# Nutritional Support After Spinal Cord Injury

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**KEY WORDS:** Indirect calorimetry, Nitrogen wasting, Nutritional support

*Neurosurgery 72:255–259, 2013* DOI: 10.1227/NEU.0b013e31827728d9 www.neurosurgery-online.com

## RECOMMENDATIONS

### Level II

- Indirect calorimetry as the best means to determine the caloric needs of spinal cord injury patients is recommended.

### Level III

- Nutritional support of spinal cord injury (SCI) patients is recommended as soon as feasible. It appears that early enteral nutrition (initiated within 72 hours) is safe, but has not been shown to affect neurological outcome, the length of stay, or the incidence of complications in patients with acute SCI.

## RATIONALE

Hypermetabolism, an accelerated catabolic rate, and rampant nitrogen losses are consistent sequelae to major trauma, particularly acute traumatic brain injury and acute SCI.[1-7] A well-documented hypermetabolic, catabolic injury cascade is initiated immediately after central nervous system injury, which results in depletion of whole body energy stores, loss of lean muscle mass, reduced protein synthesis, and ultimately in loss of gastrointestinal mucosal integrity and compromise of immune competence.[2,3,5-9] Severely injured brain and spinal cord injury patients, therefore, are at risk for prolonged nitrogen losses and advanced malnutrition within 2 to 3 weeks following injury with resultant increased susceptibility for infection, impaired wound healing, and difficulty weaning from mechanical ventilation.[3-7,10] These factors added to the inherent immobility, denervation, and muscle atrophy associated with spinal cord injury provide the rationale for nutritional support of spinal cord injured patients following trauma. The guidelines author group of the Joint Section on Disorders of the Spine and Peripheral Nerves of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons provided a medical evidence-based guideline on this topic in 2002.[11] The current review is undertaken to update the medical evidence on this important issue since that original publication.

**ABBREVIATIONS: REE,** resting energy expenditure; **SCI,** spinal cord injury; **TBI,** traumatic brain injury

## SEARCH CRITERIA

A National Library of Medicine (PubMed) computerized literature search from 1966 to 2011 was undertaken using Medical Subject Headings in combination with "spinal cord injury": nutrition (138 citations) and nutritional support (73 citations). Non-English language and duplicate citations were deleted. Titles and abstracts of the remaining publications were reviewed. A focused search on the specific issue of nutrition and human patients with acute spinal cord injuries identified 16 citations. Relevant manuscripts and reviews describing nutritional support of head-injured patients and several reports describing the nutritional status of chronic SCI patients are included in the bibliography. These efforts identified 7 Class III medical evidence studies, which describe metabolism, nitrogen wasting and the effect of feeding on nitrogen balance, and serum biochemistries in patients after acute SCI. Four of the 7 citations offer Class II medical evidence on indirect calorimetry to assess energy expenditure after SCI. All 7 are summarized in Evidentiary Table format (Table). There were no studies that examined the effects of nutritional support on neurological outcome following acute SCI.

## SCIENTIFIC FOUNDATION

Hypermetabolism, catabolism and accelerated nitrogen losses are well-recognized complications that follow traumatic injury.[3,6,7,12] They have been identified and studied in human patients who have sustained traumatic brain and spinal cord injuries. A number of publications have described the increased energy requirements and nitrogen losses of patients following acute head injury.[1,3,5,7,9,12-15] Fewer studies have focused on hypermetabolism, catabolism, and nitrogen losses following acute SCI.[4,7,10,16,17] While there are metabolic similarities between isolated traumatic brain injury and severely isolated SCI, it appears there may be important biological differences between the 2 central nervous system (CNS) injury types that have bearing on supplemental nutritional therapy.[4-8,16,17]

Severe head injury is associated with a resting energy expenditure (REE) of approximately 140% of predicted normal basal energy expenditure[3,5,7,12-15] Indirect calorimetry is the most widely used reliable means to determine individual energy requirements in hospitalized patients after traumatic injury.[3,5-7] It requires the use of a portable metabolic cart and employs a technique that measures respiratory gas exchange and the rate of oxygen utilization in a given patient. It provides an estimate of energy expenditure by the patient by determining the known caloric yield from 1 liter of oxygen based on differences in oxygen consumption and carbon dioxide production. It is performed at the bedside in the intensive care unit in severely injured patients. Metabolic expenditure is expressed as a percent of normal basal energy expenditure at rest (predicted). Indirect calorimetry is typically performed once daily for the first several days post injury and periodically thereafter.[3,5-7] The Harris-Benedict equation, with activity and stress of injury variables, has been shown to predict energy expenditure after traumatic brain injury (TBI) with reasonable accuracy without indirect calorimetry.[3,6,7,16,17]

