

## Nutritional Support of the Cancer Patient

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### Introduction

Malnutrition is a common problem in cancer patients that results in devastating quality-of-life, economic, and survival issues. "Cancer cachexia" refers to a complex, multifactorial syndrome characterized by anorexia or the spontaneous and unintended loss of appetite, generalized host tissue wasting, skeletal muscle atrophy, immune dysfunction, and a variety of metabolic alterations. The malnourished cancer patient responds poorly to therapeutic interventions, such as chemotherapy, radiotherapy and surgery, with increased morbidity and mortality com-

pared with well-nourished patients.

Many studies have reported the prevalence of malnutrition in cancer patients. In a multicenter cooperative study of more than 3,000 cancer patients, DeWys et al<sup>1</sup> reported substantial weight loss in more than 50% of patients. The highest frequency and severity of weight loss occurred in patients with gastrointestinal malignancies. Weight loss also was identified in 40% of patients with breast cancer and 60% of patients with lung cancer. The high rate seen in patients with nongastrointestinal cancers emphasizes the fact that cancer-induced malnutrition involves systemic and metabolic derangements.

A Veterans Affairs cooperative study documented that 39% of patients undergoing major cancer operations were malnourished, as determined either by a history of weight loss and low serum protein levels or by a nutritional risk index.<sup>2</sup> Brennan et al<sup>3</sup> reported preoperative nutritional impairment in most patients undergoing pancreatic resections, with an average body weight loss of 6%. In a recent study, Wigmore et al<sup>4</sup> reported an average loss of 14% of body weight at initial diagnosis in patients with unresectable pancreatic cancer.

Cancer cachexia is the result of complex and multifactorial causes. The derangements of cancer cachexia can be grouped in two major categories: (1) suboptimal nutrient intake and malabsorption and (2) host metabolic alterations.

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**Table 1**  
**Factors Contributing to Decreased Intake of Nutrients**

<p><b>Reduced Oral Intake</b> Anorexia Nausea, vomiting Altered perceptions of taste and smell</p> <p><b>Local Effects of Tumor</b> Odynophagia, dysphagia Intestinal or gastric obstruction Early satiety Malabsorption</p> <p><b>Psychosocial Factors</b> Depression, anxiety Food aversion</p>	<p><b>Effects of Cancer Treatment</b></p> <p><b>Surgery</b> Altered mastication and swallowing Postgastrectomy syndromes Pancreatic insufficiency Anastomotic stricture</p> <p><b>Chemotherapy</b> Nausea, vomiting Altered perceptions of taste and smell Stomatitis, mucositis Diarrhea</p> <p><b>Radiation therapy</b> Odynophagia, dysphagia Xerostomia, mucositis Strictures, fistulas</p>
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### Decreased Nutrient Intake

The decreased food intake seen in cancer patients may result from both direct and indirect alterations caused by tumor (Table 1).

Gastrointestinal tract malignancies—such as oropharyngeal, esophageal, and gastric tumors—may cause luminal obstruction and lead to the physical inability to ingest adequate nutrients. External impingement on the gastrointestinal tract, as seen with abdominal and retroperitoneal tumors and metastatic tumors from melanoma and ovarian cancers, may cause a substantial decrease in oral intake.

Indirect disturbances leading to anorexia include alterations in perception of taste<sup>5-7</sup> and smell<sup>8</sup> and abnormalities in the central nervous system that control food intake and the sensation of early satiety.<sup>9</sup>

The exact agents mediating tumor-induced anorexia in the central nervous system have not been defined clearly. In a model consisting of surgically coupled tu-

mor-bearing and normal rats with parabiotic cross-circulation, Norton et al<sup>10</sup> showed that anorexia and weight loss were mediated by a circulating substance rather than solely by the tumor. This observation led to the understanding that tumor-induced anorexia and cachexia are most likely mediated by multiple cytokines produced by host monocytes in response to the tumor-bearing state.

