Evidence-based Nutritional Support in the Intensive Care Unit

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Introduction

Over 2 millennia ago "Hippocrates: Father of Medicine" (460? to 377 BC) once said "If we could give every individual the right amount of nourishment and exercise, not too little and not too much, we would have found the safest way to health." That is true even now with various nutrition surveys in hospitals continue to suggest that upwards of 40% to 50% of patients, particularly those in the intensive care unit (ICU), have a moderate to severe degree of malnutrition,[1] with its significant negative impact on clinical outcomes.

Malnutrition is a change of body composition in which deficiencies of macronutrients and micronutrients result in progressive decline in body cell mass, various organs dysfunction, and abnormal serum chemistry values. Nutrition support plays a vital role in the prevention and treatment of nutritional deficiencies in at-risk, critically ill patients.[2]

Many critically ill patients are hospitalized with various comorbidities such as cardiovascular disease, asthma, and cancer, where they require nutritional supplement while recuperating from their medical

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or surgical injuries. Although there is no real agreement among the various healthcare professionals about the best method to provide such nutrition support the American College of Chest Physicians consensus statement established several attainable goals of nutrition support for the ICU patients (Table 1).[3]

This review will discuss unique metabolic changes of the critically ill patients and the challenges in estimating their daily caloric needs then providing them with adequate nutrition support while in the ICU. In addition, it will provide some of the scientific data regarding nutrition support of critically ill obese patients.

■ Metabolic Response to Critical Illness

Critically ill patients are characterized by wide variations in their carbohydrate, lipid, and amino acid (protein) metabolism.[4] Such variations can lead to increase in their energy requirement with accelerated protein catabolism and ultimately alterations of their immune and gastrointestinal systems.

In the normal weight person, the metabolic response to injury causes an increase in protein and energy requirements. As a result, endogenous substrates serve as fuel sources and as precursors for protein synthesis. This response is mediated by counter-regulatory hormones (CRHs) such as epinephrine, glucagon, cortisol, and growth hormone, which regulate the flow of endogenous substrates between the various organs and tissues. In addition, cytokines such as tumor necrosis factor-α and interleukin-1β may play an important role in this systemic response inducing hyperglycemia (Fig. 1).[5] Such stress-induced hyperglycemia has many deleterious effects including increasing infectious threat, fueling proinflammatory effect, and slowing wound healing. However, in nondiabetic patients, either after elective surgical procedures or after trauma, this "stress-induced" hyperglycemia is normally transient in nature lasting about 24 hours postinjury, and reflecting the plasma concentrations of CRHs.[5,6]

Table 1. *The American College of Chest Physicians, Goals of Nutrition Support in Intensive Care Unit Patients*

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

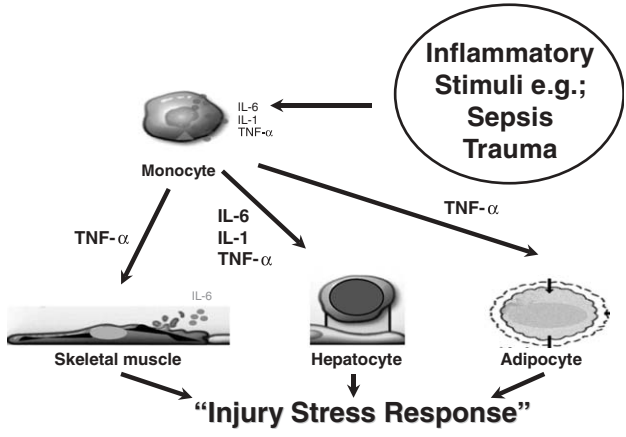

Figure 1. *A variety of conditions that needs to be taken care in an intensive care unit lead to cytokines release.*

In addition, endogenous insulin acts to counteract the deleterious effects of CRHs-induced hyperglycemia.

