Management of nutrition support in adults with moderate-severe traumatic brain injury in the acute care setting

Grayson LeDuc

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Management of Nutrition Support in Adults with Moderate-Severe Traumatic Brain Injury in the Acute Care Setting

Grayson LeDuc, RDN, LD

Seeking Masters of Family and Consumer Sciences
Emphasis in Dietetics

April 14, 2020
TABLE OF CONTENTS

STATEMENT OF PURPOSE - 3 -

METHODOLOGY - 4 -

ABBREVIATIONS - 5 -

REVIEW OF LITERATURE - 6 -

Introduction - 6 -

Nutritional Needs and TBI - 9 -
  Energy Needs - 9 -
  Protein Needs - 13 -
  Dysphagia - 13 -

Nutrition Support and TBI - 14 -
  Enteral Nutrition - 15 -
  Parenteral Nutrition - 18 -
  Transition to Oral Diet - 19 -
  Long-Term Nutrition Support - 20 -

Immunonutrition and TBI - 21 -
  Omega-3 Fatty Acids - 21 -
  Zinc - 23 -
  Glutamine - 24 -

Conclusion - 24 -

BIBLIOGRAPHY - 26 -

APPENDIX A - 39 -

Case Study - 39 -
STATEMENT OF PURPOSE

The purpose of this creative component project is to conduct a literature review examining the nutritional needs in adults admitted to the acute-care setting with moderate to severe traumatic brain injuries. Traumatic brain injury (TBI) is a public health concern as it impacts thousands of individuals each year and frequently results in disability or death. Nutrition plays an integral role in helping to manage the secondary injuries that may happen in the acute phase in individuals who suffer from a head trauma.

My current position is that of a clinical dietitian serving multiple medical neurology and neurosurgical wards as well as a neurosciences intensive care unit. In this position, I work with many patients who have experienced a TBI of varying degrees. As I have come across patients with severe TBIs, I have learned how these patients are reliant on nutrition support and realized that nutrition recommendations in regard to this population are an area that I wanted to learn more. The goal of this creative component is to help increase my knowledge on the Medical Nutrition Therapy management of patients with severe TBI, so that I can better serve this population.
METHODOLOGY

The articles used for this review of literature were gathered online from Pubmed, Ovid Medline and Embase. Pertinent search terms were used to locate peer-reviewed articles through these platforms include brain injuries, traumatic brain injuries, nutrition therapy, nutrition support, immunonutrition and energy intake. After initial search for immunonutrition; omega-3 fatty acids, glutamine, and zinc were selected as key nutrients and were included in the literature search. A search including all of these terms resulted in a total of 32 articles. Some limitations observed when conducting this review of literature included small sample sizes, lack of human studies, and lack of updated research (in the last 10 years). In the initial search, articles were narrowed to within the last 10 years however, this was expanded to include articles of any date due to the lack of current research. The articles were evaluated using the Academy of Nutrition and Dietetics’ (AND) Evidence Analysis Library (EAL) worksheet.