Nutritional support of head-injured patients is typically begun within days of admission and is guided by the metabolic information provided by indirect calorimetry and by predicted energy expenditure values derived by equation. Hypermetabolism, accelerated catabolism, and excessive nitrogen losses continue for at least 2 weeks after injury.[2,3,5,7,12,13] The exact duration of this response to injury is unknown, may vary among similar patients, and can be affected by other traumatic injuries, pancreatitis, infection, or sepsis.[3,5-7,18] Nutritional support in this setting is designed to provide nitrogen-rich, high-energy supplemental fuel to blunt excess catabolism and preserve energy stores, muscle mass, gastrointestinal integrity, and immune competence.[3,5-7,10] Nitrogen balance is difficult and often impossible to achieve, particularly within the first week of injury.[1,3,4,16,17] Matching nutritional replacement with caloric needs has therefore become the primary goal of nutritional therapy.

The extent of neuronal connectivity and the neurogenic stimuli (muscle tone) to the musculoskeletal system appears important to the level of metabolic expenditure after CNS injury.[4-7,16,17,19-23] Agitated, combative head-injured patients, for example, can have REEs as high as 200% of expected basal energy expenditure levels.[3,5,7,12] Conversely, pharmacological paralysis of head-injured patients has been associated with reductions in resting energy expenditure by 20% to 30%.[3,5,7,12] Patients who have sustained isolated acute SCI often have increased metabolic expenditure compared to normative energy expenditure levels.[4-8,16,17] However, because of the paralysis and flaccidity associated with acute SCI, measured resting energy expenditure (REE) values in these patients are considerably lower than those predicted by the Harris-Benedict equation based on age, sex, body surface area, activity, and injury severity.[6,15-17,21] Patients with the greatest neurological deficits and the least muscle tone after SCI (high cervical level quadriplegic patients) have lower measured REE values than those found in patients with incomplete spinal injuries or lower spinal cord injuries (thoracic level paraplegic patients).[1,4,6,7,16,17] Kaufman et al,[4] in 1985, described their experience with 8 acute SCI patients managed at the University of Texas. They noted accelerated nitrogen losses and ongoing negative nitrogen balance greater than expected. Differences in initial and follow-up nutritional assessments revealed deterioration in nutritional status during the 2-week period of observation, partly due to inadequate supply of protein and calories. Infective complications and prolonged respiratory support were common. The authors concluded that muscle atrophy might play an important role in the accelerated nitrogen losses they identified in patients with paralysis due to complete spinal cord injury, and that improved nutritional support might reduce medical complications following acute SCI. In 1989, Kolpek et al[24] compared urinary urea nitrogen excretion and measured energy expenditure between 7 head trauma and 7 spinal cord injury patients. They found that the difference in urinary urea nitrogen excretion between the 2 groups of patients was equivocal. When they compared the measured energy expenditure to the predicted energy expenditure, they found the ratio was 0.56 for SCI patients and 1.4 for head injury patients.

Young, Ott, and Rapp[7] reported 4 quadriplegic acute SCI patients that they assessed with indirect calorimetry. They found that indirect calorimetry provided more accurate REE values for their patients compared to Harris-Benedict equation estimates, even Harris-Benedict equation estimates without incorporating injury and activity factors. They too noted marked daily nitrogen losses and negative nitrogen balance in their SCI patients. They concluded that equation estimates of REE of SCI patients overestimate metabolic expenditure and emphasized the importance of indirect calorimetry in predicting energy expenditure following acute SCI.

Kearns et al[16] prospectively assessed and provided nutritional support to 10 acute SCI patients that they managed and monitored for 4 weeks. Their 1992 report documents the use of indirect calorimetry to determine REE and provide matched caloric supplementation. All patients had isolated SCI without associated head injury or other organ system trauma. Initial measured resting energy expenditures were 10% below predicted REE levels. All patients experienced exaggerated nitrogen and 3-methylhistidine losses indicating excessive lean body mass and muscle loss. A 10% decrease in body weight accompanied these losses despite caloric replacement matched to or exceeding measured REE values for

