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# NUTRITION IN THE BURN PATIENT

## Evidence Based Medicine Guideline

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#### **SUMMARY**

The hallmarks of burn management include resuscitation, debridement, and nutrition. Burn injuries create a sustained hypermetabolic response, leading to increased caloric and protein requirements. In order to provide appropriate nutritional therapy to burn patients, it is important to understand the physiological and metabolic requirements that occur with this traumatic injury.

#### RECOMMENDATIONS

- Level 1
  - Glutamine use should be avoided as it does not improve clinical outcomes and is associated with increased mortality in critically ill patients.
- Level 2
  - The Milner and Carlson equations can be used to calculate the resting energy expenditure (REE)  $\geq$ for burns of all sizes. The Curreri formula does not significantly differ from the Milner and Carlson equations when comparing mean energy expenditures.
  - Early administration of enteral feedings (within 24-48 hours of hospital admission) results in decreased ICU mortality.
  - Enteral nutrition is recommended over parenteral nutrition. However, supplemental parenteral nutrition may be considered when enteral feeding intolerance is present.

#### Level 3

- Early enteral nutrition decreases the rate of wound infection and ICU length of stay.
- Early enteral nutrition does not increase the incidence of gastrointestinal bleeding, abdominal compartment syndrome or ischemic bowel for the severely burned patient.
- Indirect calorimetry (IC) is the gold standard for calculating energy expenditures for critically ill patients, reduces mortality, and helps prevent underfeeding.
- High dose vitamin C therapy is not recommended and has been linked to worsening renal  $\triangleright$ function and fluid resuscitation.

#### INTRODUCTION

After a significant burn injury, the body triggers a significant hypermetabolic response and undergoes severe oxidative stress and systemic inflammatory response (1). It is well understood that a caloric deficit, negative protein balance, and micronutrient deficiency results in poor clinical outcomes and increased morbidity and mortality. Therefore, early appropriate nutritional supplementation is a core aspect of care for the burn patient (2).

From a physiologic standpoint, burn injury leads to a cascade of proinflammatory cytokines as well as excess release of stress hormones and catecholamine production. The initiation of these pathways results in glycolysis, lipolysis and protein catabolism. This hypermetabolic state places a burn patient at risk for severe malnutrition, infection, sepsis and death. The hypermetabolic state usually begins approximately 48 hours after the sustained LEVEL OF RECOMMENDATION DEFINITIONS

- Level 1: Usually based on Class I data or strong Class II evidence if randomized testing is inappropriate. Conversely, low quality or contradictory Class I data may be insufficient to support a Level I recommendation.
- Level 2: Reasonably justifiable based on available scientific evidence and strongly supported by expert opinion. Usually supported by Class II data or a preponderance of Class III evidence.
- Level 3: Supported by available data, but scientific evidence is lacking. Generally supported by Class III data. Useful for educational purposes and in guiding future clinical research.

DISCLAIMER: These guidelines were prepared by the Department of Surgical Education, Orlando Regional Medical Center. They are intended as a general statement regarding appropriate patient care practices based on the medical literature and clinical expertise at the time of development. They should not be considered protocol or policy nor are intended to replace clinical judgment or dictate care of individual patients. burn injury and can last for months. During this time frame, cardiac output is increased as well as oxygen consumption. IL-1 $\beta$  is a key inflammatory cytokine that is released and increases the body's resting energy expenditure (REE). Utilizing the REE for nutritional requirements in the burned patient is critical to appropriately account for changes that occur due to the hypermetabolic state (1).

#### LITERATURE REVIEW

#### Nutritional Assessment

Prior to starting enteral feeds in the critical care setting, it is important to assess for weight loss, previous nutrient intake prior to admission, disease severity, comorbid conditions, and function of the gastrointestinal (GI) tract to determine current nutritional status. In the intensive care unit (ICU), traditionally used markers such as albumin, prealbumin, transferrin and retinol binding protein are more closely related to an acute phase response and do not appropriately represent nutritional status in the ICU (3). In addition to nutritional status, nutritional risk should be assessed which accounts for factors that could affect the patient's hospital stay, including severity of the burn, age, and comorbid conditions such as inhalation injury or organ dysfunction. The catabolic state that occurs after a burn leads to protein breakdown and diminished body cell mass (4).

After sustaining a burn, patients enter a burn induced hypermetabolic state which can result in an elevation of the REE up to twice the normal (4). Burn patients may demonstrate a hyperdynamic nutritional profile with variations observed in REE as far out as 160 days following injury (5). Calculating REE in the burn patient is complex, but critical as overfeeding can increase fat storage and lead to increased time on the ventilator. In contrast, underfeeding can lead to a decrease in lean body mass, increased rates of infection and decreased wound healing (4). Underfeeding has also been shown to increase mortality (5).