Morbidly obese subjects {≥130% of their ideal body weight (IBW) [Body Mass Index (BMI) ≥30 kg/m^2]} represent a unique subgroup of the critically ill patients. Accordingly, few caveats must be kept in mind while managing critically ill obese patients. First, fasting obese, nonstressed subjects have higher levels of plasma components including substrates, amino acids, and hormones. Second, the effect of stress on this hormonal milieu is unknown; however, obese individuals seem to be at greater risk of postoperative complications such as pulmonary complications, bacteremia, and clinical sepsis after acute thermal injury than lean individuals.[7] Third, catabolic response to injury of the acutely injured obese patients is similar to that of normal weight patients, except that they have less lean tissue stores and excess body fat. As a result, obese individuals are likely to develop net protein oxidation with higher daily muscle mass degradation followed by the body utilization of endogenous lipids as the main source for energy.[7]

■ Determination of Nutrition Requirements

Nutrition Assessment

Assessment of nutrition status in critically ill patients begins with inquiring about any history of recent involuntary weight loss (exceeding 5% within 1 mo or 10% over 6 mo) and recording of the admission weight, as fluid overload after admission to the ICU usually prevent

accurate determination of the correct weight.[8] In addition, history should include assessment of different risk factors which impair digestion, utilization, or excretion of nutrients such as history of gastric or bowel bypass surgery. Physical examination should focus on signs of nutritional deficiencies especially protein-calorie deficiency (such as temporal wasting), signs of specific micronutrient deficiency (such as anemia, glossitis, or rash), hydration state, and edema.

Whenever possible, admission weight and height should be used to calculate the IBW, the percentage of IBW, and the BMI. For men ideal weight is calculated by using 106 lb for the first 5 feet in height then adding 6 lb for each additional inch. Whereas for women it is 100 lb for the first 5 feet in height then additional 5 lb for each additional inch. Additional adjustment are made by reducing or increasing the estimated IBW by 10% for small frame individual or adding 10% for a large frame individual, respectively. Then BMI is calculated by dividing weight in kilograms by the square of the height in meters. Normal BMI ranges from 19 to 25, however, though a BMI <14 at time of ICU admission have a poor survival prospective.

Anthropometric data (skinfold thickness and triceps-midarm circumference), and creatinine height index (the urinary creatinine level according to height), although useful in ambulatory patients, are not as accurate measures of malnutrition in the critically ill patients.[9] The latter is mostly secondary to the high incidence of fluid overload and renal dysfunction in the critically ill patients.

Serum albumin level and several other transport proteins, Table 2, are commonly measured as surrogates of visceral protein status. The daily hepatic synthesis rate for albumin is between 120 and 170 mg/kg of body weight with the albumin distributed between the intravascular and extravascular spaces.[10]

However, serum levels of albumin and other transport proteins are influenced by many factors such as synthesis and degradation rates and vascular losses into the surrounding interstitium, in addition to losses through the gut or kidney. As a result, their levels drop by inflammation, trauma, or sepsis where high levels of interleukin-6 stimulate acute phase protein production as it inhibits transport protein production.[11]

Table 2. *Transport Proteins*

Protein/Normal Value	Half Life ($T_{1/2}$)	Mild	Moderate	Severe
Albumin 3.5-5.0 g/dL	21 d	2.8-3.5	2.1-2.7	<2.1
Prealbumin 10-40 mg/dL	2-3 d	10-15	5-10	<5
Transferrin 200-400 mg/dL	8 d	150-200	100-150	<100
Retinol-binding protein 2.7-7.6 mg/dL	10 h	4-6	2-4	<2

Hence, contrary to popular belief, hypoalbuminemia is rarely present in cases of isolated calorie malnutrition.[12] Rather, hypoalbuminemia is a marker of the systemic inflammatory response and is associated with increase morbidity and mortality among hospitalized patients.[13] Therefore, serum albumin concentration can be used as a nutrition screening tool at the time of ICU admission. However, it is a poor indicator of critically ill patients' nutritional status as it only serves as a marker of injury and metabolic response to stress.[14]

Another marker for severe malnutrition is the delayed cutaneous hypersensitivity as it represents a delayed hypersensitivity reaction to 1 of 3 recall antigens (0, nonreactive; 1, <5-mm induration; and 2, >5-mm induration). However, it is also influenced by stress injury, hepatic or renal failure, and use of steroids. In addition, the delayed cutaneous hypersensitivity is a component of the Prognostic Nutritional Index (PNI). The PNI is a predictive tool that uses a combination of various indicators, Table 3, which are used to assess the risk for subsequent nutrition-related infection complications. The PNI correlates with poor outcome through an equation with a higher risk for infection as the PNI score increase.[15]