Studies have implicated cytokines produced by host and tumor (e.g., interleukin-1,<sup>11,12</sup> tumor necrosis factor [TNF]- $\alpha$ ,<sup>13,14</sup> and interleukin-6<sup>15,16</sup>) as mediators of the metabolic abnormalities seen in cachectic patients. Increases in the levels of these cytokines and other substances have been documented in tumor-bearing hosts.

It is now assumed that the host generates an immune response in which cytokines are produced in an effort to eradicate the tumor. As the host fails to accomplish this task, the chronic production of cytokines leads to the detrimental metabolic alterations seen in cancer

patients. Some even propose that host production of cytokines actually may play a role in maintaining tumor growth.<sup>17</sup>

Treatments such as surgery, chemotherapy, and radiation also can contribute to anorexia and weight loss in cancer patients.

Patients who have undergone surgery of the oropharynx, esophagus, and stomach may have a decreased ability to ingest food because of physical alterations resulting from the surgery. Extensive resections of the small bowel can lead to malabsorption of fluids and nutrients, causing stomatitis, cheilosis, glossitis, and pharyngitis. This contributes to an already tenuous nutritional intake, leading to cachexia and malnutrition. Chemotherapy may cause mucositis, ileus, and malabsorption.<sup>18,19</sup> Furthermore, food aversions may develop because of nausea and emesis.

Edema and mucositis induced by head and neck radiation therapy can cause severe dysphagia and odynophagia in patients with head and neck cancers, resulting in malnutrition and dehydration. These complications can be reduced by the placement of percutaneous endoscopic gastrostomies and home enteral nutrition until the effects of radiation subside.<sup>20</sup>

Patients receiving radiation therapy to the abdomen frequently experience mild episodes of nausea, emesis, abdominal pain, and diarrhea. These symptoms are usually mild and resolve after radiotherapy is completed. A small percentage of patients, however, develop chronic radiation enteropathy. This can result in severe, multiple gastrointestinal strictures and fistulas leading to significant nutritional deficiencies and malnutrition requiring long-term nutritional support.<sup>21,22</sup>

### Metabolic Alterations

Alterations in the metabolism of protein, carbohydrates, fat, minerals, and vi-

tamins are widespread in cancer patients. Therefore, the treatment of malnutrition in these patients not only must deal with the derangements in nutritional intake but also must address inefficient or aberrant nutrient utilization. The metabolic alterations found in cancer cachexia differ from those seen in starvation (Table 2).

#### ALTERATIONS IN ENERGY EXPENDITURE

One might assume that energy expenditure must be increased in cancer patients with tissue wasting and weight loss. However, no agreement exists about changes in energy expenditure in cancer patients, and a wide range of results have been reported citing both increased and decreased levels.<sup>23-29</sup>

Russell et al<sup>27</sup> reported a 37% increase in basal energy expenditure (BEE) in patients with newly diagnosed small-cell lung cancer and showed a substantial decline in BEE in patients who responded to chemotherapy. Elevations in basal metabolic rates have been reported in patients with a variety of other tumors, such as gastric cancer<sup>24</sup> and sarcomas.<sup>26</sup> Additionally, Luketich et al<sup>29</sup> showed a decrease in energy expenditure in patients who underwent tumor resection.

#### ALTERATIONS IN CARBOHYDRATE METABOLISM

Significant abnormalities in carbohydrate metabolism, including glucose intolerance and peripheral insulin resistance, have been observed in cancer patients. In contrast, euglycemic or hypoglycemic states are found in nonstressed starved patients.

