

UPDATES IN NUTRITIONAL SUPPORT FOR PATIENTS IN INTENSIVE CARE UNITS

An Essay

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in Intensive care medicine**

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List of Abbreviations

ADL :	Activities of daily living.
AGA :	American Gastroenterological Association.
AIDS :	Aquired Immunodeficiency Syndrom.
ALAT :	Alanine Aminotransferase.
APACHE :	Acute Physiology and Chronic Health Evaluation.
ARDS :	Acute Respiratory Distress Syndrome.
ASAT :	Aspartate Aminotransferase.
BEE :	Basal Energy Expenditure
CRP :	C-reactive protien.
CRS :	Catheter-related sepsis.
CVP :	Central venous pressure.
CVVHD :	Continuous Veno- Venous Haemodialysis.
DM :	Diabetes Mellitus.
EE :	Energy Expenditure.
EN :	Enteral Nutrition.
GALT :	Gut-associated Lymphoid Tissue.
GGT :	Gamma glutamyle transferase.
GI :	Gastrointestinal.
GIT :	Gastrointestinal tract.

GRV :	Gastric residual volume.
HIV :	Human Immunodeficiency Virus.
ICU :	Intensive Care Unite.
INR :	International normalized ratio.
IV :	Intravenous.
JPE :	Jejunal percutaneous endoscopy.
LOS :	Length Of Stay.
MAC :	Mid-arm circumference.
MALT :	Mucosa-associated Lymphoid Tissue.
MPM :	Mortality Predicting Model.
NG :	Nasogastric.
PCM :	Protien-caloric malnutrition.
PEG :	Percutaneous endoscopic gastrostomy.
PEM :	Protien energy malnutrition.
PINI :	Prognostic Inflammatory and Nutritional Index.
PN :	Parenteral Nutrition.
POI :	Postoperative ileus.
PT :	Prothrombin time.
QOL :	Quality of life.
RBP :	Retinal-binding protien.
SAPS :	Simplified Acute Physiological Score.
TPN :	Total parenteral nutrition.
UMAC :	Upper mid-arm circumference.

Introduction

An increasing nutritional deficit during a long ICU stay is associated with increased morbidity (infection rate, wound healing, mechanical ventilation, length of stay, duration of recovery), and costs (*Heidegger et al., 2008*).

Recent studies in both trauma, surgical and non surgical patients support the superiority of early enteral over early parenteral nutrition (*De Aguilar-Nascimento and Kudsk, 2008*). Guidelines that were published recently suggest that when enteral feeding is not possible, parenteral nutrition should be initiated within 7 days (*McClave et al., 2009*). or within 3 days (*Singer et al., 2009*).

The principal indication for EN is a functional GI tract with sufficient length, absorptive capacity and the inability to take nutrients through the oral route either totally or in part (*Robin et al., 2009*). While the process of administering EN may appear less complex compared with parenteral nutrition (PN), serious harm and death can result due to potential adverse events occurring throughout the process of ordering, administering, and monitoring (*Robin et al., 2009*).

There have been multiple reports of adverse events related to EN. These events include reports of enteral misconnections (*Guenter et al., 2008*) enteral access device misplacements (*Bankhead et al., 2007*) and displacements, metabolic abnormalities, mechanical tube complications, bronchopulmonary aspiration, GI intolerance related to formula contamination, and drug-nutrient interactions (*Malone et al., 2007*).

Early enteral nutrition is recommended for critically ill patients. Supplemental parenteral nutrition combined with enteral nutrition can be considered to cover the energy and protein targets when enteral nutrition alone fails to achieve the caloric goal (*Heidegger et al., 2008*).

There seems to be increased favor for combined enteral-parenteral therapy in cases of sustained hypocaloric enteral nutrition. The key issue is when the dual regimen should be initiated (*De Aguilar-Nascimento and Kudsk, 2008*).

Both Parenteral and enteral nutrition are used for metabolic support when patients cannot take adequate amounts of intake orally with the primary goal of avoiding progressive lean tissue catabolism due to starvation (*Kenneth and William, 2007*).

Nutritional State

Nutritional state is a major determinant of the response to illness; in particular resistance to infection, maintenance of gut, lung function and wound healing (*Biolo et al., 2007*).

The nutritional state of hospitalized patients reflects directly on their clinical course, given that there are greater rates of hospital-acquired diseases and deaths and greater risk of clinical complications among malnourished patients, increasing the hospital length of stay (LOS) and reducing quality of life. This leads to high hospital costs because these patients have a greater need for intensive care or specialized services (*Kyle et al., 2005*).

Nutrient demand is exacerbated in the case of illness. The underlying nutritional status of the patient and the complexity of the metabolic response to injury and critical illness provide the basis for nutritional therapy. Critical illness, in particular, is associated with a marked increase in metabolism, leading to greater energy (i.e. caloric) requirements and loss of lean body mass (*Hadley and Hinds, 2002*).