Finally, the Prognostic Inflammatory Nutrition Index (PINI)[4] seems to correlate with recovery from injury after acute illness. It reflects the elevations in acute phase proteins that occur with simultaneous reductions in transport proteins, and is calculated by the following equation:

$$\text{PINI} = (\text{CRP})(\text{AAG})/(\text{PA})(\text{ALB})$$

where CRP is C-reactive protein, AAG is α 1-acid-glycoprotein, PA is prealbumin, and ALB is albumin. The PINI decreased significantly after enteral nutritional support of critically ill trauma patients, as it influenced primarily by a decrease in CRP concentration.[16]

Determination of Caloric Requirements

Basic metabolic needs [basal energy expenditure (BEE)] is difficult to predict in all critically ill patients as it is influenced by multiple changing factors related to the acute illness, level of stress, and treatments. Determination of BEE is most commonly accomplished by using the Harris-Benedict (H-B) equation, although few other formulas are used but to a lesser extent.[17] The H-B equation was driven from a population-based analysis in 1919. It is the most widely used equation to

Table 3. *Prognostic Nutritional Index*

PNI (% risk) = 158% – 16.6 (Alb) – 0.78 (TSF) – 0.20 (TFN) – 5.8 (DH)

Alb indicates serum albumin; DH, skin test reactivity; PNI, Prognostic Nutritional Index; TFN, serum transferrin; TSF, triceps skin fold.

estimate the BEE from body weight/height, sex, and age. BEE for women = 65.5+ 9.6 (weight in kg)+ 1.9 (height in cm)− 4.7 (age in years). Whereas BEE for men = 66+ 13.7 (weight in kg)+ 5.0 (height in cm)− 6.76 (age in years). In general, most of the available equations include weight as a variable for estimating the energy expenditure.

However, there is a continuous debate about whether to use the actual body weight (ABW) or the IBW weight in the critically ill patients especially with accompanied obesity?[18] An earlier study of obese surgical and nonsurgical patients with a mean age of 52 years used a regression analysis model and demonstrated that age, sex, ventilatory status, and ABW, rather than IBW, were significantly correlated with BEE. Accordingly, they recommended that ABW should be used in various predictive equations of energy expenditure in obese patients.

But as the "gold standard" most available data support the use of indirect calorimetry as the only validated method to measure the energy expenditures of the nonobese and obese (>30% above IBW) hospitalized patients.[7] Through the indirect calorimetric techniques the respiratory quotient (RQ) (the ratio of CO_2 produced to O_2 consumed), and the resting energy expenditure (REE) are determined by the following formula: $REE = 3.9 \times VO_2 + 1.1 \times CO_2 - 2.8 \times UUN$ (urine urea nitrogen).

As a general principle, overfeeding carbohydrates results in an RQ close to 1.0, whereas consumption of fuels that are primely lipid-based yields RQs closer to 0.7 (mixed fuels, 0.8 to 0.9). Type of eternal nutrition (EN), see below, and composition of the parenteral nutrition (PN) can be modified to prevent overfeeding in general, and overfeeding carbohydrates in particular, hence, indirect calorimetry can be an important management tool in the critically ill.

■ Nutrition Regimens for the Critically Ill Patients

Enteral Nutrition Support

It is conceivably agreeable that EN is preferred over PN, as the former is more physiologic, less likely to be associated with biliary stasis or hyperglycemia, and certainly less expensive.[19] In addition, many studies have demonstrated that PN is associated with higher infection rates than is EN.[20] In the Veterans Administration study,[21] 395 patients requiring laparotomy or noncardiac thoracotomy were randomized to 7 to 15 days of preoperative nutrition or no specialized nutrition support and evaluated for postoperative complications. Incidences of major noninfectious complications, such as wound or anastomotic dehiscence, were similar between unfed and parenterally fed patients with mild to moderate malnutrition, but parenterally fed patients sustained significantly additional infectious complications. However, patients with

