The conundrum of underfeeding vs overfeeding neurocritically ill patients

Is less better?

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Critically ill patients experience a hypercatabolic state from inflammatory mediators, sympathetic nervous system hyperactivity, dysregulation of endogenous glucose metabolism, and development of insulin resistance.[1] The premise of increased inflammatory response and hypermetabolism of critical illness has led to the implementation of nutrition therapy in patients in the intensive care unit (ICU). In fact, introduction of early enteral nutrition or delayed parenteral nutrition to supplement enteral feedings has been associated with modest reduction in complications and faster recovery.[2,3] However, critically ill patients may express varied responses to injury and nutrition therapy and not all will derive the same benefit from the latter. For example, there is evidence that the beneficial effect of increased caloric intake may only be seen in patients with body mass index (BMI) <25 and ≥ 35,[4] and that a detrimental effect of underfeeding is seen in those patients receiving mechanical ventilation >7 days.[5] In an attempt to address these issues, McClave et al.[6] have suggested using severity of illness and markers of inflammation and starvation indices to identify critically ill patients who may benefit from more aggressive nutrition. Furthermore, there is evidence that nutrition delivery may help determine outcome. This is supported by standardized nutrition therapy protocols that may reduce the duration of mechanical ventilation and ICU length of stay.[7]

The literature offers incomplete and sometimes confusing evidence regarding the widespread application and methods of nutritional therapy in the ICU.[8] Some of the factors that need to be studied and more clearly delineated in future studies include the following: variability in nutrition therapy protocols across regions and countries; practitioners' perception of nutrition therapy as adjunctive support rather than primary treatment; standardized measurement of calorie consumption in the ICU; the common perception that obese patients may have adequate nutrition support and therefore may not need early nutrition treatment; determination of optimal methods to monitor nutritional requirements and response to therapy; use of immune-modulating enteral formulas; and a better understanding of the adequacy of nutrition therapy and the consequences of underfeeding and overfeeding in the ICU environment.

The evidence for nutritional status evaluation and nutrition therapy in the neurocritically ill patient is scant.[9] In this issue of *Neurology*®, Badjatia et al.[10] report on an analysis of the relationship of inflammation and negative nitrogen balance (NBAL) to nutritional status and outcomes after subarachnoid hemorrhage (SAH). The authors performed a single-center prospective observational study of 229 SAH patients. They performed measurements of C-reactive protein (CRP), transthyretin (TTR), resting energy expenditure (REE), and NBAL at 4 preset time periods during the first 14 postbleed days in addition to daily caloric intake. The latter were used as surrogate markers of systemic inflammation and energy and nitrogen requirements. In addition, they determined functional outcome at 3 months as defined by the modified Rankin Scale score. Subsequently, the authors built multivariable regression models to determine factors associated with changes in REE and NBAL. The main findings reported include the following: higher REE was associated with younger age, male sex, and more severe SAH; negative NBAL was associated with lower caloric intake, higher BMI, aneurysm clipping, and higher CRP:TTR ratio. Hospital-acquired infections were associated with older age, more severe SAH, lower caloric intake, and negative NBAL. Poor functional outcome at 3 months was associated with more severe SAH, older age, negative NBAL, hospital-acquired infection, higher CRP:TTR ratio, higher BMI, and delayed cerebral ischemia. The authors concluded that negative NBAL after SAH is influenced by inflammation and associated with an increased risk of hospital-acquired infections and poor outcome.

This study is important for several reasons. First, this is the largest observational study of SAH patients analyzing the association among REE, NBAL, and inflammatory markers with clinical outcome and the development of hospital-acquired infection. Second, the study design made repeated measurements of REE, NBAL, and inflammatory markers, which provide a better understanding of the natural history of the catabolic state following SAH. Third, underfeeding

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may be more common than previously realized in SAH patients, particularly in those who are not receiving mechanical ventilation. However, there are important limitations that need to be highlighted: the study was performed at a single center, limiting its generalizability. Also, the inflammatory markers used are nonspecific and offer only a crude measurement of the host of events that occur in SAH patients. Further, NBAL may not adequately represent or be sensitive enough to detect catabolism in all SAH patients. Finally, there was no determination of important clinical outcomes such as cognitive function and quality of life.

Regardless of its limitations, the study by Badjatia et al. raises important points for clinical practice and future studies of nutritional status determination and nutrition therapy in SAH patients. Clinicians caring for SAH patients must be vigilant of underfeeding or overfeeding and should work closely with their nutrition support teams to develop nutritional assessment and management protocols that fit their institutional practices and resources. Future validation studies in larger and diverse SAH populations need to be undertaken before the practice of serial REE and NBAL is recommended. If such validation studies become available, then more specific inflammatory and catabolic markers need to be studied with the goal of determining in future randomized controlled clinical trials what dosage and method of nutrition therapy is optimal for SAH patients.

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