Table 1. Inclusion and Exclusion Criteria for Review of Literature Articles

<table>
<thead>
<tr>
<th>Inclusion Criteria</th>
<th>Exclusion Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults (age 18 years and older)</td>
<td>Pediatrics (age 17 years and younger)</td>
</tr>
<tr>
<td>Articles written in English</td>
<td>Articles not available in English</td>
</tr>
<tr>
<td>Full-Text Articles</td>
<td>Abstract Only</td>
</tr>
<tr>
<td>Interventions in Acute Care Setting</td>
<td>Interventions for post-acute or rehab setting</td>
</tr>
<tr>
<td>Animal and Human Studies</td>
<td></td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Description</td>
</tr>
<tr>
<td>--------------</td>
<td>-------------</td>
</tr>
<tr>
<td>ASPEN</td>
<td>American Society of Parenteral and Enteral Nutrition</td>
</tr>
<tr>
<td>BMI</td>
<td>Body Mass Index</td>
</tr>
<tr>
<td>CRRT</td>
<td>Continuous Renal Replacement Therapy</td>
</tr>
<tr>
<td>EVD</td>
<td>External Ventricular Drain</td>
</tr>
<tr>
<td>GCS</td>
<td>Glasgow Coma Score</td>
</tr>
<tr>
<td>IBW</td>
<td>Ideal Body Weight</td>
</tr>
<tr>
<td>ICH</td>
<td>Intracerebral Hemorrhage</td>
</tr>
<tr>
<td>IC</td>
<td>Indirect Calorimetry</td>
</tr>
<tr>
<td>ICP</td>
<td>Intracranial Pressure</td>
</tr>
<tr>
<td>ICU</td>
<td>Intensive Care Unit</td>
</tr>
<tr>
<td>IDDSI</td>
<td>International Dysphagia Diet Standardization Initiative</td>
</tr>
<tr>
<td>IL</td>
<td>Interleukin</td>
</tr>
<tr>
<td>SLP</td>
<td>Speech-Language Pathology</td>
</tr>
<tr>
<td>TSH</td>
<td>Thyroid Stimulating Hormone</td>
</tr>
<tr>
<td>CSF</td>
<td>Cerebrospinal Fluid</td>
</tr>
<tr>
<td>CT</td>
<td>Computed Tomography</td>
</tr>
<tr>
<td>DHA</td>
<td>Docosahexaenoic Acid</td>
</tr>
<tr>
<td>ED</td>
<td>Emergency Department</td>
</tr>
<tr>
<td>EPA</td>
<td>Eicosapentaenoic Acid</td>
</tr>
<tr>
<td>NG</td>
<td>Nasogastric</td>
</tr>
<tr>
<td>NJ</td>
<td>Nasojejunal</td>
</tr>
<tr>
<td>OT</td>
<td>Occupational Therapy</td>
</tr>
<tr>
<td>PEG</td>
<td>Percutaneous Endoscopic Gastrostomy</td>
</tr>
<tr>
<td>PEJ</td>
<td>Percutaneous Endoscopic Jejunostomy</td>
</tr>
<tr>
<td>PUFA</td>
<td>Polyunsaturated Fatty Acid</td>
</tr>
<tr>
<td>RD</td>
<td>Registered Dietitian</td>
</tr>
<tr>
<td>SAH</td>
<td>Subarachnoid Hemorrhage</td>
</tr>
<tr>
<td>TBI</td>
<td>Traumatic Brain Injury</td>
</tr>
</tbody>
</table>
Introduction

Traumatic Brain Injury (TBI) is a leading cause of death and disability in the United States. The Centers for Disease Control and Prevention (CDC) defines TBI as a disruption in the normal function of the brain that can be caused by a bump, blow, or jolt to the head or penetrating head injury. The primary injury can be a result of either a penetrating (open-head) or a non-penetrating (closed-head) injury. TBIs can occur from many common events including falls, vehicle-related collisions, violence, high-impact sports injuries, and explosive blasts/combat injuries. Falls account for almost one-half (48%) of all TBI-related Emergency Department (ED) visits, which increases significantly in individuals aged 65 years and older (81%). Although anyone can be at risk for a TBI, they are most commonly seen in children (less than 18 years of age) and older adults as well as males in any age group.

Around 2.87 million TBI-related ED visits, hospitalizations and deaths are reported annually; with an average of 155 individuals who die daily from TBI-related injuries in the United States. Additionally, approximately 5.3 million individuals live with a disability caused by a TBI. Acute management of this trauma is important for the prognosis and quality of life for the affected individual.