preexisting severe malnutrition sustained a significant reduction in the same major noninfectious complications with a trend toward a reduction in infectious complications. Thus, PN increased the rate of complications in patients at low risk of nutrition-related complications but improved outcome in a select high-risk group. Similar results were demonstrated by Brennan et al[22] who randomized 117 mildly malnourished patients to either postoperative PN or to intravenous fluids alone. In this relatively low nutritionally at-risk patient population, infectious complications were significantly greater in parenterally fed patients owing to a higher rate of intra-abdominal abscess formation. However, interpretation of the above studies warrants an examination of both the clinical outcome and the patient population entered into each clinical trial.

Timing of Nutrition Support

Overall early nutrition support is referred to feedings initiated within the first 24 to 72 hours after injury or acute illness requiring intensive care. Several studies have examined practices related to EN in the ICU and found that inadequate nutrition support is common.[23,24] Delay in providing adequate nutrition support 72 hours after an acute illness may lead to many unwanted consequences. Underfeeding may reduce the integrity of the gastrointestinal tract, and maintenance of immunity and the stress response to injury. McClave et al[25] found that 66% of EN withholding was avoidable through protocol adjustments. These adjustments include continuing EN until 4 hours before surgical or endoscopic procedures without increasing risk for aspiration or impeding of the view at the time of the procedure.

A consensus statement published by the American College of Chest Physicians supports the initiation of EN as soon as possible after resuscitation to promote better ICU outcomes.[3] Table 4[26–31] provides a summary of selected studies related to early EN with the overall evidences suggests that early EN is beneficial to critically ill patients. However, further studies are warranted to determine if only certain types of critically ill patients benefit from early nutrition support and if outcomes differ between early gastric versus early intestinal enteral feeding.

Intragastric feeding requires adequate gastric motility and emptying. Review of the medical literature[32] reveals that EN is frequently withheld from eligible ICU patients for subjective reasons or clinical criteria, which have not been consistently proved to cause complications. High gastric residual volumes (RVs), plan surgical procedures or diagnostic tests, bedside nursing interventions, gastrointestinal intolerance, and mechanical delivery problems are the most commonly cited reasons for withholding feeding. The relationship of gastric RV to risk of pulmonary aspiration of gastric contents is perhaps the most widespread

Table 4. *Outcome of Early EN Feedings*[26–31]

Study	Year	Protocol	Population of Patients	Results
Marik and Zaloga[26]	2001	Meta-analysis of 15 randomized early vs. late EN	Patients hospitalized after surgery, trauma, head injuries, and burns (N = 753)	Starting EN early decrease infection rate and length of hospital stay
Lewis et al[27]	2001	Meta-analysis of 11 randomized trials comparing early enteral feeding with no oral intake	Elective gastrointestinal surgical patients (N = 837)	Group in which enteral feeding was started earlier had lower infection rate and shorter hospital stay
Minard et al[28]	1998	Randomized trial of early vs. late immune-enhancing formula	Severe closed-head injury patients (N = 30)	No difference between the groups for length of stay or infection
Ibrahim et al[29]	2002	Randomized trial of early vs. late standard polymeric EN	Mechanically ventilated medical ICU patients (N = 150)	Increase infections and longer stays in early EN group
Martin et al[30]	2004	Randomized trial of evidence-based algorithms to improve early ($\leq$24 h of admission) nutritional support in the ICU	All patients expected to stay in the ICU $\geq$48 h (N = 462)	Compared with control group, study patients received more days of EN ($P = 0.042$), had a significantly shorter mean stay in hospital ($P = 0.003$), and had trend toward reduced mortality ($P = 0.058$). The mean stay in the ICU did not differ ($P = 0.7$)
Nguyen et al[31]	2008	Randomized trial of early ($\sim$24 h of admission) vs. late (on day 4 of admission) standard polymeric EN	Mechanically ventilated medical ICU patients (N = 28)	Starting EN early decrease length of ICU stay and duration of mechanical ventilation

EN indicates enteral nutrition; ICU, intensive care unit.

