**Nutrition Management in the ICU***

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Nutrition support plays an important role in the management of nutritional deficiencies in properly selected critically ill patients. A full nutritional assessment allows the calculation of appropriate feeding goals. The route of feeding, enteral or parenteral, is determined by the presence or absence of a functioning intestine and hemodynamic status of the patient. The specific roles of carbohydrates, fats, and protein need to be considered in order to prevent overfeeding and other complications. The efficacy of certain disease-specific enteral formulas has been demonstrated in clinical trials, however, careful cost-benefit analyses are required.

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Malnutrition is an alteration of body composition in which deficiencies of macronutrients and micronutrients result in reduced body cell mass, organ dysfunction, and abnormal serum chemistry values. Nutrition support plays a vital role in the prevention and treatment of nutritional deficiencies in appropriately selected, at-risk, critically ill patients in the ICU. Patients most likely to benefit from nutritional support are those with baseline malnutrition in whom a protracted period of starvation would otherwise occur. In well-nourished persons with short (<1 week) anticipated duration of nil per os status, it is very difficult to demonstrate improvement in outcome with nutrition support.

Assessment of malnutrition in critically ill patients begins with obtaining any history of recent, involuntary weight loss (exceeding 5% within 1 month or 10% over 6 months), although fluid overload usually prevents the accurate determination of dry weight in the ICU. Physical examination should focus on signs of protein-calorie deficiency (such as temporal wasting), signs of specific micronutrient deficiency (such as anemia, glossitis, or rash), hydration state, and edema. Dry weight and height are used to calculate the ideal body weight, the percentage of ideal body weight, and the body mass index (BMI). Ideal weight can be calculated as follows:

Men = 106 lb for 5 feet in height plus 6 lb for each additional inch.

Women = 100 lb for 5 feet in height plus 5 lb for each additional inch.

If an individual’s frame is small, the estimated ideal body weight may be reduced by 10%; conversely, for a large frame, 10% may be added. BMI is defined as the weight in kilograms divided by the square of the height in meters. Normal BMI ranges from 19 to 25. Survival at a BMI below 14 is very unusual.

Anthropometric data (skinfold thickness and arm muscle circumference), as well as creatinine height index (the urinary creatinine level according to height), while useful in ambulatory patients, are significantly less accurate measures of malnutrition in the critically ill patient, particularly in those who have fluid overload or renal dysfunction.

Albumin is the most common laboratory measurement of visceral protein status. Contrary to popular thinking, hypoalbuminemia is rarely present in cases of isolated calorie malnutrition. Hypoalbuminemia is more commonly a marker of the systemic inflammatory response and, as such, has prognostic importance. It has been associated with increased morbidity and mortality among hospitalized patients. The daily hepatic synthesis rate for albumin is 120 to 170 mg/kg of body weight. Albumin is distributed between the intravascular and extravascular spaces. During injury, the liver increases production of acute-phase proteins and reduces albumin synthesis. The decrease in albumin coupled with extravasation and enhanced catabolism (both mediated by cytokines) culminates in hypoalbuminemia. Therefore,

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serum albumin concentration is a poor index of nutritional status but rather serves as a marker of injury and metabolic stress during injury response.7

The goals of nutrition support in ICU patients as summarized by a consensus statement from the American College of Chest Physicians are as follows8:

1. To provide nutrition support consistent with the patient’s medical condition and the available route of nutrient administration.
2. To prevent and treat macronutrient and micronutrient deficiencies.
3. To provide doses of nutrients compatible with the existing metabolism.
4. To avoid complications related to the technique of dietary delivery.
5. To improve patient outcomes such as those affecting resource utilization, medical morbidities and mortalities, and subsequent patient performance.

**Total Parenteral Nutrition in ICU Patients**

In general, the enteral route is preferred over the parenteral route, as the former is more physiologic, is less likely to be associated with biliary stasis and hyperglycemia, and is significantly less expensive.9 Many studies have purported to show that total parenteral nutrition (TPN) is associated with higher infection rates than is enteral feeding, although this has not been confirmed when equivalent calories have been administered by each route and when overfeeding with TPN is avoided.10 Contraindications to enteral feeding include diffuse peritonitis, intestinal obstruction, intractable vomiting, paralytic ileus, and severe diarrhea. Hypotension with hemodynamic instability is associated with reduced intestinal blood flow, and low tolerance to enteral feeding is the rule.