The term “mass lesion” is frequently used when referring to TBI injuries and describes the area of localized injury that may cause pressure within the brain. Mass lesions in the context of TBI are most commonly hematomas and contusions. Hematomas are blood clots that may occur anywhere in the brain and include epidural hematomas (between the dura mater and the inside of the skull) and subdural hematomas (between the dura mater and arachnoid layer). Contusions that occur as a result of brain injury can result in bleeding within the brain tissues, or
intracerebral hemorrhage (ICH). Subarachnoid hemorrhages (SAH), however are classified by bleeding into the subarachnoid space and are common after a TBI, even in mild cases. The severity of TBI is assessed after initial resuscitation and within 48 hours of the injury and is typically classified by using the Glasgow Coma Scale (GCS) (Table 1). The GCS assess the verbal, motor skills and eye response and a severe TBI is generally graded by a GCS score less than 8, moderate is a score between 9-12, and 13-15 is considered a mild head injury.

<table>
<thead>
<tr>
<th>Eye Response</th>
<th>Spontaneous</th>
<th>4 points</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>To verbal stimuli, command, speech</td>
<td>3 points</td>
</tr>
<tr>
<td></td>
<td>To pain only</td>
<td>2 points</td>
</tr>
<tr>
<td></td>
<td>No response</td>
<td>1 point</td>
</tr>
<tr>
<td>Verbal Response</td>
<td>Oriented</td>
<td>5 points</td>
</tr>
<tr>
<td></td>
<td>Confused Conversation, able to answer questions</td>
<td>4 points</td>
</tr>
<tr>
<td></td>
<td>Inappropriate Words</td>
<td>3 points</td>
</tr>
<tr>
<td></td>
<td>Incomprehensible Speech</td>
<td>2 points</td>
</tr>
<tr>
<td></td>
<td>No response</td>
<td>1 point</td>
</tr>
<tr>
<td>Motor Response</td>
<td>Obeys Commands</td>
<td>6 points</td>
</tr>
<tr>
<td></td>
<td>Purposeful movement to painful stimuli</td>
<td>5 points</td>
</tr>
<tr>
<td></td>
<td>Withdraws in response to pain</td>
<td>4 points</td>
</tr>
<tr>
<td></td>
<td>Flexion in response to pain</td>
<td>3 points</td>
</tr>
<tr>
<td></td>
<td>Extension in response to pain</td>
<td>2 points</td>
</tr>
<tr>
<td></td>
<td>No response</td>
<td>1 point</td>
</tr>
</tbody>
</table>

Due to our incomplete knowledge of the cellular and molecular mechanisms of TBI there are currently no therapies to reduce TBI-associated damage. This is why the majority of interventions are aimed at reducing secondary injuries in an effort to reduce total damage.
Secondary injuries may include ischemia, hypoxia, hypotension, hypertension, cerebral edema, increased pressure in the skull, meningitis, biochemical changes and epilepsy.\textsuperscript{2} As a result of the secondary injury changes in levels of consciousness, memory disturbances, new onset or worsening of seizure disorder, visual field deficits, hemiparesis, dysphagia, and aphasia may occur.\textsuperscript{2,3,6}

A more commonly used invasive intervention is the use of intracranial pressure (ICP) monitoring. The gold standard for ICP monitoring is the external ventricular drain (EVD), which is a catheter that is placed in the third ventricle of the brain.\textsuperscript{7} This is used to not only monitor pressure in the brain but also works to drain cerebrospinal fluid (CSF), which can help treat intracranial hypertension. Less invasive interventions that help manage secondary injuries include elevating the head of the bed to 30 degrees to promote venous drainage, mannitol therapy and hypertonic saline, which act as a diuretic to reduce ICP, and high dose barbiturate therapy to reduce ICP.\textsuperscript{7,8} Surgical interventions may be needed if cerebral edema does not respond to non-invasive treatments or is required by the degree of swelling.\textsuperscript{7} If surgery takes place it is usually a decompressive hemicraniectomy followed by eventual cranioplasty (bone flap replacement).\textsuperscript{7}