Several reports indicate that in response to a glucose challenge, hyperglycemia and delayed glucose disappearance occur frequently in cancer patients.<sup>30,31</sup> Cersosimo et al<sup>32</sup> showed that peripheral glucose uptake was impaired even with high-dose insulin infusions. These metabolic derangements

**Table 2**  
**Metabolic Alterations in Starvation**  
**Compared with Those in Cancer Cachexia**

	<b>Starvation</b>	<b>Cancer Cachexia</b>
<b>Resting energy expenditure</b>	Decreased	Normal/increased
<b>Carbohydrate metabolism</b>		
Glucose tolerance	Decreased	Decreased
Insulin sensitivity	Decreased	Decreased
Glucose turnover	Decreased	Increased
Serum glucose level	Decreased	Unchanged
Serum insulin level	Decreased	Unchanged
Hepatic gluconeogenesis	Increased	Increased
Serum lactate level	Unchanged	Increased
Cori cycle activity	Unchanged	Increased
<b>Fat metabolism</b>		
Lipolysis	Increased	Increased
Lipoprotein lipase activity	Unchanged	Decreased
Serum triglyceride level	Unchanged	Increased
<b>Protein metabolism</b>		
Protein turnover	Decreased	Increased
Skeletal muscle catabolism	Decreased	Increased
Nitrogen balance	Negative	Negative
Urinary nitrogen excretion	Decreased	Unchanged

Adapted with permission from Gentilini et al.<sup>74</sup>

have been observed most frequently in the latter stages of the disease process and have been associated with extensive metastasis.<sup>33</sup>

Increased endogenous glucose production from hepatic gluconeogenesis occurs in cancer patients; in contrast, reduced rates of gluconeogenesis are seen in nonstressed starved patients.<sup>30,34</sup> It has been reported that glucose turnover rates increase as the extent of the tumor burden increases.<sup>35,36</sup> A study by Shaw and Wolfe<sup>36</sup> observed a correlation between increasing tumor burden and greater glucose production. They showed decreased glucose use after curative resection of the tumor.

High rates of glucose turnover may be caused, in part, by the increased rates of the Cori cycle. This cycle involves the metabolism of glucose to lactate by tumor cells and the conversion of the lactate to glucose in the liver. The conversion of lactate to glucose results in a net loss of adenosine triphosphate (ATP) and appears to be an energy-wasting biochemical process that may contribute to the tissue wasting and weight loss seen in cancer patients.<sup>37,38</sup>

**ALTERATIONS IN FAT METABOLISM**

Extensive abnormalities in fat metabolism in cancer patients contribute to the excessive loss of fat mass relative to lean

body mass.<sup>35,39</sup>

The decrease of fat mass in cancer patients who lose weight has been related to increased rates of lipolysis with increased free fatty acid and glycerol turnover compared with those of cancer patients who do not lose weight.<sup>36,40</sup> Under normal conditions, glucose infusion suppresses lipolysis, but this effect is reduced in cancer patients.<sup>36</sup> In addition, cancer patients who lose weight are usually hyperlipidemic.<sup>41</sup> Hyperlipidemia has been linked to decreased lipoprotein lipase activity, possibly modulated by the cytokine TNF- $\alpha$ .<sup>42</sup>

#### ALTERATIONS IN PROTEIN METABOLISM

Another common observation in malnourished cancer patients is the increase in whole body protein turnover<sup>43</sup> and subsequent loss of body nitrogen, which have been reported to occur in 50% to 70% of cancer patients.<sup>34,44</sup>

The phenomenon occurs in spite of weight loss, which conversely results in an adaptive decrease in protein turnover. Therefore, the cancer patient has a relative inability to conserve protein, resulting in constant muscle catabolism with subsequent depletion of muscle mass.<sup>45</sup> This inappropriate protein breakdown provides substrate for gluconeogenesis, which cannot be fully suppressed even with glucose infusion.<sup>36</sup> Studies have shown that total parenteral nutrition (TPN) does not suppress the protein turnover.<sup>45</sup> Body nitrogen losses continue in spite of increases in body fat and total weight gain.<sup>46</sup> Clearly, the cancer patient with cachexia has metabolic abnormalities that differ substantially from those of patients with simple starvation. The malnourished cancer patient is unable to conserve energy. Aberrant adaptation to weight loss plus numerous energy-consuming and inefficient metabolic derangements render reversal of the malnourished state extremely difficult, even with the provision of nutritional support.

