

# Chapter 18. Nutritional support

## I. RECOMMENDATION

*A. Standards.* There are insufficient data to support a treatment standard for this topic.

*B. Guidelines.* There are insufficient data to support a treatment guideline for this topic.

*C. Options.* Replace 130–160% of resting metabolism expenditure after traumatic brain injury (TBI) in pediatric patients. Weight-specific resting metabolic expenditure guidelines can be found in Talbot's tables (1).

Based on the adult guidelines, nutritional support should begin by 72 hrs with full replacement by 7 days.

*D. Indications from Adult Guidelines.* At a guideline level, replace 140% of resting metabolism expenditure in nonparalyzed patients and 100% of resting metabolism expenditure in paralyzed patients, by using enteral or parenteral formulas containing  $\geq 15\%$  of calories as protein by day 7 after injury (2).

At an option level, jejunal feeding by gastrojejunostomy is preferred due to ease of use and avoidance of gastric intolerance.

## II. OVERVIEW

The nutritional status of pediatric TBI patients may be critical to the recovery process. The question remains unanswered whether nutritional formulations; glucose metabolism; the amount, type, method, or timing of feeding; or any other specific nutritional intervention influences outcome of pediatric TBI patients. There are only two studies (one class II and one class III evidence) that adequately addressed metabolism in children with a brain injury, and no studies that looked at differences in morbidity or mortality rates (3, 4).

## III. PROCESS

We searched Medline and Healthstar from 1966 to 2001 by using the search strategy for this question (see Appendix A) and supplemented the results with literature recommended by peers or identi-

fied from reference lists. Of 35 potentially relevant studies, two were used as evidence for this question (Table 1).

## IV. SCIENTIFIC FOUNDATION

Two studies (one class II and one class III) addressed hypermetabolism and nutritional support in pediatric TBI (3, 4). Phillips et al. (3) examined energy expenditure, nitrogen excretion, and serum protein levels in pediatric TBI patients with a Glasgow Coma Scale of 3–8. The study included eight adolescents (age 11–17 yrs) and four children (age 2–5 yrs). Eleven sustained a blunt head injury, and one had a penetrating head injury by gunshot. All patients were initially intubated and mechanically ventilated for 5–14 days. No patients received corticosteroids. Four patients received neuromuscular blockade. Seven patients were started on total parental nutrition on days 2–6 after injury, and five were started on enteral nutrition on days 3–12 after injury. The study demonstrated a significant elevation in the mean energy expenditure of approximately 130% above predicted levels for the group as a whole (Harris-Benedict equation). Seventy percent achieved nitrogen balance by 4–14 days. The mean urinary nitrogen excretion was 307  $\text{mg}\cdot\text{kg}^{-1}\cdot\text{day}^{-1}$  for the adolescents (mean nitrogen balance of  $-13.6$ ) and 160  $\text{mg}\cdot\text{kg}^{-1}\cdot\text{day}^{-1}$  for the children (mean nitrogen balance of  $-4.1$ ). Mean serum albumin levels decreased slightly (from 2.9  $\text{mg}/\text{dL}$  down to 2.4  $\text{mg}/\text{dL}$ ) whereas total protein levels increased (from 5.4  $\text{mg}/\text{dL}$  to 6.0  $\text{mg}/\text{dL}$ ). Weight loss ranged from 2 to 26 lb during the 2-wk period showing, despite aggressive nutritional support, a significant decrease in body weight of 9% for adolescents and 4% for children. The effect of neuromuscular blockade on metabolism was not addressed.

Moore et al. (4) measured the metabolic profiles of 20 severe TBI patients (Glasgow Coma Scale  $< 7$ ). Although they studied both adult and pediatric patients (13 and seven, respectively), the data spe-

cific to the pediatric patients were sufficiently reported to allow inclusion as class III evidence. All patients were mechanically ventilated. Two of the patients received corticosteroids. Neuromuscular blockade was not addressed. In all patients, feeding was initiated within 48 hrs of admission to the trauma units. It was difficult to ascertain which patients received total parental nutrition vs. enteral nutrition. In the pediatric group (age 3–16 yrs), the study demonstrated an average of 180% of predicted for oxygen consumption and 173% predicted of resting energy expenditure. The average respiratory quotient was 0.68 for both the whole group and the pediatric subgroup.