TPN plays an important role in patients in whom the gut cannot be used. Administration of 25 kcal/kg of usual body weight is adequate for most patients with normal BMI.8 In most patients this goal approximates the one calculated from the Harris Benedict equation. With BMI < 19, overfeeding may result in a refeeding syndrome characterized by electrolyte abnormalities (hypophosphatemia, hypokalemia, and hypomagnesemia), volume overload, and congestive heart failure.11 Refeeding syndrome is less likely if TPN is introduced gradually. Start with no more than 100 to 150 g dextrose and low concentrations of sodium chloride, and implement stringent monitoring of electrolytes (daily for the first 2 to 3 days) and blood sugars (every 6 h until persistently euglycemic with dextrose at goal).

The amount of CO₂ produced when fuel is burned may be clinically important in patients for whom ventilator weaning is problematic. Indirect calorimetric techniques are used to determine the respiratory quotient [RQ] (the ratio of CO₂ produced to O₂ consumed), as well as the resting energy expenditure.12 Overfeeding carbohydrates results in an RQ close to 1.0, whereas consumption of fuels that are predominantly fat-based yields RQs closer to 0.7 (mixed fuels, 0.8 to 0.9).13 Composition of the TPN can be altered to prevent overfeeding in general, and overfeeding carbohydrates in particular, thus, calorimetry may be an important tool in certain patients.

The protein (amino acid) goal in TPN ranges from 1.2 to 1.5 g/kg/d and should be adjusted with periodic monitoring to promote nitrogen retention and to support protein synthesis.8 However, in critically ill persons, it is usually impossible to effect a positive nitrogen balance, as the cytokine and catabolic hormone cascade prevent anabolism. The administration of protein in higher quantities is unlikely to promote lean mass accrual. Azotemia can be aggravated by a high protein load, and thus, BUN values > 100 mg/dL might be an indication to decrease nitrogen intake, although this is not well validated in the acute illness setting. A more usual issue in feeding the patient with acute renal failure is that volume restrictions limit the quantity of feeding. In persons with chronic renal insufficiency, 0.8 g/kg/d of protein is sufficient. Another possible indication for limiting protein consumption in TPN occurs in persons in whom hepatic encephalopathy is a major clinical problem. Reducing the amino acid load or using a high quantity of branched-chain amino acids (BCAAs) have been shown to improve mental status.14

The lipid component of TPN consists of omega-6-polyunsaturated fatty acids that may be administered separately from the dextrose/protein or as part of a three-in-one solution. Theoretical concerns with overfeeding of lipids include injury to the reticuloendothelial system, which might lead to immunosuppression and can negate the beneficial effect of nutrition support.15 However, limiting fat calories to 30% of total calories is unlikely to lead to this complication, especially when the fat is infused slowly as with the three-in-one solution. Triglyceride levels > 400 mg/dL are a relative contraindication to adding lipids.

Carbohydrates should constitute the remainder of the total calories at between 3 and 5 g/kg/d.8 However, the specific amount should be adjusted appropriately to maintain a blood glucose level < 220 mg/dL. Many patients require coinfusion of regular insulin (usually as a component of the TPN) with supplemental subcutaneous administration of slid-
ing-scale regular insulin if necessary. Postoperative hyperglycemia (blood glucose level > 220 mg/dL) has been shown to increase the risk of nosocomial infection to a degree that nullifies the benefits of nutritional repletion. Severe stress (eg, postoperative patients) is accompanied by rising plasma levels of the counterregulatory hormones glucagon, epinephrine, and cortisol, and thus, postoperative patients are most at risk from TPN-induced hyperglycemia.

Fluid restriction is often vital in cardiac, pulmonary, postoperative, and renal patients in the ICU. For such patients, TPN can be restricted to 1 L. Maximally concentrating nutrients allows the provision of 1,000 kcal and 70 g of protein per liter, which is often a substantial percentage of the weight-based feeding goal. Vitamins and trace elements are usually administered as components of the TPN. In addition, a number of medications, such as histamine-2 receptor antagonists and metoclopramide, can be mixed in with the TPN solution.