**TABLE. Evidentiary Table: Nutritional Support**[a]

| Citation | Description of Study | Evidence Class | Conclusions |
|---|---|---|---|
| Dvorak et al,[27] *Spine*, 2004 | Randomized to early (<72 hrs) or late (>120) enteral feeding in 17 acute cervical SCI patients. | III | No differences in the incidence of infection, nutritional status, feeding complications, number of ventilator hours, or length of stay. |
| Rowan et al,[26] *Injury*, 2003 | Retrospective review of 33 patients with acute SCI, 27 received early enteral feeding (0.5-4.8 days). | III | No major complications seen with early enteral feeding. |
| Cruse JM et al,[8] *J Spinal Cord Medicine*, 2000 | Comparison of nutritional, immune, endocrine status in 15 acute SCI patients vs 16 matched controls. | III (II for indirect calorimetry) | SCI patients have hormonal changes, poor nutritional status, and decreased immune function compared to controls. |
| Rodriguez DJ et al,[6] *Spinal Cord*, 1997 | Prospective assessment and treatment of 12 acute SCI patients. | III (II for indirect calorimetry) | REE less than predicted, marked "obligatory" nitrogen losses due to flaccidity and atrophy of denervated muscle after SCI. |
| Kearns PJ et al,[16] *J Parenteral Enteral Nutrition*, 1992 | Prospective assessment of 10 acute SCI patients over 4-week period of observation. | III (II for indirect calorimetry) | Exaggerated nitrogen and 3MeH excretion marked weight loss. Lower REE than predicted after SCI. |
| Young B et al,[15] *Critical Care Clinics*, 1987 | Four acute SCI patients assessed via indirect calorimetry. | III (II for indirect calorimetry) | Indirect calorimetry best means to determine energy expenditure after acute SCI. |
| Kaufman HH et al,[4] *Neurosurgery*, 1985 | Assessment of nutritional status of 8 SCI patients over 2-week period of observation. | III | Deterioration in nutritional status despite attempted treatment. Marked nitrogen losses. Increased infectious and respiratory complications. |

[a]REE, resting energy expenditure; SCI, spinal cord injury.

each patient. The specifics of nutrition administration (mix and route of delivery) were not presented. The authors noted an increase in REE over time in part due to reductions in body weight and in part due to return of muscle tone. The authors concluded that acute isolated SCI is associated with lower REE values compared to predicted values. Acute SCI patients have exaggerated nitrogen and 3-methylhistidine losses due to atrophy of denervated muscle. They attributed the reduced metabolic activity seen in these patients to the flaccidity of denervated musculature after severe SCI and noted that as muscle loss and weight reductions progress, REE increases, particularly if recovery of motor function and/or return of muscle tone occurs.

Rodriguez et al[6] studied the metabolic response to SCI in 12 acute trauma patients. Assessment and nutritional support were instituted immediately after injury and continued for 4 weeks post injury. Harris-Benedict estimations of energy expenditure were compared to values obtained from indirect calorimetry in each patient. All patients had accelerated nitrogen losses and negative nitrogen balance. Eleven of 12 patients had negative nitrogen balance for the entire 4 weeks of therapy despite matched caloric replacement. The single patient in whom nitrogen balance was realized had an incomplete SCI. The Harris-Benedict equation with an activity factor of 1.2 and a stress/injury factor of 1.6 consistently overestimated energy expenditure in these 12 patients and would have resulted in excessive feeding. The authors concluded that large nitrogen losses after severe SCI are "obligatory" as a result of atrophy and wasting of denervated musculature below the level of injury. Patients with complete traumatic myelopathy had greater obligatory nitrogen losses than patients with incomplete spinal cord injuries. They recommended that indirect calorimetry be used as the energy expenditure assessment method after SCI, particularly in the early post-injury period. If the Harris-Benedict equation is used in these patients in this setting, they recommend that the activity factor should be eliminated and the stress/injury factor of the equation should be reduced.

Three different author groups[6,15,16] provide consistent medical evidence that equation estimates of REE for SCI patients overestimate energy expenditure. All provide convincing comparative evidence that indirect calorimetry is the most accurate means to assess energy expenditure in SCI patients. [6,15,16] For these reasons and because the differences are substantial and the medical evidence is consistently positive in all 3 published studies, indirect calorimetry as the most reliable means to assess REE in SCI patients is upgraded to Class II medical evidence.

Cruse et al[10] examined the neurological, immune, endocrine, and nutritional status of 15 male SCI patients and compared them to 16 healthy age-matched control subjects. The timing of assessment in relation to SCI for each patient was not specified. Their report described decreased natural and adaptive immune responses in the SCI patient population beginning within 2 weeks of injury that reached a nadir 3 months after injury. They noted increased ACTH and plasma cortisol levels; decreased zinc, albumin, and prealbumin serum levels; surface marker changes in both lymphocytes and granuloycytes; and decreased adhesion molecule binding ability after SCI compared to healthy control patients. They concluded that patients with severe acute SCI have decreased immune function, impaired nutritional status and a decreased number of adhesion molecules, all of which occur within weeks after acute injury. The authors note that these hormonal alterations, nutritional deficiencies, and changes in immune function may increase susceptibility to infection and may contribute to delayed wound healing.