#### Nutritional Assessment

Nutritional assessment provides an estimate of body composition, such as fat, skeletal muscle protein, and visceral protein. This helps to identify patients who are at risk of cancer-induced malnutrition and to estimate the magnitude of nutritional depletion in patients who are already malnourished. Several variables are used to assess nutritional status in patients. These assessment techniques include a thorough patient history and physical examination and specific laboratory tests.

Nutritional assessment should start with patient history and physical examination. The history should reveal usual body weight, any recent weight change, or incorporation of new or special diets. Unintentional weight loss of 10% or more of body weight within the previous 6 months signifies a substantial nutritional deficit and is a good prognosticator of clinical outcome.<sup>1</sup> Physical examination may reveal signs of malnutrition such as muscle wasting, loss of muscle strength, and depletion of fat stores.

Anthropometric measurements quantify body compartments and correlate them with values from age- and sex-matched normal populations. Mid-arm muscle circumference provides a measure of muscle mass. Subscapular and triceps skinfolds represent an index of body fat. These values may vary with the patient's hydration status.

Concentrations of serum proteins such as retinol-binding protein, transferrin, prealbumin, and albumin can be used to estimate the degree of visceral protein depletion.

The relationship between malnutrition and serum protein levels is related to the hydrational status of the patient and the half-life of the individual protein. Prealbumin and retinol-binding protein have the shortest half-lives, 0.5 and 2.0 days, respectively. Therefore, prealbumin and retinol-binding protein levels are depressed initially and reflect recent dietary

changes. Transferrin and albumin have the longest half-lives, 8.0 and 20.0 days, respectively. Decreases in the serum concentration of these proteins are related to longer periods of nutritional deficiency.

Several studies have shown that a low serum albumin concentration predicts a poor prognosis in cancer patients.<sup>47,48</sup> Reasons for the depressed concentrations of albumin in cancer patients include decreased synthesis, increased protein turnover, and increased transcapillary losses. Transcapillary losses may result in a significant amount of albumin (up to 29% of total body albumin) being sequestered in ascites or pleural effusions.

Objective data can be used to define the nutritional status of a patient. The Prognostic Nutritional Index (PNI) proposed by Buzby and colleagues<sup>49</sup> has been shown to predict clinical outcome in cancer patients. The PNI is based on serum albumin level, serum transferrin level, triceps skinfold thickness, and delayed cutaneous hypersensitivity. The nutritional status scale uses changes in ideal body weight and levels of serum albumin, serum transferrin, total body nitrogen, and potassium. It can be used for classifying patients into nutritional categories of normal status, mild malnutrition, or severe malnutrition.<sup>50</sup>

Several studies have reported hand grip strength as an indicator of nutritional state and have shown a correlation between it and postoperative complications.<sup>51,52</sup> Malnutrition results in alterations of muscle function by blunting the response to electrical stimuli. In addition, changes in muscle function in response to malnutrition occur before any changes are observed in body nitrogen or protein concentrations.

### Nutritional Support

The goal of nutritional support in the cancer patient is to prevent or reverse the cachexia of malignancy. Nutritional therapy can be used as adjuvant treatment

during anticancer therapy or as a long-term administration of nutrients to patients unable to maintain adequate intake. Nutrients can be delivered by the oral, enteral, or parenteral route.

### ORAL NUTRITION

Oral supplementation is the preferred modality in patients who are able to eat but require special diets because of impairments in the gastrointestinal tract.

The oral diet is modified based on the physiologic and anatomic constraints of the disease process. Examples include multiple small meals that are low in monosaccharides in patients after gastric resections and the use of soft foods with an emphasis on high-calorie liquid nutritional supplements in patients with esophageal strictures. Many of these supplements are commercially available and are particularly useful when the main problem is inadequate food intake.

The main drawback with use of these oral supplements is taste fatigue. Nevertheless, such supplements can be invaluable in providing outpatient nutrition. They avoid more complex and costly methods of enteral or parenteral feeding.