**Enteral Nutrition Support in ICU Patients**

Intragastric feeding requires adequate gastric motility and emptying; a residual of > 150 mL is a relative contraindication to gastric feeding as the risk of aspiration is high. Nutrition support with TPN or small-bowel feeding is then appropriate. Postpyloric enteral feeding is often effective even in the presence of gastric atony and/or colonic ileus. For effective small-bowel feeding, simultaneous nasogastric decompression may be required. The presence of bowel sounds and the passage of flatus or stool are not necessary to initiate postpyloric enteral feeding. Secretory diarrhea may occur and is not an absolute indication to discontinue enteral feedings unless output exceeds 1,000 mL/d. Output in this range requires an evaluation.

Enteral feeding is usually started with an elemental formula with reduced fat content at low rates until tolerance is determined. Rates may be advanced toward the goal every 8 h, as tolerated, as long as the gastric residual is low, and abdominal distension and pain are absent. Multiple vitamins need to be ordered separately. Caloric requirements are calculated as for TPN. The main difference is that many disease-specific enteral formulas exist.

**Disease-Specific Formulations**

**Immune-enhancing**

One recent advance in enteral nutrition has been the use of so-called “immune-enhancing” formulas that include arginine, glutamine, nucleotides, and/or omega-3 fatty acids (fish oil) in septic and catabolic patients. A multicenter prospective randomized clinical trial with administration of such a formula (Impact; Novartis Pharmaceuticals; Basel, Switzerland) for 7 to 10 days showed reduced rates of infection and wound complications and shorter hospital stays for critically ill patients. In another multicenter trial, trauma patients receiving such a formula experienced significantly fewer intra-abdominal abscesses and less multiple organ failure.

**Pulmonary**

Pulmonary formulas are designed to be high in fat (50%) and low in carbohydrates to reduce CO₂ production, thereby reducing ventilatory demand. In preclinical studies, a tailored pulmonary formula reduced pulmonary neutrophil accumulation and inflammatory cytokines and improved cardiopulmonary hemodynamics and gas exchange. This disease-specific pulmonary formulation contains eicosapentaenoic acid and γ-linolenic acid (which modify production of proinflammatory cytokines) and antioxidants (vitamin E, vitamin C, and beta-carotene), and is a calorically dense formula, suitable in particular for fluid-restricted patients with ARDS.

**Hepatic**

Hepatic enteral formulas contain relative large amounts of the BCAAs valine, leucine, and isoleucine, with low quantities of aromatic amino acids. These products are tailored for patients with hepatic encephalopathy. The rationale is that infusion of BCAA corrects the imbalance between aromatic amino acids and BCAAs in plasma and the CNS that might contribute to the mental disturbances that are common. The use of BCAA-enriched formulas for short periods may be beneficial because they improve nitrogen balance and lessen encephalopathy, but their use for longer periods becomes expensive and may limit protein synthesis, resulting in an inadequate nitrogen balance.

**Renal**

Specific renal formulas are usually low in protein or contain variable proportions of BCAA. The solutions are usually calorically dense and contain up to 2 kcal/mL. To achieve this density, some formulas may contain significant amounts of fat, the ingestion of which may result in bloating and delayed gastric emptying. Potassium, phosphorus, and magnesium are present in substantially lower amounts than is the case for typical enteral feeds. Renal patients are also at increased risk of certain micronutrient toxicities.
However, it is important to feed patients adequately to avoid body cell mass catabolism and malnutrition. For critically ill patients, it is best to use dialysis to clear nitrogen and fluid and to feed them an adequate protein diet than to underfeed protein.8

**Novel Pharmaconutrients on the Horizon**

Glutamine, BCAA, peptides, growth hormone, arginine, omega-3-polynsaturated fatty acids (fish oils), and antioxidants (selenium, vitamins C, vitamin E, and beta-carotene) are being evaluated for their individual effects on specific metabolic functions. As with other nontraditional formulations, cost is usually higher, and thus, benefits need to be demonstrated in prospective, randomized, clinical trials before widespread recommendations can be made.

**References**