yet unproven issue at hand. Using a computer model simulating typical formula delivery and normal gastric secretion rates, Lin and Van Citters[33] described gastric RVs at various gastric emptying rates. They

concluded that gastric RVs plateau at volumes seen in normal postprandial stomachs ($\leq$900 mL), unless gastric emptying is absent. Thus, arbitrary selection of a volume threshold may not make physiologic sense, and may only contribute to inadequate caloric and volume delivery. Nevertheless, most experts will agree that a gastric residual of $\leq$150 mL should be adequate for intragastric feeding as long as patient head can be maintained at $\geq$30 degree.[34] Postpyloric EN is an alternative in the presence of gastric atony and/or colonic ileus. However, the presence of bowel sounds and the passage of flatus are not necessary to initiate postpyloric enteral feeding.

Secretory diarrhea is common in critically ill patients especially after severe illness and after prolonged periods of fasting. However, it is not an absolute indication to discontinue EN unless the output $\geq$1000 mL/d. At output of this range, testing for infectious etiology may be indicated and the initiation of antidiarrhea medications is warranted if the tests are negative.

■ Nutritional Goals for Critically Ill Patients

Daily nutritional goals for critically ill patients are commonly set at 104.6 to 146.44 kJ/kg body weight for energy intake. Administration of 25 to 30 kcal/kg of usual body weight is adequate for most patients with normal BMI as calculated from the H-B equation.[3]

Total caloric needs are calculated with a goal for daily protein (amino acid) intake in the range of 1.0 to 2.0 g/kg/d. Protein intake should be adjusted with periodic monitoring of clinical response such as healing of wounds rather than routine laboratory revaluation to promote nitrogen retention and to support hepatic protein synthesis. In critically ill patients with chronic renal insufficiency, initiation of protein intake at 0.8 g/kg/d might be warranted while following the progression of the renal functions.

However, in critically ill patients, it is usually impossible to induce a positive nitrogen balance, as the ongoing cytokine and catabolic hormone cascade prevent adequate anabolism. Furthermore, the administration of protein in higher quantities is unlikely to promote lean mass accrual. In fact, azotemia can be aggravated by a high protein load, and thus, blood urea nitrogen values >100 mg/dL might be an indication to decrease nitrogen intake.

The lipid component of EN is limited to daily caloric intake at 30% of total calorie needs and can be reduced if serum triglycerides exceed 400 mg/dL. Finally, carbohydrates should constitute the remainder of the total daily calories at between 3 and 5 g/kg/d; however, the specific amount should be adjusted appropriately to maintain a blood glucose level less than 150 mg/dL.[35]

■ Disease-specific Formulations

Pulmonary

The potential for altered nutritional status in critically ill patients with either acute or chronic pulmonary disease is significant. Nutrition support is often indicated as a therapeutic or treatment modality. Several EN formulas had been suggested to help counteract the possible adverse respiratory effects associated with a standard formula with higher carbohydrates content to reduce patients' ventilatory demand.

However, the use of these specialized enteral formulas in individuals with pulmonary disease remain controversial. Data supporting the routine use of higher fat contents (50%) and low carbohydrates to reduce CO_2 production in hospitalized patients with pulmonary dysfunction are limited. It is suggested that this type of formula should be reserved for patients with marginal respiratory reserve (severely malnourished) or who fail to wean from mechanical ventilation despite prevention of overfeeding.[36]

Hepatic

Hepatic formulas offer increased amounts of branched chain amino acids (BCAA): valine, leucine, and isoleucine; and reduced amounts of aromatic amino acids: phenylalanine, tyrosine, and tryptophan, compared with standard products. The rationale is that infusion of BCAA promotes the reduction in uptake of aromatic amino acids at the blood-brain barrier, reducing the synthesis of false neurotransmitters and thereby ameliorating the neurologic symptoms that occur with hepatic encephalopathy (HE).[37] Although the use of BCAA-enriched formulas for short periods may be beneficial, as they may improve nitrogen balance and lessen encephalopathy, their use for longer periods becomes expensive and may limit protein synthesis, resulting in declining nitrogen balance.[38]

Renal

Generally, renal formulas are lower in protein, contain variable proportions of BCAA, calorically dense, and have lower levels of potassium, magnesium, and phosphorus when compared with standard formulas. Hence, persistent hyperkalemia, hypermanganesemia, or hyperphosphatemia is often the driving factor that leads most clinicians to switch from a standard formula to a renal product. However, there are no clinical trials comparing the efficacy of renal formulas against standard products.

