Nutrition in Critical Care

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Many hospitalized patients with cancer are malnourished. Some become critically ill and experience delayed wound healing, loss of muscle strength, and reduced infection fighting ability as a consequence of the loss of nutritional reserves. Complications of critical illness may cause interruption in normal gastrointestinal function and result in shock, sepsis, hypochlorhydria, systemic inflammatory response syndrome, and other disorders. As a result, critically ill patients may require nutritional support.

Hospital studies estimate that 20%–55% of inpatients 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

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