The role of the registered dietitian (RD) is integral throughout the entirety of a patient’s recovery from a TBI. In the acute phase, the RD is responsible for determining the nutritional needs, guide the appropriate route for nutrition (e.g. parenteral vs. enteral nutrition) minimize complications of nutrition support, and eventually guide the transition from nutrition support to an oral diet. RDs can be invaluable to the patient in the outpatient setting to assist with weight management. In the rehabilitation phase after a TBI, patients often struggle with weight management as a result of behavior disturbances, cognitive impairment, side effects of medications, alcohol and tobacco use, poor motor function and poor sleep quality and fatigue.\textsuperscript{9,10}
Nutritional Needs and TBI

After a brain injury, it is essential to provide adequate nutrition in order to maintain normal body processes and promote healing. The RD plays a primary role in determining estimated energy needs based on predictive equations. These equations may be influenced by the policies set by the particular health care system or facility, research-based best practices, and/or clinical judgement. The estimated energy needs are then used to determine if the patient’s intake is adequate to maintain their nutritional status through an oral diet or to guide nutrition support needs. Determining the energy and protein needs and the ability to consume an oral diet lays the foundation for effective nutrition interventions after a TBI.

Energy Needs

The body responds to trauma in a cascade of metabolic changes commonly called the ebb and flow response. The ebb phase, or decreased metabolic rate, occurs early following the injury which is characterized by a decrease in total body energy and nitrogen excretion in an effort to reduce energy depletion.\textsuperscript{11–13} This phase typically lasts less than 24 hours. Hemodynamic instability can occur due to decreased circulating volume. In this phase there is an increase in endocrine hormones such as catecholamines and cortisol, which leads to the flow phase.\textsuperscript{13}

The ebb phase is followed by the flow phase, or the catabolic stage. After resuscitation from the shock of the trauma there is an increased metabolic turnover, activation of the immune system and induction of the hepatic acute-phase response.\textsuperscript{11–13} This causes a significant increase in oxygen consumption and energy expenditure and is characterized by a negative protein balance (-3 to -16 g nitrogen per day).\textsuperscript{14} In this phase the body’s primary function is to protect vital organ functions and rebuild tissue damage.\textsuperscript{13} When adequate energy and protein substrates are not available these functions suffer and places the patient in an increasingly critical state. In
the early catabolic stage an increase in catecholamines, glucagon, and corticoids, insulin resistance, cytokines, eicosanoids, and oxygen radicals produce a subsequent increase in energy production and consumption, which results in increased energy needs. Catabolism typically occurs in peripheral tissue such as muscle, adipose, and skin and is used to create the required response for wound healing. This response to stress can only be reversed through medical management such as reducing infection, inflammation and heat loss. Initiation of nutrition support can reduce negative energy and protein balance but may not completely reverse a negative protein balance until the anabolic stage begins.

The final stage of the stress response is the anabolic stage. The transition from the catabolic to anabolic stage depends on the injury severity and may take several weeks after severe trauma. This stage is characterized by the reduction of nitrogen excretion and is known as the corticoid withdrawal phase. The early stages of this phase may take weeks to months depending on nutrition supply and protein storage prior to injury and provision of nutritional needs post-injury. Nutrition support is impactful in the recovery of the patient during this phase, largely demonstrated by provision of ample protein and calorie intake. Achieving a positive nitrogen balance through adequate protein intake correlates to an increase in protein synthesis and progressive restoration of weight and muscle strength over a period of weeks to months.

As is common with many trauma patients, those who have suffered a TBI also have a corresponding hypermetabolic state. While it has been historically well documented that there is an increase in energy needs, there is no clear consensus on the extent of hypermetabolism, reason for the increase, or the length in which energy needs are increased. Studies have indicated that in the acute stage of head injury, energy needs vary from 87% to 200% of basal energy
These energy needs may be increased for as long as 30 days in the post-injury stage. It also has been suggested that the GCS is inversely related to the degree of hypermetabolism indicating that lower GCS scores may require further increased energy needs. 9

Indirect calorimetry (IC) is the gold standard for determining estimated energy needs in critically ill patients. 19,20 However, IC is rarely used in practice due to cost, equipment availability, and the many variables in the intensive care unit (ICU) that reduce the accuracy of the measurements (e.g., air leaks/chest tubes, supplemental oxygen, ventilator settings, continuous renal replacement therapy (CRRT), anesthesia, excessive movement, etc.). 19 As a result, many practitioners rely on predictive energy equations to assess calorie needs, which results in a risk of over- or underfeeding the patient.

