Nutrition in Critical Care

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Hospital studies estimate that 20%–55% of patients are nourished inadequately (Huang, 2001). This number includes many patients with cancer and patients on critical care units. When confronted with critical illness, the body generates a fight or flight response that causes the release of catabolic hormones to increase the body’s metabolic rate and caloric demands. To meet the demand for increased energy, the body breaks down existing fat and muscle tissue, leading to a negative nitrogen balance and compromised defense mechanisms. Loss of nutritional stores causes delayed wound healing, loss of muscle strength, diminished activity tolerance, and reduced white blood cell activity and has been linked to increased mortality. The gastrointestinal (GI) tract, a major storage site for immune cells, is highly susceptible to compromise if early nutritional support is not implemented. A great deal of clinical debate has ensued regarding the nature of the nutrients and best nutritional supplement methods for patients with critical illness.

The Role of the Gut in Critical Illness

Gut dysfunction is a significant contributor to morbidity in chronic critical illness. The exact physiologic mechanism for this is unclear. Some have proposed that its role as an immunologic organ and high tendency for ischemic damage predispose patients to bacterial translocation through inflamed and permeable bowel walls (Moore, 1999). Because the gut is the reservoir of ingested microorganisms (e.g., bacterial, fungal, viral) and normal flora bacteria, this is a feasible explanation for higher rates of bacteremia in patients with compromised gut function. Additionally, several immunologic functions that decrease the development of microorganisms within the GI tract exist (see Table 1).

The internal lumen of the GI tract is lined with a mucosal layer that is highly susceptible to atrophy and degeneration when the bowel is perfused inadequately or not exposed to nutrients. Many critically ill patients have some degree of perfusion deficit, and the gut is one of the first organs to have blood shunted away from it. Reduced blood flow not only causes mucosal atrophy but also leads to reduced peristalsis. During normal intestinal motility, pathogens attached to the mucosa are dislodged and moved into the colon for removal, limiting patients’ colonization with bacteria. Reduced peristalsis and altered mucosal surfaces combine to create an internal GI environment conducive to bacterial growth and translocation across the bowel wall into the blood stream. If administered, enteral foods can stimulate the gut mucosa to create a thicker mucus barrier to microorganisms (Lord & Sax, 1994). Other gastrointestinal complications of critical illness that cause interruption in normal gut function include circulatory shock, endotoxemia or sepsis, systemic inflammatory response syndrome, reperfusion injury, hypochlorhydria, aspiration pneumonia, and bacterial translocation.

Sepsis is a condition during which increased cardiac output and vasodilatation occur as a result of a primary or secondary infection (commonly nosocomial infection). The major nosocomial infections found most often in critically ill patients include urinary tract infections, wound infections, abscesses, and aspiration pneumonia. During sepsis, the GI tract’s oxygenation needs are increased. If oxygen needs are not met, GI hypoxic cell injury can occur. In the worst-case scenario, intestinal ischemia can progress to irreversible bowel necrosis, which replaces the affected area with fibrotic tissue and becomes gangrenous.

Hypochlorhydria occurs when blood flow to the stomach is decreased and oxygen needs are unable to be met, thus causing ischemia. Stress ulcers occur when layers of mucosa dissolve and superficial mucosa begins to erode (Lord & Sax, 1994). Stress ulcer prophylaxis uses H2 blockers or antacids to decrease hydrochloric acid in the stomach. However, current practice recommends use of a proton pump inhibitor (i.e., Protonix® [pantoprazole sodium, Wyeth-Ayerst, Philadelphia, PA]) in place of or as an alternative to H2 blockers. Another alternative is the use of sucralfate, which reacts with hydrochloric acid to form a protective gastric mucosal barrier.

Aspiration pneumonia occurs in critically ill patients when gastric acids reflux into the respiratory tract as a result of esophageal atony present after trauma, shock, or surgery (Lord & Sax, 1994). Nasogastric (NG) tubes can prevent reflux by decompressing and draining the stomach but also can be a cause...
of infection. Bacteria can travel up the NG tube and rest in the nasopharynx. The bacteria can proliferate and, if aspirated, lead to pneumonia.

Bacteria translocation occurs during periods of stress or deprivation of intestinal nutrients. The exact mechanism of bacteria translocation is not fully known, but an alteration in intestinal wall permeability is thought to be the source for bacteria to move from the bowel lumen to the bloodstream. Normal gut flora also move freely through the bowel wall to provide protection against pathogenic bacteria, but their presence in the lymph nodes and circulatory system ultimately can lead to sepsis (MacFie et al., 1999).