Indirect calorimetry (IC) is a method for calculating REE using O<sub>2</sub> consumption and CO<sub>2</sub> production (4). IC is considered the gold standard for REE and appropriate utilization has been associated with reduced mortality in the ICU population (5). However, IC requires specialized equipment for calculating REE and is not always available in every hospital (4). There are many predictive equations that have been developed to account for the hypermetabolic changes after a burn injury. Shields et al. completed a prospective, observational study recommending the Carlson and Milner equations for the calculation of REE. The Curreri and Harris-Benedict equations also correlated closely to mean energy expenditure, however not for burns at 66-100% TBSA. When comparing results for mean energy expenditure, the Milner, Carlson, Curreri, Xie and Harris-Benedict equations were not significantly different from each other. When calculating energy expenditure, burn size is the largest factor contributing to changes in the resting energy expenditure. Based on Level II evidence, this study supports the use of the Milner and Carlson equations for the calculation of REE for the first 30 days following burn injury for all burn sizes (4). At our institution. we elect to use the Curreri formula which estimates a patient's ideal caloric intake. When comparing the Curreri formula to its counterparts, it was found to overestimate a patient's energy expenditure (6.7). In clinical practice, we find this beneficial as patients may not meet their caloric needs due to multiple trips to the operating room with intermittent cessation of enteral feeds or limited nutritional intake due to the need for vasopressors. As suggested by Rochlin et al., tube feed hours held was an independent predictor for having lower tube feeding goals. Perioperative care was the most common reason that tube feedings were held. Volume based tube feeding was observed to be superior in accounting for daily nutritional goals which supports use of the overestimation observed in the Curreri formula (8).

Equation:	
Carlson <sup>4</sup>	BMR × [0.89142 + (0.01335 × TBSA)] × BSA × 24 × AF
Curreri <sup>4</sup>	(25 x WT) + (40 x TBSA)
ounon	
Milner <sup>4</sup>	[BMR × (0.274 + 0.0079 × TBSA - 0.004 × PBD) + BMR] × 24 × BSA × AF
BMR: basal metabolic rate	
TBSA: total body surface area (%)	

TBSA: total body surface area (%) BSA: Body surface area AF: Activity factor WT: Weight in kg

#### Enteral Feeding

For the burn patient, early administration of enteral feeding, within the first 24-48 hours of hospital admission, leads to decreased ICU mortality (3,9,10). Initiation of enteral feeding within 24 hours of hospital admission also reduces ICU length of stay and decreases wound infection rate (1). Early administration decreases the incidence of bacterial translocation and leads to quicker attenuation of the body's stress response (1). Additionally, it helps prevent ileus, stress ulceration and dampens the effects of the hypermetabolic state (11). Starting enteral feeds as soon as possible after burn injury is well supported by the American Burn Association (12). During the initial phase of burn resuscitation, patients undergoing aggressive fluid resuscitation with either fluids or vasopressors may be at risk of poor gut perfusion. These patients should be considered for trophic feeding rates as opposed to advancing tube feeds to goal. Enteral feeds are not recommended when patients require vasopressor support due to the risk of intestinal ischemia. During low flow states, the initiation of enteral nutrition is suspected to increase oxygen demand above the capability of oxygen delivery, resulting in ischemia. Reports are conflicting, however, as to the point at which early alimentation and ischemic bowel are correlated (13). Once patients are hemodynamically stable, weaned from their vasopressor requirement, and have less than 200 mL of gastric output per 24-hour period, gastric feeding can be initiated at a rate of 0.5-1 mL/kg/hr (14).

Early enteral feeding is not without risk. Gastrointestinal complications after burn injury can include colonic pseudoobstruction, paralytic ileus from bowel wall edema, and abdominal compartment syndrome. Pseudo-obstruction and paralytic ileus are more likely to resolve with conservative management. However, for burns >20% TBSA, the significant amount of fluid resuscitation required can place patients at risk for intra-abdominal hypertension and abdominal compartment syndrome. Additionally, burn injury decreases splanchnic blood flow and can place patients at risk for ischemic bowel. Although these are risks associated with early enteral feeding, enteral feeding is still recommended within 24-48 hours of admission, with the knowledge of potential risks (1). Mosier et al performed a retrospective multicenter cohort study where there was no demonstrated increased incidence of gastrointestinal bleeding, abdominal compartment syndrome or ischemic bowel among patients receiving early enteral feeding after a severe burn injury (11). Patients with early enteral nutrition also had statistically significant decreased rates of wound infection as well as decreased ICU length of stay.

