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Influence of inhalation injury on energy expenditure in severely burned children☆

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ABSTRACT

Objective: Determine the effect of inhalation injury on burn-induced hypermetabolism in children.

Design: Prospective study comparing hypermetabolism (i.e., resting energy expenditure and oxygen consumption) in burned children with and without inhalation injury during acute hospitalization.

Setting: Single pediatric burn center.

Patients: Eighty-six children (1–18 years) with ≥40% total body surface area burns were stratified to two groups: no inhalation injury and inhalation injury.

Interventions: None.

Main measurements and results: Inhalation injury was diagnosed based on bronchoscopic evaluation. At admission, PaO_2:FiO_2 ratios (an index of respiratory distress) were significantly higher in patients with no inhalation injury than in patient with inhalation injury. No differences were detected in resting energy expenditure or percent of the predicted basal metabolic rate between groups. Additionally, oxygen consumption did not significantly differ between groups.

Conclusions: Inhalation injury does not augment the burn-induced hypermetabolic stress response in children, as reflected by resting energy expenditure and oxygen consumption.

1. Introduction

The hypermetabolic stress response that occurs following thermal injury is typified by a hyperdynamic state that is associated with elevations in cardiac output [1]. The release of catecholamines, cortisol, and glucagon results in a severe catabolic reaction marked by increased energy expenditure [2]. Protein is mobilized and used as a major substrate for energy, resulting in muscle wasting and loss of lean body mass [3]. As a consequence, immune function is impaired, the incidence of sepsis and pneumonia is increased, wound healing is prolonged, and survival is diminished [4].

Inhalation injury in the presence of a severe burn is a major predictor of mortality and increased morbidity [5]. Inhalation injury alone increases oxygen consumption (VO_2) within 2 h of

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the injury, leading to increases in metabolic burden [6]. Exposure to smoke or other toxic inhalants causes an acute inflammatory response at the injured site. The liberation of reactive oxygen species, lipid peroxides, and neutrophil proteases increases vascular permeability, which results in airway edema. Tissue oxygen demand increases, leading to elevations in VO_2, and work of breathing becomes more labored [7,8].

Past work in experimental animals, particularly sheep, demonstrates that the degree of inhalation injury occurring alongside the burn correlates with the severity of lung injury, which is confined to the trachea and large airways [9]. Inhalation injury is known to increase resuscitation fluid estimates, metabolic demands, and VO_2 [10–14]. Wilmore et al. showed that percent total body surface area (TBSA) burned correlates with metabolic rate [2]. At one week after burn, children with >40% TBSA burned had metabolic rates of 178% of their predicted basal according to the Harris Benedict equation. Fully healed, the same children had 153% of their predicted basal metabolic rate [3]. Although it is well established that burn increases metabolic rate, no studies have clarified whether additional changes in energy expenditure occur when thermal injury is complicated with inhalation injury.

Indirect calorimetry provides an easy, non-invasive method for measuring caloric energy utilisation at the bedside. Saffle and colleagues have shown that resting energy expenditure (REE) measures obtained through indirect calorimetry are valuable in monitoring the nutritional status in burn patients [15] and that this approach is more precise than traditional nutritional calculations, such as the Curreri formula [16].

This study was undertaken to determine the effect of inhalation injury on the metabolic rate in the severely burned pediatric population. We compared energy utilisation as assessed using indirect calorimetry between burned children with and without inhalation injury throughout acute care hospitalization.

2. Materials and methods

2.1. Patients

This study was performed under a protocol approved by the Institutional Review Board at the University of Texas Medical Branch. Eighty-six severely burned children admitted to Shriners Hospitals for Children (Galveston, TX) during 1998–2002 were enrolled into this prospective study. Children under 18 years old with ≥40% TBSA burns were eligible for study participation. Exclusion criteria included anoxic brain injury, severe psychological disorders, quadriplegia, or severe behavioral or cognitive disorders. Before enrollment, each patient's guardian provided written informed consent, with assent from patients aged ≥7 years.

All study subjects received identical treatment by the same surgical team. Standard treatment included early excision of the burn wound, systemic antibiotic therapy, and continuous enteral feeding (Age in years: 0–1 Galveston Infant 2100 kcal/m^2 + 1000 kcal/m^2 burn; 1–11 Galveston Revised 1800 kcal/m^2 + 1300 kcal/m^2 burn; 12–16 Galveston Adolescent 1500 kcal/m^2 + 1500 kcal/m^2 burn) [17].

2.2. Inhalation injury diagnosis

Study subjects were stratified into two groups depending on their diagnosis: no inhalation injury (No Inh) and inhalation injury (Inh). Inhalation injury was documented by bronchoscopy within 24 h of admission. Inhalation injury was diagnosed based on the presence of infraglottic soot. Alternatively, it was diagnosed based on early inflammatory alterations in the tracheal mucosa such as hyperemia, edema, superficial mucosal sloughing, and ulcerations. Treatment of inhalation injury consisted of bronchial hygiene therapy, heparin (5000 U) and N-acetylcysteine (3 ml of a 20% solution) aerosolized every 4 h with albuterol dosed every 4 h [18], chest physiotherapy, and pressure control mechanical ventilation if indicated.

2.3. Indirect calorimetry

REE measurements were obtained within 1 week of admission and during all subsequent weeks throughout the acute hospitalization. A second metabolic study was conducted at the height of the hypermetabolic state (approximately burn day 10) to adjust for the ebb and flow stages of the hypermetabolic response. REE measurements were obtained from subjects while they slept (12 am–5 am) using a SensorMedics Vmax 29 metabolic cart (Yorba Linda, CA). These measurements coincided with continuous enteral feeding administered at a rate of 1500 kcal/m^2 TBSA + 1500 kcal/m^2 BSA burned. For testing, patients were in a supine position under a ventilated hood. Measurements were obtained at 30 °C, the standard temperature of rooms in our burn ICU. REE was calculated based on CO_2 production and VO_2 using an equation described by Weir [19]. REE measures were used to determine the metabolism levels of patients and to guide their nutrition. For surviving study subjects, the REE measurement taken at discharge was used to assess hypermetabolism when the burn wounds were 95% healed. The primary outcome measure was REE (kcal/m^2 day), as measured by indirect calorimetry. The secondary outcome measure was VO_2 (ml/min).

2.4. Statistics

Demographic and metabolic data were analyzed using a one-way analysis of variance (ANOVA) and a two-way ANOVA, respectively. Chi square analysis was used to compare the incidence of pneumonia among groups. Data are reported as mean ± SEM. Significance was set at $p < 0.05$.

3. Results

3.1. Demographics

Of the 86 study subjects, 44 had inhalation injury and 42 did not (Table 1). Mean ages were comparable among the survivor groups.