Growth velocity during early infancy is higher than at any other time during childhood and is exceeded only by intrauterine growth rates\(^1\). Acute injury, however, markedly alters energy needs. First, acute injury induces a catabolic response that is proportional to the magnitude, nature, and duration of the injury. Increased serum counter-regulatory hormone concentrations induce insulin and growth hormone resistance\(^1\), resulting in the catabolism of endogenous stores of protein, carbohydrate, and fat to provide essential substrate intermediates and energy necessary to support the ongoing metabolic stress response. During this catabolic response, somatic growth cannot occur and, therefore, the caloric allotment for growth, which is substantial in infancy, should not be administered until the acute metabolic stress response resolves. Second, children treated in the intensive care setting are frequently sedated, and their activity level is markedly reduced, further lowering energy needs. Third, the intensive care environment is temperature-controlled and insensible energy losses are substantially reduced. This is especially true for children who are mechanically ventilated because, in addition to reduced energy needs for the work of breathing, these patients are ventilated with heated, humidified air. This can reduce insensible losses by one third. In concert, these factors result in a substantial decrease in energy needs\(^1\), even though some variation based on the magnitude and duration of injury response metabolism can occur\(^2\). To account for these alterations in energy metabolism, caloric amounts equal to measured energy expenditure values or basal energy requirements should be provided\(^3\). The significance of this therapeutic strategy is to avoid overfeeding (i.e., the provision of calories and/or nutritional substrates in excess of the energy required to maintain the metabolic homeostasis). Overfeeding increases ventilatory work by increasing CO\(_2\) production\(^1,4\).

This phenomenon can create or prolong the need for mechanical ventilation\(^1\), especially in the preterm baby with immature pulmonary development\(^5\). Overfeeding may also impair liver function by inducing hepatic steatosis and cholestasis, and can increase the risk of infection secondary to hyperglycemia\(^1,6,7,8\).
Nutrition assessment of critically ill pediatric patients can be quantitatively accomplished by measuring (1) the visceral (or constitutive) protein pool, (2) the acute-phase protein pool, (3) nitrogen balance, and (4) energy expenditure \(^1\). Serum prealbumin and CRP concentrations are readily measured in most hospitals and are good markers for the visceral and acute phase protein pools, respectively \(^1\). Albumin should not be used because, due to its substantially larger pool size and much longer half-life, it is more difficult to detect subtle acute catabolic and anabolic changes which occur in association with an evolving metabolic response to injury. Within 12 to 24 hours following injury, serum prealbumin levels fall, reflecting catabolic metabolism, and CRP levels rise because of hepatic reprioritization of protein synthesis in response to injury \(^1\). Serum prealbumin and CRP levels are inversely related (i.e. serum prealbumin levels decrease and CRP levels increase with a magnitude proportional to injury severity, and then return to normal as the acute injury response resolves), and should be measured serially to establish the response pattern. Serum prealbumin and CRP concentrations have been shown to be useful in predicting clinical outcome in critically ill infants \(^9,10\). Furthermore, serum CRP concentrations have been shown to correlate well with MEE in this patient population \(^11\). Decreases in serum CRP values to less than 2 mg/dL in conjunction with increases in serum prealbumin levels have been associated with the return of anabolic metabolism \(^4\). Energy expenditure can be measured a bedside using a metabolic cart (indirect calorimetry) \(^1,12\). Although a variety of predictive equations have been proposed, clinical and patient variability (especially in the infant population) frequently cause these estimates to be inaccurate. Equations which incorporate metabolic stress variables (such as body temperature) can provide improved predictive results in appropriate patient populations \(^1,13\) however, direct measurement of energy expenditure in individual patients achieves greater accuracy and can help to avoid overfeeding. In addition, serial measurements in acutely ill infants can show changes in respiratory quotient which help to identify resumption of anabolic metabolism after injury \(^1,4\).

**Clinical Strategy**

To avoid overfeeding children during acute metabolic stress, energy expenditure should be measured. A study of critically ill children less than 2 years of age in a pediatric intensive care setting showed that actual measured energy expenditure values average about 50% of what the predicted energy requirement would be for those children if they were healthy and normally active \(^1\). Energy delivery should not exceed measured values during the acute metabolic stress period. When the respiratory quotient decreases to 1.0 in infants up to 1 year of age, caloric
intake may be advanced judiciously to meet predicted energy requirements for age and weight. In the absence of indirect calorimetry, published basal energy requirements based on age, weight, and gender offer reasonable guidelines for basal energy requirements 1,3. During metabolic stress, for infants up to 1 year of age, macronutrient substrates should be provided as follows: protein (2.5. to 3.0 g/kg per day), carbohydrate (8.5 to 10 g/kg per day), and fat (1 gm/kg per day).

Decrease of serum CRP values to less than 2 mg/dL and rising prealbumin values may be used as alternative guidelines for advancing caloric nutritional support to normal predicted levels based on age and weight 1,4. Increasing serum prealbumin concentrations are associated with increased energy intake after the acute metabolic stress has resolved 1, and can be useful to assess the adequacy of energy delivery during the post-stress recovery period. Enteral nutrition is preferred to parenteral nutrition, assuming adequate intestinal absorptive function and peristalsis are present.

However, the gut mucosal barrier can be supported with a small fraction of the total daily energy requirement 1, even if gut function is partially compromised. The visceral protein response is significantly greater and occurs earlier with enteral nutrition versus parenteral nutrition after severe injury 1. In severely injured infants and children, a soft trans-pyloric feeding tube can provide safe enteral access for early post-injury support (particularly in premature infants with delayed gastric emptying). A number of studies have demonstrated a significant benefit of enteral nutrition over parenteral nutrition in appropriately-matched, critically ill adults 7. However, a recent critical review of numerous human studies involving critically ill patients receiving TPN determined that the predominant cause of stress-associated hyperglycemia in these study populations, in addition to the intense counter-regulatory hormone and cytokine response to injury, was due to an excessive intravenous administration of dextrose 7. This observation is important because hyperglycemia can increase the risk of sepsis, wound infection, and abscess formation, all of which have been associated in greater frequency with parenteral in contrast to enteral nutritional administration in various clinical trials 7.

Iatrogenic hyperglycemia due to intravenous carbohydrate overfeeding has been associated with significant increases in mortality and morbidity due to infectious complications in a substantial number of studies involving critically ill patients receiving TPN 1,5,7.
REFERENCES