Medical and surgical critically ill patients are subjected to stress, infection and impaired organ function, resulting in a hypercatabolic state, leading to metabolic derangement and malnutrition (*Griffiths, 2003*).

The incidence of malnutrition worsens over time in patients who require prolonged hospitalization (*McClave et al., 2006*).

Protein-energy malnutrition is associated with skeletal-muscle weakness, an increased rate of hospital-acquired infection, impaired wound healing, and prolonged convalescence in patients who are admitted to an intensive care unit (ICU) (*Singer et al., 2009*).

Critically ill patients are characterized by a number of alterations in carbohydrate, lipid, amino acid, protein and electrolytes metabolism. These changes (proportional to the severity of illness) lead to increased energy requirement and protein catabolism and contribute to alterations of the immune system, the body composition and the muscle and gastrointestinal tract functions. The metabolic response to injury mobilizes amino acids from lean tissues to support wound healing, immunologic response and accelerated protein synthesis for repairing processes. The endogenous protein breakdown leads to depletion and sub clinical malnutrition of rapid onset. There is strong evidence that malnutrition is an independent risk factor for higher morbidity, increased length of hospital stay, higher readmission rates, delayed recovery, lower quality of life as well as higher hospital costs and higher mortality (*Zanella et al., 2006*).

Critical illness is typically associated with a catabolic stress state in which patients commonly demonstrate a systemic inflammatory response. This response is coupled with complications of increased infectious morbidity, multi-organ dysfunction, prolonged hospitalization, and disproportionate mortality. Over the past 3 decades, the understanding of the molecular and biological effects of nutrients in maintaining homeostasis in the critically ill population has made exponential advances. Traditionally, nutrition support in the critically ill population was regarded as adjunctive care designed to provide exogenous fuels to support the patient during the stress response. This support had 3 main objectives: to preserve lean body mass, to maintain immune function, and to avert metabolic complications. Recently these goals have become more focused on nutrition therapy, specifically attempting to attenuate the metabolic response to stress, to prevent oxidative cellular injury, and to favorably modulate the immune response. Nutritional modulation of the stress response to critical illness includes early enteral nutrition, appropriate macro- and micronutrient delivery, and meticulous glycemic control. Delivering early nutrition support therapy, primarily using the enteral route, is seen as a proactive therapeutic strategy that may reduce disease severity, diminish complications, decrease length of stay in the ICU, and favorably impact patient outcome (*McClave et al., 2006*).

The body's reaction to stress is associated with an elevation in body temperature, in cardiac output and in substrate turnover rate, all of which should lead to an increase in energy demand. The elevation in energy expenditure (EE), primarily controlled by the counter-regulatory hormones, is directly related to the extent and type of injury and sepsis. For example, minor or localized infections generally have little effect on EE and increases above 5-15% are rarely observed. Elevations of 10-15% are frequently seen in patients presenting with severe infection or multiple trauma. The greatest increases in EE are documented in patients with uncontrolled sepsis, with or without the Acute Respiratory Distress Syndrome (ARDS), and in burn patients. On the other hand, prolonged starvation, severe PCM, physical immobilization, sedation and/or muscle relaxation associated with critical illness decrease EE by 15-20% and counter the hypermetabolic effects of the underlying injury (*Nitenberg, 2000*).

Critical illness is associated with catabolic hormonal and cytokine responses. Cytokines act synergistically with the stress hormones in mediating much of the metabolic disturbances seen after injury, trauma or sepsis (*Lin et al., 2000*).

These include increased blood levels of counter regulatory hormones (e.g. cortisol, catecholamines, and glucagon), increased blood and tissue levels of proinflammatory cytokines (e.g. interleukin-1, interleukin-6, interleukin-8, and tumor necrosis

factor α), and peripheral-tissue resistance to endogenous anabolic hormones (e.g. insulin and insulin-like growth factor 1) (**ASPEN, 2002**). This hormonal milieu increases glycogenolysis and gluconeogenesis, causes a net breakdown of skeletal muscle, and enhances lipolysis, which together provide endogenous glucose, amino acids, and free fatty acids that are required for cellular and organ function and wound healing (**ASPEN, 2002**).

Unfortunately, although plasma substrate levels may be increased, their availability for use by peripheral tissues may be blunted (because of factors such as insulin resistance and inhibition of lipoprotein lipase), and plasma levels of certain substrates (e.g. glutamine) may be insufficient to meet metabolic demands (**Bongers et al., 2007**).

Recovery from critical illness is characterized by anabolism exceeding catabolism. Nutritional support provides substrate for the anabolic state, during which the body corrects hypoproteinemia, repairs muscle loss, and replenishes other nutritional stores (**Dvir et al., 2006**).

Malnutrition is characterized by protein/energy depletion. Clear correlations between parameters reflecting poor nutrition, such as prealbumin or body mass index and rate of in-hospital complications, readmissions and mortality (**Correia and Waitzberg, 2003**).

Malnourished patients recover more slowly from illness and experience more complications such as poor wound healing or altered immune function (***Middleton et al., 2001***).