#### Parenteral Nutrition

Parenteral nutrition has an increased rate of infections and hepatic dysfunction. There must be careful consideration of the nutritional composition and appropriate intravascular catheter care to decrease the risk of infection. In contrast to enteral feeding, parenteral nutrition is calculated by the rate of substrate utilization instead of a predetermined energy goal. Using this calculation prevents overfeeding of intravenous nutrients. For burn patients, an infusion rate for glucose should be less than 5 mg/kg/min to prevent hyperglycemia (14). In burn patients, parenteral nutrition is not recommended alone, but may be used in combination with enteral feeding when patients have enteral feeding intolerance (15).

### Vitamin C

Vitamin C, or ascorbic acid, is a water-soluble antioxidant and reactive oxygen species scavenger. It can be effectively used to enhance tissue oxygenation and be used in the treatment of sepsis and ischemic injuries. Vitamin C has been extensively studied in animal models and was previously thought to decrease the total IV fluid requirement after a burn injury (16). Vitamin C continues to be a recommended part of burn treatment, however literature regarding the use of high-dose ascorbic acid is conflicting as the benefits may not outweigh the risks when given as a high dose infusion. Osmotic diuresis is a concern with high dose infusion and may lead to worsening renal function and fluid resuscitation without an observed improvement in patient outcome (17). High dose infusions have been demonstrated in case reports to cause secondary calcium oxalate nephropathy and subsequent renal failure (18). Tanaka et al. studied the use of high-dose vitamin C in burn patients and found retention of fluid was reduced in the ascorbic acid group and patients had a significantly decreased length of time on mechanical ventilation (19). Overall mortality rate was not reduced with the use of vitamin C. Dose-response studies have not yet determined the optimal dosing for patients with thermal injuries and there is a discrepancy in what constitutes a high dose infusion (16). Current recommendations are for vitamin C supplementation, with doses ranging from 500 to 1500 mg per day, for patients undergoing increased stress and need for wound healing (18).

#### Glutamine

Glutamine is known as a conditionally essential amino acid with low glutamine levels being associated with poor clinical outcomes during critical illness (20). Following burn injury, glutamine decreases because of its use in the liver, kidneys and gastrointestinal tract (21). The use of parenteral glutamine was previously recommended to prevent apoptosis at Peyer's patches and to decrease bacterial translocation (20). Recently, however, there has been a shift away from the use of supplemental enteral glutamine as this has been shown to increase mortality at

28 days and 6 months (22). In a blinded study by Heyland et al.,1223 ICU level patient were divided into a glutamine, glutamine plus antioxidants group, or a placebo group. There was a nonsignificant increased mortality at 28 days for the patients receiving glutamine versus those who did not (32% vs. 27%, p=0.05). There was however a significant increase in 6-month mortality with the use of glutamine. Glutamine had no demonstrated effects on organ failure or infectious complications. The mechanism in which glutamine increases mortality is unknown, but it is no longer recommended in critically ill patients for nutritional support (22). In a second multi-center double-blind trial of 1209 patients, Heyland et. al identified that enteral glutamine supplementation did not demonstrate a reduction in time to discharge alive (23). Patients with 2nd or 3rd degree burns were given 0.5 g per kg of body weight enterally or a placebo. The median time to discharge alive for the glutamine group was 40 days and the placebo group was 38 days (p=0.17).

#### <u>Zinc</u>

Zinc (Zn) is a trace element that plays an important role in immune function, wound healing and gene expression. Repletion of Zn has been included in burn guidelines for the European Society of Clinical Nutrition (ESPEN) as well as the American guidelines (ASPEN) (24). After significant burn injury, trace element deficiencies are thought to occur through exudative losses, with up to 5-10% of Zn stores being lost within 7 days after a burn. In a systematic review and meta-analysis by Kurnis et al., multiple studies looked at the effect of oral Zn supplementation after burn injuries. Based on compiled studies, there was not a significant decrease in time to wound healing with Zn supplementation (p= 0.26). However, one study by Sahib et al. demonstrated a significant decrease in positive wound cultures at day 3 (13% vs. 50%) and at discharge date (10% vs. 16.7%) with a p < 0.05. There was no observed decrease in mortality with Zn supplementation. Combining oral and parenteral Zn supplementation was demonstrated to significantly lower wound (p=0.02) and urinary tract infections (p=0.006) (25).

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