Controversies in Critical Care Nutrition Support

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KEYWORDS

• Nutritional risk • Enteral nutrition • Parenteral nutrition • Trophic feeding
• Autophagy • Permissive underfeeding • Shock

KEY POINTS

- Nutritional risk is the risk of acquiring complications and other adverse outcomes that might have been prevented by timely and adequate nutrition support.
- In critically ill patients with an intact gut, early enteral nutrition (EN) is preferred to parenteral nutrition (PN).
- In low-risk patients with contraindications for EN, starting PN can be delayed for 7 to 10 days, but in high-risk patients with contraindications for EN, starting PN within 24 to 48 hours is reasonable.
- The exact timing for adding supplemental PN to hypocaloric EN remains controversial but, in high-risk patients, adding supplemental PN after day 7 is reasonable.
- Despite studies showing tolerability and lack of complications, further research is needed to evaluate dose and timing of EN in shock.

INTRODUCTION

Critical illness predisposes individuals to highly variable metabolic and immune responses, leading to muscle mass loss, impaired healing, immobility, and susceptibility to infections and cognitive impairment.[1] Previously thought of as adjunctive therapy, nutrition support is a form of primary therapy in critically ill patients with both nutritional and non-nutritional benefits. Recommendations for nutrition in critically ill patients are supported by observational studies, small randomized controlled trials (RCTs), and mechanistic data.[2] Controversies such as the type, quantity, and timing

Disclosure: The authors have nothing to disclose.

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Crit Care Clin 32 (2016) 173–189
http://dx.doi.org/10.1016/j.ccc.2015.11.002 **criticalcare.theclinics.com**
0749-0704/16/$ – see front matter

of nutrition support and the role of nutrition during hemodynamic instability have limited the widespread application of nutrition support. This article reviews the physiologic basis for nutrition support and the concept of nutritional risk, and reviews various controversies in critical care nutrition support. This article does not discuss controversies in composition of nutrition support or nutrition support in specific disease subsets. The reader is referred to societal guidelines for a thorough review of these topics.[3,4]

PHYSIOLOGIC BASIS FOR NUTRITION SUPPORT

In 1930, David Paton Cuthbertson described 3 successive phases of the metabolic response to critical illness. The first is an ebb phase, reducing basal metabolism. The second is a hypercatabolic flow phase, characterized by major protein catabolism, and leading to a reduced pool of muscle amino acids and glutamine. Third is an anabolic phase of muscle mass reconstruction, healing, and progression toward homeostasis.[1] However, at the onset of the anabolic phase, muscle atrophy may be severe and reduced muscle mass has been associated with poor intensive care unit (ICU) outcomes.[1,5]

The metabolic response to stress is complex and involves activation of neuroendocrine, inflammatory/immune, adipose tissue hormones, and gastrointestinal (GI) hormone components. The neuroendocrine component begins within seconds to minutes of the stress, activating the sympathetic nervous system and hypothalamic-pituitary axis. The inflammatory/immune components are activated within days, and lead to release of cytokines and inflammatory mediators such as tumor necrosis factor, interleukin-1, and interleukin-6. In addition to orchestrating the systemic inflammatory response syndrome (SIRS), these cytokines also induce weight loss, proteolysis, and lipolysis.[6] In addition, gene expression is altered in response to severe stress with increased expression of genes involved in the SIRS response and suppression of genes involved in adaptive immunity.[6] Adipokines (released from fat tissue) such as leptin, resistin, and adiponectin potentially contribute to the metabolic response to stress. In addition, levels of the GI tract hormone ghrelin are reduced and levels of cholecystokinin and peptide YY are increased.[6] Activation of these pathways ultimately leads to uncontrolled catabolism, insulin resistance, increased energy expenditure, and use of energy substrates (**Fig. 1**).

Uncontrolled catabolism leads to a cumulative calorie deficit. A negative cumulative energy balance has been associated with occurrence of acute respiratory distress syndrome, renal failure, need for surgery, and pressure sores.[7] The delivery of exogenous nutrients via the enteral or parenteral routes can provide sufficient calories; it can deliver micronutrients, and antioxidants for energy substrate repletion and maintenance of daily caloric balance. In addition, protein supplementation can restore protein stores and preserve lean body mass.[8]

However, it is enteral nutrition (EN), as opposed to parenteral nutrition (PN), that provides non-nutritional benefits (**Box 1**). Consider the consequences of not providing EN. In the absence of luminal nutrients, there is loss of structural and functional integrity.[8] Reduced gut contractility promotes bacterial overgrowth and increases bacterial virulence with contact-dependent programmed enterocyte apoptosis. Enterocyte death leads to structural defects, enhancing gut permeability for bacterial translocation and ultimately increasing the SIRS response.[8]

Providing EN maintains functional and structural integrity by stimulating intestinal contractility to sweep bacteria downstream and reduces bacterial overgrowth.