### Nutritional Support

In an ideal scenario, nutritional support should be started within 48 hours of critical illness or injury. In reality, nutrition needs of critically ill patients are considered lower priority, as cardiopulmonary function is essential during the initial 24–48 hours. During this period, patients’ metabolic demands increase to provide the energy necessary to combat illness and begin the process of repair. As patients become hypermetabolic and hypercatabolic secondary to their critical illness, clinicians should provide interventions such as enteral or parenteral nutrition to avoid an increased hypercatabolic state and improve desired outcomes (Stamatos & Reed, 1994). Such efforts may include enhancing cardiac output (e.g., using inotropic agents), maximizing oxygen carrying capacity (e.g., administering blood products), and conserving energy reserves (e.g., administering sedatives). Once patients pass the acute phase, nutritional support must be considered. At this point, nutritional requirements must be met to avoid potential complications such as muscle wasting, immune suppression, sepsis, and eventual multisystem organ failure.

The gut can atrophy and lose 50% of its muscle mass within three to five days of nonfeeding (Lord & Sax, 1994). Therefore, enteral feedings must be started as soon as possible. Enteral feeding is considered the route of choice, but, if necessary, parenteral feeding may be used until enteral feedings can be started. Patients with severe bowel injury or pancreatitis and immediately postoperative patients may not be able to be fed enterally. Because the small bowel does not lose its function readily, the duodenum is the preferred placement for a feeding tube for patients requiring enteral nutritional support (Keithley & Eisenberg, 1993). This placement bypasses the slow gastric motility, allowing for increased absorption and less risk of pulmonary aspiration. Alterations in the GI tract, hyperosmolarity of feeding solutions, and the use of antimicrobial agents may cause increased production of bacteria and lead to diarrhea. Administering yogurt mixed with water in a 1:1 ratio twice a day can reduce or alleviate diarrhea. Critically ill patients must not be overfed, particularly with glucose calories, because overfeeding can lead to increased carbon dioxide production (Pingleton, 2001). Several pulmonary care enteral formulations are available.

When parenteral nutrition is used until enteral feedings are possible or in conjunction with enteral feeding to provide adequate total nutrition, it can provide the majority of nutrients, fluids, and electrolytes necessary for patients and be regulated for optimal acid base management. However, two setbacks to parenteral nutrition exist. First, the cost is three times that of enteral therapy, and second, it does not supply the protective factors of enteral therapy (Kirby, 2001).

### Vitamins and Minerals

Little is known about vitamin and mineral requirements in various disease states. The administration of minimum daily requirements of vitamins and minerals is routine practice, but adequacy of absorption should be considered if nutrients are being administered enterally. Water-soluble vitamins (e.g., thiamine, riboflavin, niacin, pyridoxine, pantothenic acid, B12, folic acid, biotin, ascorbic acid) are absorbed via the small bowel, but fat-soluble vitamins (e.g., A, D, E, K) are absorbed in the large bowel. Patients experiencing diarrhea in conjunction with their enteral feedings may experience fat-soluble vitamin deficiency.

Resources indicate that vitamin and mineral requirements are altered because of increased losses, greater utilization, or both. Traumatic stress results in alteration of trace elements and minerals. Vitamins A and C are necessary for wound healing. Thiamine and folic acid are necessary in patients with a history of alcohol abuse, and increased zinc and potassium are required in patients with excessive GI losses. Iron is essential for growth of many bacteria; therefore, replenishment can lead to increased risk of sepsis.

### Laboratory Values

Albumin, prealbumin, and transferrin screenings are the three most important laboratory tests to follow in assessment of nutritional status. Serum albumin screening is the most commonly used laboratory test for malnutrition, although not the most sensitive. Albumin has a half-life of 20 days, so patients may be malnourished before their serum albumin actually falls below normal. Albumin levels lower than 3.1 g/dL can contribute to decreased immune function, poor wound healing, severity of pressure ulcers, tissue edema, pulmonary edema, and adult respiratory disease syndrome (Lord & Sax, 1994). Low albumin levels also cause changes in gut permeability and defense mechanism failure. Prealbumin and transferrin correlate with negative protein balance. Prealbumin has a half-life of two days; transferrin is more discriminating than albumin and has a half-life of 2–10 days, but it also can decrease if a patient is iron deficient. Prealbumin and transferrin tests are expensive, hence the prevalent use of albumin as an indicator of nutritional deficits.

