Critical Care Nutrition

Where's the Evidence?

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KEYWORDS

• Critical care nutrition • Enteral nutrition • Parenteral nutrition • Nutritional risk
• Supplemental parenteral nutrition • Permissive underfeeding • Trophic feeding
• Metabolic stress response

KEY POINTS

- Critical illness takes one of three paths: the first is fulminant sepsis and end organ disease leading to death, the second; the patient returns to a preinjury or illness state and third; the patient enters a state of persistent inflammatory state.
- Enteral nutrition has numerous non-nutritional benefits including; attenuation of the metabolic response, helps maintain the gut associated lymphoid tissue and supports the gut integrity.
- The concept of defining nutritional risk and using that risk assessment to focus attention to those at greatest risk yields the optimal benefit from nutritional therapy.
- Despite the numerous benefits of enteral nutrition parenteral nutrition is still a valuable tool to support those patients in which enteral is either fully or partially unsuccessful.

INTRODUCTION

Critical illness induces a highly complex and variable metabolic response. Consequences of this metabolic response often lead to immunosuppression, reduced muscle mass, impaired wound healing, immobility, and cognitive impairment.[1] Randomized controlled trials (RCTs), observational studies, and mechanistic data suggest provision of nutrition is beneficial for critically ill patients to attenuate or

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prevent some of the consequences of metabolic response. The purpose of this review is to (a) outline the physiologic response of critical illness, (b) describe the concept of nutrition therapy and differentiate it from nutrition support, (c) define nutritional risk and describe which critically ill patients may benefit from early aggressive nutritional optimization, (d) discuss the optimal dose of enteral nutrition (EN), (e) define the role and timing of parenteral nutrition (PN) in critical illness, and (f) define which critically ill patients may benefit from immunonutrition.

THE METABOLIC RESPONSE TO CRITICAL ILLNESS AND THE NUTRITION SUPPORT

In general, critically ill patients follow 1 of 3 clinical trajectories. The first trajectory is the patient who has an acute insult (eg, trauma, sepsis) that leads to rapid and widespread organ dysfunction. Despite aggressive resuscitation and supportive care measures, this trajectory leads to fulminant death. The second trajectory is the patient who has an acute insult, wherein resuscitation and supportive care measures follow. The underlying insult is reversed, and homeostasis is re-established; the metabolic response shifts from catabolic to anabolic. In the second trajectory, the duration of intensive care unit (ICU) stay may be a few days up to 2 weeks. The third trajectory is an extension of the second except the catabolic response persists and leads to chronic critical illness manifesting as persistent inflammation, muscle wasting, immunosuppression with propensity for nosocomial infections, and persistent organ dysfunctions (eg, dependence on mechanical ventilation and renal replacement therapy). This third phenotype is also known as persistent inflammatory immunosuppressed catabolic syndrome.[2]

What is the role of nutrition in each of these trajectories? When it is clear that patients enter the first trajectory, there is probably little or no role for early and/or aggressive nutrition support, because despite resuscitative and supportive care measures, the outcome is fulminant death. To better understand the role of nutrition in the second and third trajectories, it is important to comprehend the metabolic response to stress.

The metabolic stress response activates numerous pathways, including neuroendocrine, immune/inflammatory, and adipokine/gastrointestinal. Within seconds to minutes, sympathetic nervous system activation increases adrenergic receptor activity and adrenal medullary output of norepinephrine and epinephrine. Within seconds to hours, the neuroendocrine component turns on the sympathetic nervous systems and hypothalamic-pituitary axis. Within hours, the hypo-thalamic-pituitary axis increases anterior pituitary release of adrenocorticotropic hormone, thyroid-stimulating hormone, follicle-stimulating hormone, luteinizing hormone, and growth hormone. An immune response increases cytokines and inflammatory mediators. Tumor necrosis factor, interleukin-1 (IL-1), and IL-6 induce fevers, proteolysis, and lipolysis and trigger anorexia.[3] Adipokines, released from adipose tissue, such as leptin, resistin, and adiponectin, which are now thought to be important in the overall response, are currently being investigated for their role in modulating the metabolic stress response.[3] Gastrointestinal responses to metabolic stress are exceedingly complex and include increased cholecystokinin and peptide YY, which release digestive enzymes and reduce hunger, changes in gut integrity, and distorted motility.[3,4] The new paradigm for the human immune response to stress (eg, trauma, sepsis) includes simultaneous activation of both systemic inflammatory innate immune and compensatory anti-inflammatory responses with simultaneous adaptive immunity gene suppression.[4]

Activation of the neuroendocrine, immune/inflammatory, and adipokine/gastrointestinal pathways leads to accelerated catabolism, insulin resistance, increased