Formula selection depends upon the patient's degree of renal function, the presence or absence of renal replacement therapy, and the

patient's overall nutrient requirements. The solutions are usually calorically dense and contains ≤ 2 kcal/mL. Patients undergoing renal replacement therapy have significantly increased protein requirements that may not be met only with the available renal formulas. Accordingly in patients undergoing renal replacement therapy, especially continuous venovenous hemodialysis, renal formulas are not always necessary. These patients typically do not require fluid restriction and have higher protein requirements of 1.5 to 2.0 g/kg/d.[39] In the absence of elevated levels of potassium, magnesium, and phosphorus, patients on dialysis should continue to receive a standard, high-protein formula.

■ Immune Enhancing

Early animal studies illustrated that diets supplemented with eicosapentaenoic (EPA) and gamma-linoleic acid (GLA) may have beneficial effects in models of acute lung injury (ALI) (Fig. 2.[40] Expanding on these early animal investigations, multiple human studies (Table 5)[41–47] were designed to examine the immunologic effects of feeding ICU patients' diets supplemented with EPA, DHA, GLA, and antioxidants. The aim was to investigate the changes on pulmonary inflammation, eicosanoid mediators of inflammation, endogenous antioxidants, and pulmonary function in patients with acute pulmonary inflammation. On one such study, patients with acute respiratory distress syndrome or ALI fed an enteral diet supplemented with an elemental formula (Oxepa) containing Ω-3 fatty acids, GLA, and antioxidants. The authors reported reduction in pulmonary neutrophil recruitment, improved oxygenation, significant decrease in the number of ventilator-dependent days, length of ICU stay, and decreased mortality compared with patients fed the control diet (Pulmocare).[43]

When taking into account the relatively low cost of enteral therapy with the EPA+ GLA diet combined with its safety and minimal side effect profile, EPA+ GLA-enriched enteral diet may prove to be an integral component of our management strategy for ALI and acute respiratory distress syndrome. The latter is in agreement with the various scientific organizations' clinical practice guidelines for nutrition support of critically ill adult patients on mechanical ventilation.[48]

■ PN

As mentioned earlier, various studies have reported that PN can be associated with higher infection rates than is EN in certain patients' population such as after major gastrointestinal surgery.[20]

Indications for PN may include diffuse peritonitis, intestinal obstruction, intractable vomiting, paralytic ileus, and severe diarrhea.