Although neither of these studies addressed the effect of nutritional support on outcome, the data demonstrate a sizable increase in energy expenditure. The increase in expenditure was highly variable among patients. These studies suggest an increased need for nutritional support in pediatric TBI patients. Insufficient data prevent thorough comparison between the metabolism in adults and children, but the pediatric findings are similar to those well established in the adult literature.

## Key Elements from the Adult Guidelines Relevant to Pediatric TBI

Hypermetabolism after TBI has been well documented in the adult literature (2–20). At least 12 class I studies have been completed. Nine class I studies examined the effect of amount of feeding, type of feeding, route of feeding, and corticosteroids on nitrogen balance and serum biochemistries. One class I study examined the effect of insulin growth factor I on the catabolic state and on outcome (21). Two class I studies examined the relation between extent of nutritional replacement and outcome (22, 23).

Data from investigators measuring metabolic expenditure in rested comatose patients with isolated TBI showed a mean increase of approximately 140% of the

Table 1. Evidence table

Reference	Description of Study	Data Class	Conclusion
Phillips et al. (3), 1987	Energy expenditure, nitrogen excretion, and serum protein levels in pediatric TBI patients with a GCS of 3–8. Total n = 12 (eight adolescents age 11–17 yrs and four children age 2–5 yrs). Eleven blunt head injuries and one GSW. All patients were initially intubated and mechanically ventilated in the emergency department and continued for 5–14 days. No patients received steroids. Four patients were paralyzed with Pavulon. Seven patients were started on TPN on days 2–6 after injury, and five were started on EN on days 3–12 after injury.	II	The mean energy expenditure was approximately 130% above predicted for the whole group. Seventy percent achieved nitrogen balance by 4–14 days. The mean urinary nitrogen excretion was 307 mg <sub>db1</sub> ·kg <sup>-1</sup> ·day <sup>-1</sup> for the adolescents (mean nitrogen balance of -13.6) and 160 mg·kg <sup>-1</sup> ·day <sup>-1</sup> for the children (mean nitrogen balance of -4.1). Mean albumin levels decreased slightly (2.9 mg/dL down to 2.4 mg/dL), whereas total protein levels increased (from 5.4 mg/dL to 6.0 mg/dL). Weight loss ranged from 2 to 26 lb during the 2-wk period showing, despite aggressive nutritional support, a significant decrease in body weight of 9% for adolescents and 4%.
Moore et al. (4), 1989	The metabolic profiles of severe closed TBI patients (GCS <7) were measured. Mixed adult and pediatric, total n = 20 (13 adults, seven children). All patients were mechanically ventilated. Two received steroids. Paralysis was not addressed. In all patients, feeding was initiated within 48 hrs of admission to the trauma units. No distinction between TPN vs. EN.	III	In the pediatric group (age 3–16 yrs), the study demonstrated an average of 180% of predicted for oxygen consumption and 173% predicted of resting energy expenditure. The average respiratory quotient was 0.68 for both the whole group and the pediatric subgroup.

TBI, traumatic brain injury; GCS, Glasgow Coma Scale; GSW, gunshot wound; TPN, total parenteral nutrition; EN, enteral nutrition.

expected metabolic expenditure, with variations from 120 to 250% of that expected. In TBI patients, neuromuscular blockade or barbiturate coma decreased metabolic expenditure from a mean of 160% of the expected to 100–120%. These findings suggest that a major part of the increased metabolic expenditure is related to muscle tone. Even with neuromuscular blockade, energy expenditure remained elevated by 20–30% in some patients (7). In the first 2 wks after injury, energy expenditure seems to increase regardless of neurologic course.