The American Society for Parenteral and Enteral Nutrition (ASPEN) reported that when over 200 predictive equations were measured against IC none stood out against the others in the ICU. 19 Further, many of the formulas are frequently inaccurate in the ICU due to constantly changing variables such as medications, temperature and medical treatments. 19 For instance, barbiturates, paralyzing agents, and sedatives can reduce energy needs by 12-36%. 18 Table 3 outlines several energy equations. In critically ill patients who are non-obese, the Penn State equation is the most accurate (79%) followed by Swinamer (55%), and Ireton-Jones (52%). 20 However, Harris Benedict and simplified formulas (typically 25-30 kcal/kg in normal weight individuals with body mass index (BMI) <25 kg/m²) are frequently used in practice. 21 Permissive underfeeding (reduced calorie intake, high protein intake) is a commonly used protocol in patients who are obese (>30 kg/m²) to preserve lean body mass, mobilize fat stores and minimize metabolic complications. 22 Ultimately, it is critical for the practitioner to use clinical judgement and to take a multifactorial approach to determining energy needs.
In a case study comparing these common formulas conducted as part of this creative component, it was found that the Harris- Benedict, Mifflin-St. Jeor equations multiplied by a stress factor of 1.2-1.5, the simplified equation (25-30 kcal/kg) were comparable (Appendix A). In comparison, Penn-State, Swinamer, and Ireton-Jones equations multiplied by the same stress factor all had higher calorie estimates by an average of 266-364 kcal. In this case, indirect calorimetry is not available to determine actual requirements for comparison.

**Table 3. Equations for Estimated Energy Needs**

<table>
<thead>
<tr>
<th></th>
<th>Equation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Penn State</strong></td>
<td><strong>RMR = (Mifflin – St. Jeor x 0.96) + (V_E x 32) + (T_max x 167) – 6212</strong></td>
</tr>
<tr>
<td></td>
<td><strong>T_max= max body temp in previous 24h (degrees Celsius); V_E= minute</strong></td>
</tr>
<tr>
<td></td>
<td><strong>ventilation (L/min)</strong></td>
</tr>
<tr>
<td><strong>Swinamer</strong></td>
<td><strong>RMR = (BSA x 945) – (A x 6.4) + (Temp x 108) + (RR x 24.2) + (VT x</strong></td>
</tr>
<tr>
<td></td>
<td><strong>81.7) – 4349</strong></td>
</tr>
<tr>
<td></td>
<td><strong>BSA=body surface area (m^2); A= age (years); Temp = degrees Celsius;</strong></td>
</tr>
<tr>
<td></td>
<td><strong>RR= respiratory rate (breaths/min); VT = tidal volume (L)</strong></td>
</tr>
<tr>
<td><strong>Ireton-Jones</strong></td>
<td><strong>RMR = 1784 – (11 x A) + (5 x W) + (244 x S) + (239 x T) + (804 x B)</strong></td>
</tr>
<tr>
<td></td>
<td><strong>A= age (years); W=weight (kg); S=sex (1-male, 0-female); T= trauma</strong></td>
</tr>
<tr>
<td></td>
<td><strong>(1-present, 0-absent); B=burns (1-present, 0-absent)</strong></td>
</tr>
<tr>
<td><strong>Mifflin-St. Jeor</strong></td>
<td><strong>Men: RMR = 5 + (10 x W) + (6.25 x H) – (5 x A)</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Women: RMR = -161 + (10 x W) + (6.25 x H) – (5 x A)</strong></td>
</tr>
<tr>
<td></td>
<td><strong>W=weight (kg); H=height (cm); A= age (years)</strong></td>
</tr>
<tr>
<td><strong>Harris Benedict</strong></td>
<td><strong>Men: RMR = 66.47 + (13.75 x W) + (5 x H) – (6.76 x A)</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Women: RMR = 665.1 + (9.56 x W) + (1.7 x H) – (4.7 x A)</strong></td>
</tr>
<tr>
<td></td>
<td><strong>W=weight (kg); H=height (cm); A= age (years)</strong></td>
</tr>
<tr>
<td><strong>Simplified</strong></td>
<td><strong>BMI &lt;25 kg/m^2 - 25- 30 kcal/kg Actual BW</strong></td>
</tr>
<tr>
<td></td>
<td><strong>BMI 30-50 kg/m^2 – 11-14 kg/kg Actual BW</strong></td>
</tr>
<tr>
<td></td>
<td><strong>BMI &gt;50 kg/m^2 - 22-25 kg/kg Ideal BW</strong></td>
</tr>
</tbody>
</table>
Protein Needs

