital heart disease, chronic liver disease, or chronic renal disease need to have recognized, documented and treated their secondarily malnourished state. The secondarily malnourished child needs as much nutritional support as does his primarily malnourished counterpart.

REFERENCES