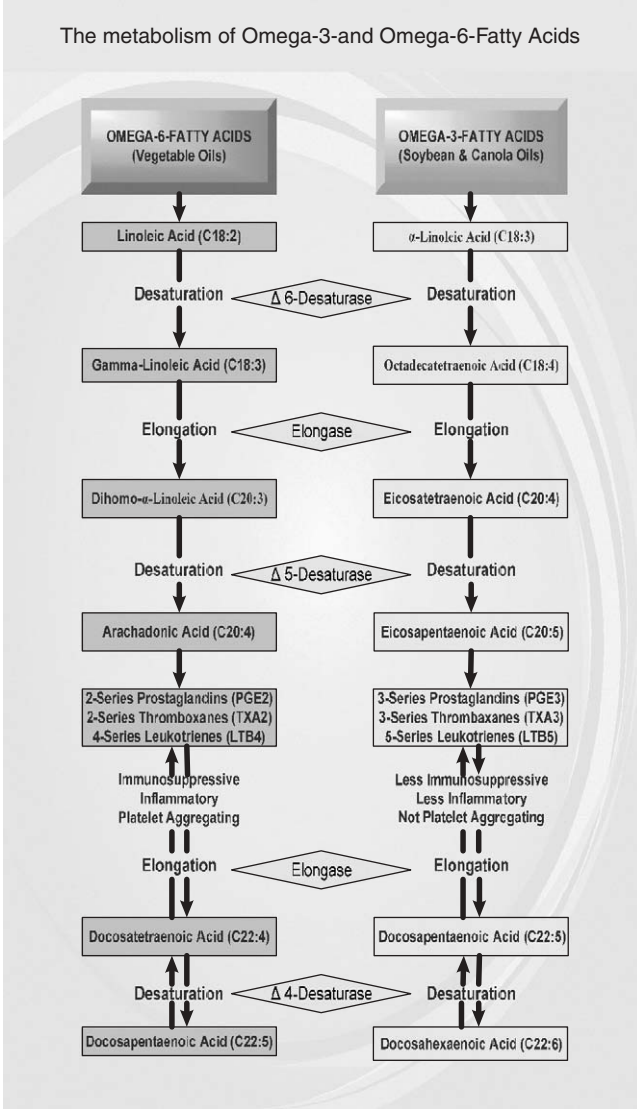

Figure 2. *The metabolism of Ω-3 and Ω-6-fatty acids.*

In addition, severe hypotension and hemodynamic instability may lead to reduced intestinal blood flow, with reduced tolerance to enteral feeding. However, the latter is not absolute contraindication for EN and those patients might benefit from continuous EN to maintain their gut barriers.[49]

As with EN administration of 25 to 30 kcal/kg of usual body weight is adequate for most critically ill patients with normal BMI. The calculation

Table 5. *Enteral Feeding With Eicosapentaenoic + Gamma-linoleic Acid Clinical Outcomes Summary*[41–47]

Study Population	Improved Oxygen Status	Reduced Intensive Care Unit LOS	Less Time on Vent	Fewer New Organ Failures	Reduced Mortality	Safety
Acute respiratory distress syndrome	X[42,44,45]	X[42,43]	X[43]	X[42,43]		X [42,43,45]
Acute lung injury	X[46]	X[46]	X[46]		X[46]	X[46]
Sepsis	X[41]	X[41]	X[41]	X[41]	X[41]	X[41]
Pediatric burn	X[44]					X[44]
Pediatric intensive care unit						X[47]

of protein (amino acid), lipid, and carbohydrates intake for PN follows the same pattern as with EN. However, in patients with acute renal failure volume restrictions may limit the quantity of feeding through PN. For such patients, volume of PN can be restricted to maximally concentrating nutrients allowing the provision of 1000 kcal and 70 g of protein per liter, which is often a substantial percentage of the weight-based feeding goal. In addition, in critically ill patients with HE, reducing the amino acid load or using a high quantity of BCAAs has been shown to improve mental status.[50]

The lipid component of PN consists primarily of Ω-6-polyunsaturated fatty acids that are administered daily either separately from the dextrose/protein or as part of a 3-in-1 solution. However, there have been concerns that daily administration of lipids may results in lipids' overfeeding with possible injury to the reticuloendothelial system that can lead to immunosuppression.[51] Yet, by limiting fat calories to 30% of total calories it is unlikely to lead to this complication, especially when the fat is infused slowly as with the 3-in-1 solution with weekly monitoring of serum triglyceride levels. A serum triglyceride level $\geq$400 mg/dL is a relative contraindication to adding lipids to PN.

As with EN, carbohydrates should constitute the remainder of the total calories of PN with close monitoring of blood glucose level and coinfusion of regular insulin in PN in addition to supplemental subcutaneous administration of sliding-scale regular insulin if necessary. Vitamins and trace elements provisions are usually administered as components of the PN at least 3 times per week.[52] In addition, a number of medications, such as histamine-2 receptor antagonists, can be added to the PN solution.

However, with BMI <19, PN overfeeding may result in a refeeding syndrome characterized by multiple electrolyte abnormalities especially hypophosphatemia, hypokalemia, and hypomagnesemia. Furthermore, PN may lead to volume overload secondary to poor hydrostatic gradients and the development of congestive heart failure.[53] Though, refeeding syndrome can be avoided by introducing PN gradually with 100 to 150 g of dextrose daily and low concentrations of sodium chloride. Then daily monitoring of electrolytes for the first 2 to 3 days and blood sugars every 4 to 6 hours until euglycemia is achieved while receiving dextrose at goal concentration.