### ENTERAL NUTRITION

Enteral feeding provides nutrients to the gastrointestinal tract by a catheter or tube. As does oral feeding, it requires absorption of nutrients by the gastrointestinal tract.

Enteral feeding is preferred to parenteral feeding because it preserves the gastrointestinal architecture and prevents bacterial translocation from the gut. This method has fewer complications and is less expensive than TPN.

Compared with oral feeding, enteral nutrition has several advantages, such as the ability to deliver nutrients beyond obstructed areas, particularly in patients with malignancies of the oropharynx, esophagus, and stomach.<sup>53</sup> Nutrients can be delivered at a slow, continuous rate, thereby permitting a longer period for

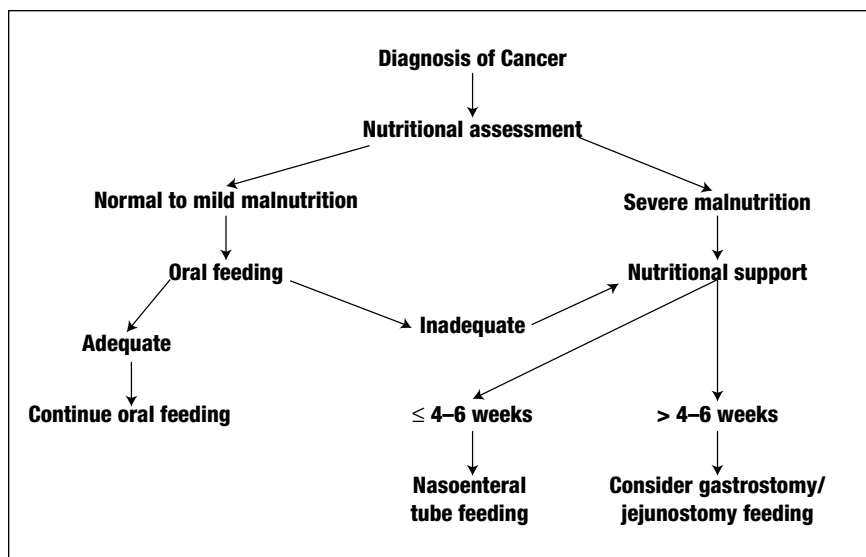


Figure. Indications for enteral feeding. Adapted with permission from Gentilini et al.<sup>74</sup>

nutritional absorbency in patients with limited absorptive capacity. This is beneficial in those with extensive small bowel resections and in patients with mucosal injury from chemotherapy or radiation therapy.

Enteral feedings may be delivered by a variety of techniques. Nutrients should be administered distal to the ligament of Treitz so that the complications of aspiration pneumonia and gastric ileus can be avoided.

For short-term feeding, a pliable silicone rubber nasoduodenal tube may be used. If enteral support is expected to last longer than 4 weeks, the preferred method is a gastrostomy or jejunostomy (Figure), which can be placed either surgically or endoscopically. Significant advances in endoscopic techniques have made enteral feeding a relatively safe, economical, and common therapy in cancer patients.<sup>53</sup> More than 80 different enteral feeding formulas are commercially available, many of which provide com-

plete nutrition.<sup>54</sup>

Carbohydrates usually represent the major source of calories. Casein or whey provides the protein component. Fat is supplied as triglycerides or vegetable oils. For patients with renal, cardiac, or hepatic insufficiency, several formulas are adjusted appropriately for volume and electrolyte balance.