The change in energy expenditure identified in patients following acute SCI appears to persist long after the initial injury and recovery phase.[6,19-23,25] Several investigators have noted long-standing reductions in REE in spinal cord injury patients, reductions that correlate to the degree of neurological injury and the extent of lean body mass loss after paralysis.[6,19-23,25] Cox et al[21] measured energy expenditure in stable nonacute SCI patients in the rehabilitation setting. They reported that quadriplegic patients required 22.7 kcal/kg/day compared to 27.9 kcal/kg/day for paraplegic patients they studied. Most investigators conclude that equation methods to estimate energy expenditure in SCI patients are inaccurate, both in the acute and chronic settings.[7,17,22,23,25]

The literature on nutritional support for head injury patients supports using the enteral route for nutritional supplementation if the gut is functional.[3,5-7,12,13,17] This general policy appears to have been followed by investigators of nutritional support for acute SCI patients.[4,6,7,16] The potential benefits of enteral feeding over parenteral delivery include maintenance of gut integrity and function, reduced expense, lower risk of infection and avoidance of intravenous catheter-related complications.[3,5-7,12,13,17] Naso-duodenal or nasojejunal feeding tubes usually allow full caloric, high-nitrogen, high-volume feeding within days of injury. In patients with bowel injury, mechanical bowel obstruction, or prolonged ileus, it is recommended that parenteral nutrition be initiated until the bowel recovers and conversion to enteral nutrition can be accomplished.[3,5-7,13]

Since the publication of the original guidelines on this topic, there have been 2 studies published comparing early to late enteral feeding in patients with acute SCI. Rowan et al[26] published a retrospective study of a group of 33 patients who received enteral feeding at a median of 2 days (range 0.5-4.8 days) following admission. The most common reason for interruption of enteral feeds was high gastric aspirates, which occurred in 67% of patients. Two patients developed ileus, requiring conversion from nasogastric to nasojejunal feeding tubes. Dvorak et al[27] prospectively randomized 17 acute SCI patients to either early (less than 72 hours) or late (greater than 120 hours) enteral feeding. While they found no difference in the incidence of infection, nutritional status, feeding complications, number of ventilator hours, or length of stay between the 2 groups, the numbers in each treatment group were too few to draw meaningful conclusions.

There has been no report assessing the mix or composition of nutritional supplementation for SCI patients. The literature on nutritional support for head injury patients suggests beginning with a high nitrogen enteral or parenteral solution containing at least 15% of calories as protein, no greater than 15% glucose/dextrose, a minimum of 4% of total energy needs as essential fatty acids, and the addition of vitamins, essential elements, and trace minerals.[3,5-7,9,14,15]

There has been no study published that has examined the effect of nutritional support on neurological outcome following acute SCI.

## SUMMARY

Alterations in metabolism occur after acute SCI, but the marked hypermetabolic response seen after acute traumatic brain injury appears to be blunted in SCI patients by the flaccidity of denervated musculature after spinal cord transection/injury. As a result, REE is lower than predicted after acute SCI. Equation estimates of REE in these patients have proven to be inaccurate. Comparative Class II medical evidence supports the use of indirect calorimetry as the recommended technique to assess energy expenditure in both the acute and chronic settings among patients with SCI.

Protein catabolism does occur after acute, severe SCI, and marked losses in lean body mass due to muscle atrophy result in huge nitrogen losses, prolonged negative nitrogen balance, and rapid weight loss. Nutritional support of the SCI patient to meet caloric and nitrogen needs, not to achieve nitrogen balance, is safe and may reduce the deleterious effects of the catabolic, nitrogen wasting process that occurs after acute spinal cord injury. It appears that early enteral nutrition (initiated within 72 hours) is safe, but has not been shown to affect neurological outcome, the length of stay, or the incidence of complications in patients with acute SCI.

## KEY ISSUES FOR FUTURE INVESTIGATION

An assessment of the timing, route of administration, and the composition of nutritional therapy on outcome, both neurological and medical, should be performed. This could be accomplished with a multicenter case control study.

### Disclosure

The authors have no personal financial or institutional interest in any of the drugs, materials, or devices described in this article.

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