Class I data suggest that when TBI patients are not fed within the first week, mortality rate is increased. Data in critically ill patients without TBI show that a 30% weight loss was associated with an increased mortality rate. After severe TBI, both energy requirements and nitrogen excretion markedly increase. Fasting patients with severe TBI continue to lose 14–25 g N/day (11). Data show that starved TBI patients lose sufficient nitrogen to reduce weight by 15% per week. Given adequate nutritional support, the contribution of protein to consumed calories after TBI increases to levels as high as 30% (24). Class II data show that 100–140% replacement of resting metabolism expenditure with 15–20% nitrogen calories reduces nitrogen loss. The data strongly support full nutritional replacement by the end of the first week after injury.

The optimal method of feeding has not been established. Similarly, it has not been shown that earlier feeding (full feeding before 7 days) improves outcome. Studies showed that, with nearly equivalent quantities of feeding, the mode of administration (total parental nutrition or enteral nutrition) had no effect on neurologic outcome, despite other potential advantages of enteral nutrition (decreased risk of hyperglycemia, lower costs, lower risk of infection). For enteral nutrition, jejunal feeding was better tolerated than gastric feeding (25, 26).

Based on the level of nitrogen wasting documented in TBI patients and the nitrogen sparing effect of feeding, it is a guideline that full nutritional replacement be instituted in the adult TBI patient by day 7. It is suggested that enteral nutrition begin no later than 72 hrs.

Finally, studies of glucose homeostasis suggest that serum glucose control may be critical to limiting secondary neurologic damage. In animal models, hyperglycemia has been shown to worsen ischemic brain injury (27–29). Hyperglycemia was associated with a worse outcome in critically ill patients (30, 31), and tight control of glucose levels (<110) in nondiabetic patients was associated with improved outcome (32). In two studies of severe head injury, hyperglycemia also was associated with worse outcome (33, 34). These studies sug-

gest that serum glucose levels should be tightly controlled in TBI patients.

## V. SUMMARY

Due to the limited data that exist on the nutritional requirements of pediatric TBI patients, recommendations can only be made at the option level. Of the two studies addressing pediatric patients, both showed a significant increase in the metabolic rate associated with TBI. These findings are similar to those reported in the adult literature. Without further data, the adult guidelines, adjusted for weight, should be considered when providing nutritional support to pediatric patients with TBI.

## VI. KEY ISSUES FOR FUTURE INVESTIGATION

- Little research has been conducted on nutritional support and its influence on outcome of pediatric TBI. These patients are likely to be highly dependent on precise nutritional interventions tailored to age, weight, body surface area, and other factors.
- Recent studies of glucose homeostasis suggest that glucose control may be critical to secondary damage and neurologic outcome. This topic needs investigation in pediatric patients.
- The issue of hypermetabolism in pediatric TBI needs further confirmation

**W**ithout further data, the adult guidelines, adjusted for weight, should be considered when providing nutritional support to pediatric patients with traumatic brain injury.

and clarification. The effects of timing, type, quality, and methodology of nutritional support on the outcome of pediatric patients after TBI need to be investigated.

- Special issues such as vitamin and mineral replacement and other nutritional interventions need to be explored. Authors need to stratify different age groups (infants, toddlers, children, adolescents) in these studies to further delineate developmental differences in nutritional requirements. Without further data, the adult guidelines, adjusted for weight, should be considered when providing nutritional support to pediatric patients with traumatic brain injury.

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See APPENDIX on Next Page

# APPENDIX: LITERATURE SEARCH STRATEGIES

## SEARCHED MEDLINE AND HEALTHSTAR FROM 1966 TO 2001

### Chapter 18. Nutrition

1. exp craniocerebral trauma/
2. head injur\$.tw.
3. brain injur\$.tw.
4. 1 or 2 or 3
5. exp parenteral nutrition/ or "parenteral nutrition".mp.
6. nutritional support/ or "nutritional support".mp.
7. 5 or 6
8. 4 and 7
9. limit 8 to (newborn infant <birth to 1 month> or infant <1 to 23 months> or preschool child <2 to 5 years> or child <6 to 12 years> or adolescence <13 to 18 years>)