Several randomized clinical trials have shown that clinical outcome is improved in severely malnourished cancer patients who receive enteral nutrition in the perioperative period.<sup>55,56</sup> Others have suggested that the addition of specific nutrients to enteral feeding solutions may provide immunologic and metabolic benefits. Among the nutrients suggested are the fish oil  $\omega$ -3 fatty acid, glutamine, arginine, and RNA.<sup>57-60</sup>

Two recent studies addressed postoperative, specially enriched enteral nutritional support.<sup>59,60</sup> Daly et al<sup>59</sup> conducted a prospective trial of 60 adult patients with upper gastrointestinal tract malig-

nancies. Patients were randomized to receive enteral diets supplemented with arginine, RNA, and  $\omega$ -3 fatty acids or standard enteral feedings. Significant reductions in postoperative infections and in length of hospital stay were reported in the patients fed the supplemented diet.

A more recent prospective, randomized study by Gianotti et al<sup>60</sup> reported similar improvements in infections and decreased hospital stay in a subgroup of surgical oncology patients who received enteral feedings enriched with arginine,  $\omega$ -3 fatty acids, and RNA. Klimberg et al<sup>61</sup> showed enhanced response to methotrexate therapy with increased tumor volume loss and decreased host toxicity in tumor-bearing rats fed a glutamine-supplemented diet.

by Dudrick and colleagues<sup>62</sup> many questions still exist regarding the application of TPN in cancer patients.

Improvements in nutritional indices such as body weight and total body fat content have been shown, as has the ability to replenish specific minerals, trace elements, and vitamins in patients undergoing chemotherapy who receive TPN.<sup>46,63</sup> Although TPN appears to improve these variables, it does not stop the loss of body nitrogen or abrogate the increase in protein turnover or lipolysis.<sup>45</sup> Discontinuation of TPN drastically reduces gains in body weight, fat, and electrolytes.<sup>46</sup> It may cause a decrease in appetite and oral intake after cessation of TPN.<sup>27</sup>

Therefore, the relevant issue is whether TPN favorably affects the com-

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***Malnourished cancer patients commonly  
have increased whole body protein turnover and  
subsequent body nitrogen loss.***

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Larger prospective, randomized trials are necessary to assess this specific issue.

#### TOTAL PARENTERAL NUTRITION

In patients unsuitable for oral or enteral nutritional support, TPN remains an important option. It delivers nutrients directly into the circulation, avoiding the problems with poor oral intake and gastrointestinal dysfunction seen in patients with cancer-induced cachexia. The hyperosmolar TPN solutions require catheterization of the central venous system to reduce the incidence of phlebitis and venous thrombosis. Common catheterization sites include the subclavian and internal jugular veins. Inherent complications, which are infrequent, include pneumothorax, venous thrombosis, arterial trauma, and sepsis.

Twenty years after the initial work

by Dudrick and colleagues<sup>62</sup> many questions still exist regarding the application of TPN in cancer patients.

Several randomized studies using different chemotherapeutic protocols to treat various types of cancer have addressed this issue.<sup>58,64-66</sup> The results of these studies showed that TPN did not confer an advantage in decreasing chemotherapy-induced morbidity or tumor responsiveness. In fact, an increased rate of infection was noted in the TPN-treated patients.<sup>67,68</sup> The interpretation of these studies is difficult because most patients were not necessarily malnourished and several had adequate oral intake.

In a randomized study by Weisdorf et al<sup>69</sup> recipients of bone marrow transplants were given either TPN or intravenous fluids 6 days before cytoreductive therapy. The TPN-treated group showed



a significant decrease in malignant relapse and long-term mortality compared with the control group.

Several randomized studies have examined whether TPN decreases postoperative complications and mortality in cancer patients undergoing surgical procedures.<sup>2,3,70-72</sup>

The results are conflicting for several reasons, including studies with small numbers of patients, inadequate periods of TPN, and use of populations of both malnourished and well-nourished cancer patients.

In an earlier nonrandomized study, Daly et al<sup>72</sup> studied the impact of TPN on 244 patients treated surgically for esophageal malignancies. They showed a significant decrease in postoperative

ized study by Brennan et al<sup>3</sup> evaluated the use of postoperative TPN versus intravenous fluids and electrolytes in 117 patients undergoing resection for pancreatic cancer. They showed no benefit from routine postoperative TPN. A higher incidence of infectious complications occurred in patients who received TPN compared with controls.