■ Nutritional Care of the Obese ICU Patient

Although the benefits of enteral versus PN are well established for obese ICU patients, data regarding hypocaloric over eucaloric nutrition are limited.[7] All, but one, of the hypocaloric, high-protein feeding studies entail the use of PN rather than EN support. In a recent retrospective study by Dickerson et al,[54] hypocaloric nutrition was compared with higher caloric EN regimen in 40 adult critically ill obese patients (weight 125% of IBW) in surgical ICU. Patients received either 19 total kcal/kg ABW per day (30 total kcal/kg IBW/d; n = 12) or 11 total kcal/kg ABW per day (22 total kcal/kg IBW/d; n = 28). Mean protein intakes were 1.9 g/kg IBW per day and 1.5 g/kg IBW per day, respectively. They reported their results after 4 weeks of evaluation with no statistically significant difference in nitrogen balance between the 2 groups (-1.6 ± 5.8 g/d vs. -3.9 ± 7.0 g/d during the first week, P = NS; -1.4 ± 6.0 g/d vs. -2.7 ± 5.9 g/d during the second week, P = NS). In addition, both groups had statistically significant rises in serum prealbumin concentrations from baseline by day 16 of nutrition support therapy. But, the hypocaloric group had a shorter ICU length of stay, shorter duration of antibiotic use, and a trend toward decrease in the number of ventilator days. Overall, average serum glucose concentrations and length of hospital stay was not significantly different between groups. The researchers concluded that low-calorie (hypocaloric) EN support was at least as effective as a higher-caloric feeding and recommended a large, prospective, randomized, and double-blind-controlled trial to confirm their results.

Other hypocaloric studies used the parenteral route with variable dosing of protein feeding and total kcal intake. Choban et al[55] demonstrated better glucose control with lesser need for exogenous insulin, but with no difference in mortality between the 2 groups. Overall, the average total caloric intakes in those studies ranged from 11 to 14 total kcal/kg ABW per day or 22 to 25 total kcal/kg IBW per day.[7] Accordingly, most clinicians will consider enteral over parenteral hypocaloric high-protein nutrition to obese ICU patients with a

BMI >27 kg/m^2 or who are >125% to 130% of IBW. However, for obese ICU patients with BMI >30 kg/m^2 or current weight >150% of IBW, hypocaloric high-protein nutrition would be considered a less controversial indication.[7]

Few caveats should be kept in mind when prescribing hypocaloric high-protein nutrition. First, although the primary objective is to avoid overfeeding while providing sufficient protein and calories to achieve anabolism and minimize catabolic losses. Weight loss in the form of body fat loss can be only a secondary benefit and should not be the primary objective. Second, progressive renal insufficiency without renal replacement therapy and HE are possible contraindications to the use of hypocaloric, high-protein approach as higher protein intake may aggravate the underlying conditions. Other potential contraindications for hypocaloric, high-protein feeding include patients with a history of hypoglycemia, age greater than 60 years old, or severely immunocompromised hosts.[7]

■ Conclusions

In conclusion, providing nutrition support for the critically ill patients represents a unique challenge to the medical team. However, certain recommendations can be stressed. First, begin nutrition support as soon as possible to attenuate the stress-induced hypermetabolism. Second, ABW should be used in energy expenditure calculations by H-B equation or other similar equations. Third, use indirect calorimetry for accurate determination of energy needs especially in the presence of fluid overload or obesity. Fourth, when nutrition support is initiated optimize protein administration followed by lipids and finally carbohydrates. Fifth, for critically ill obese patients consider hypocaloric, high-protein nutrition support with the potential for improved protein anabolism and better glycemic control.

Finally, once these initial goal intakes are achieved, the nutritional regimen may need to be readjusted particularly additional protein, depending on the patient's clinical response such as healing of wounds rather than routine laboratory revaluation.

These and other similar activities can be effectively enhanced by the hospital "Nutrition Support Team" who can be instrumental in establishing safe, timely, and cost effective provision of nutrition support for all critically ill patients.[56]

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