Other laboratory tests that should be conducted include phosphorus, magnesium, and carbon dioxide screenings. Decreased phosphorus levels can cause decreased tissue oxygenation and respiratory muscle weakness, low magnesium levels can cause respiratory muscle weakness and increased weaning difficulties in ventilated patients, and low or high carbon dioxide levels can lead to decreased respiratory effort and prolonged ventilatory weaning.

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**TABLE 1. MAJOR IMMUNOLOGIC GASTROINTESTINAL FUNCTIONS**

<table>
<thead>
<tr>
<th>GASTROINTESTINAL FUNCTION</th>
<th>IMMUNOLOGIC BENEFIT</th>
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<tbody>
<tr>
<td>Gastric acid</td>
<td>Destroys most bacteria</td>
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<tr>
<td>Peristalsis</td>
<td>Limits colonization and overgrowth of microorganisms</td>
</tr>
<tr>
<td>Presence of gut microflora</td>
<td>Limits colonization and overgrowth of microorganisms</td>
</tr>
<tr>
<td>Weekly enteral desquamation</td>
<td>Limits colonization and overgrowth of microorganisms</td>
</tr>
<tr>
<td>Presence of gastrointestinal mucus layer</td>
<td>Enzymes and glycoproteins found in mucus disrupt bacterial cell walls.</td>
</tr>
<tr>
<td>Presence of bile</td>
<td>Detoxifies foreign substances</td>
</tr>
</tbody>
</table>

*Note. Based on information from Lord & Sax, 1994; Moore, 1999.*
Indirect Calorimetry

Indirect calorimetry measures oxygen consumption and carbon dioxide production to calculate resting energy expenditure (Trujillo, Robinson, & Jacobs, 1999) and provides a precise measurement of patients’ daily caloric expenditures and respiratory quotients. Energy expenditure takes into account the effects of disease state, stress, and trauma. An activity factor can be used if patients are not on bedrest. The respiratory quotient is the ratio of expired carbon dioxide to inspired oxygen. The normal ratio level is 0.7–1.0. A ratio level of less than 0.2 indicates malnutrition; a ratio level of more than 1.0 indicates overfeeding (Battezzati & Vigano, 2001). Measurement of energy expenditure by indirect calorimetry is useful in critically ill patients who undergo changes in body composition that alter balance between body weight and metabolic active tissue. Substantial edema, limb amputation, and hypermetabolic states can change caloric requirements. Energy needs of patients who cannot be weaned from mechanical ventilation should be measured to ensure that they are not being over- or underfed and that the macronutrient composition of the feeding is appropriate (Trujillo et al.).

Underfeeding critically ill patients can significantly impair their ability to respond to stress, lead to deficiencies in vitamins and minerals, impair the production and maturation of lymphocytes, and disrupt the mucosal integrity of the GI tract. On the other hand, overfeeding can lead to respiratory complications secondary to hyperglycemia, fatty liver, increased triglycerides, and increased lipogenesis and may increase the potential for the development of cholestasis and pancreatitis. The excess carbon dioxide production associated with lipogenesis can cause respiratory failure if patients are not able to adequately increase ventilation (Trujillo et al., 1999).

Conclusions

Now that clinicians recognize the clinical importance of malnutrition and patients at highest risk for developing it, research has turned its focus to finding the best nutrient combination or substrates to maximize clinical benefits. Because most critically ill patients are hypermetabolic or infected, general nutrition prescriptions can provide for high protein and low glucose calories. A formula providing a common mixture of nutrients that will help to maintain lean muscle mass and provide the substrates necessary for increased energy demands is made up of 20% protein, 60%–70% carbohydrates, and 20%–30% fat (Pingleton, 2001). A new process known as immunonutrition uses increased additives such as essential amino acids, glutamine, and growth hormones to supply the nutrients needed to enhance gut mucosal proliferation and decrease inflammatory mediators (Heyland et al., 2001). These additives are thought to help to restore the immune dysfunction experienced with critical illness, but existing studies are small and clinical benefits have been inconsistent (Aosasa, Mochizuki, Yamamoto, Ono, & Ichikura, 1999; Heyland et al.). Further research in how to best meet the nutritional needs of critically ill patients still is needed.

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References


For more information on this topic, visit the following Web sites:

Total Parenteral Nutrition in the Critically Ill Patient: A Meta-Analysis
http://www.trauma.org/anaesthesia/metanalysis/tpn.html

Yale University: Nutrition Literature Resource
http://info.med.yale.edu/pediat/criticare/NutritionResource.htm

These Web sites are provided for information only. The hosts are responsible for their own content and availability. Links can be found using ONS Online at www.ons.org.