The recent study by Fan et al<sup>70</sup> on the use of routine perioperative TPN in patients undergoing hepatectomy for hepatocellular carcinoma reported a clear benefit from TPN. In this study, 144 patients were randomized to 1 day of preoperative TPN followed by 7 days of postoperative TPN versus intravenous fluids with electrolytes. Patients in the TPN group had less postoperative mor-

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***Enteral and parenteral feeding are safe, effective methods of delivering nutrients to cancer patients unable to ingest adequate amounts orally.***

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complications in patients who received TPN compared with those who did not. This study also found that patients who received 5 days of preoperative TPN had significantly fewer complications than did patients who received TPN only in the postoperative period.

In a randomized study in which patients underwent operations for a variety of gastrointestinal malignancies, Muller et al<sup>71</sup> showed a significant reduction in major postoperative complications and mortality in patients receiving 10 days of preoperative TPN. Postoperative morbidity and mortality appear to be significantly reduced in cancer patients with severe malnutrition who receive TPN as preoperative nutritional support.

Postoperative TPN support in cancer patients has been addressed in several recent studies. A prospective, random-

ized study by Brennan et al<sup>3</sup> evaluated the use of postoperative TPN versus intravenous fluids and electrolytes in 117 patients undergoing resection for pancreatic cancer. They showed no benefit from routine postoperative TPN. A higher incidence of infectious complications occurred in patients who received TPN compared with controls.

Indiscriminate use of TPN in cancer patients offers little to no benefit. In several instances, TPN may do more harm, increasing morbidity. TPN does not appear to benefit well-nourished or mildly malnourished patients undergoing chemotherapy, radiotherapy, or surgery, particularly those with adequate oral intake. TPN as an adjuvant anticancer therapy must be used in very limited fashion and in specific situations as identified in several randomized studies.<sup>69-71</sup>

When appropriately selected, certain cancer patients receiving TPN have shown significant decreases in morbidity

and mortality. These include patients with severe malnutrition receiving perioperative TPN and bone marrow transplant recipients.

The American Society of Parenteral and Enteral Nutrition has recommended TPN supplementation in patients expected to have inadequate oral or enteral nutrition intake for more than 10 to 14 days.<sup>73</sup>

## Conclusions

Malnutrition is a common problem in cancer patients that adversely affects quality of life and survival. It results from a multifactorial process involving host and tumor interactions. These interactions alter nutritional intake and cause massive metabolic disturbances. Many of these metabolic aberrations are wasteful and energy-consuming in an already energy-deprived host. Anticancer therapies may compound the malnutrition by increasing gastrointestinal side effects, leading to decreases in nutritional intake.

The treatment goal for malnourished cancer patients is provision of nutritional support to reverse or decrease the cachectic state. Optimal nutrition improves therapeutic modalities and the clinical course and outcome in cancer patients.

Oral nutrition should be used when-

ever possible. Special oral diets are targeted for specific anatomic and physiologic considerations. The various nutritional supplements commercially available can be an invaluable source of nutrition in cancer patients, and use of such supplements can avoid more complex and costly methods. Counseling by a dietitian is often required.

Enteral and parenteral feeding are safe and effective methods to deliver nutrients to cancer patients who are unable to ingest adequate amounts orally. Severely malnourished cancer patients and those in whom gastrointestinal toxicity is expected to hinder substantially oral intake for more than 1 week have benefited from enteral or parenteral feeding.

The indiscriminate use of specialized nutritional support is not indicated in well-nourished cancer patients or in mildly malnourished patients undergoing surgery, chemotherapy, or radiotherapy in whom adequate oral intake is anticipated.

The role of enteral feeding as an adjuvant to anticancer therapy has not been evaluated fully. In the future, nutritional support in cancer patients probably will be directed toward attenuating the host response to malignancy by specific immunomodulating compounds that target cytokine production and its downstream effects. CA

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