After trauma, protein is the most important macronutrient for healing wounds, maintaining lean body mass and supporting immune function. 21,23 ASPEN suggests using a simplistic equation of 1.2 to 2.0 g/kg of protein for individuals who are categorized as normal weight (BMI <25 kg/m²) in the ICU is sufficient. 21 For patient in the ICU who are categorized as obese, increased protein intake with permissive underfeeding is indicated. 22 ASPEN recommend providing up to 2 g/kg ideal body weight (IBW) for individual with a BMI of 30-40 kg/m² and up to 2.5 g/kg IBW for BMI ≥50 kg/m².22 However, there are currently no studies available that have investigated the protein needs for patients with TBI. In order to provide adequate protein intake without providing excess calories, modular protein products (such as Beneprotein® or Prosource®) may be required. This is particularly important since overfeeding critically ill patients can cause hyperglycemia, increased carbon dioxide (CO₂) production, increased risk of infection. 23

Dysphagia

Dysphagia, or impaired swallowing is a frequent TBI-related complication. Many factors that impair swallowing in these individuals including surgeries, prolonged disuse of swallowing muscles, medications that may impair swallowing function (e.g., muscle relaxers, medications that cause xerostomia, antipsychotics/ neuroleptics), intubation-related injuries, and prolonged intubation/tracheostomy placement. 24 For individuals with a severe TBI (GCS 3-5), computed tomography (CT) can show midline shift, brainstem involvement, and brain pathology which may require emergent operative intervention. These individuals are at the highest risk for abnormal swallow function, aspiration and delay in initiation of oral nutrition. 25
The incidence of dysphagia is as high as 93% in individuals who have suffered a brain injury, although reports vary significantly.24,26–28 Identifying dysphagia is important as complications that arise from impaired swallowing can be contribute to secondary complications such as infections leading to longer hospital stays, increased cost, and can lead to weight loss, malnutrition and dehydration.29,30 Abdelmalik and others reported that 94% of individuals who were unable to safely consume oral nutrition at time of the initial injury were ultimately able to successful take oral nutrition with improvement in neurological status and therapy.7

Dysphagia diets, such as the International Dysphagia Diet Standardization Initiative (IDDSI) provide a progression of textures from safest (e.g. pureed diet, pudding-thick/extremely thick liquids, etc.) to a regular diet.31 IDDSI is the framework facilities are adapting to help standardize dysphagia diet terminology and diet progression globally. 32 The goal of switching to standardized language and testing is to ensure quality and safety of the diet across all platforms and to ensure that the patient is receiving the highest standard of care. The RD, with help from the occupation therapy (OT) or speech language pathology (SLP) services, provide education and ensure compliance with the patient’s diet. Due to the inability to consume adequate oral intakes while swallow function is impaired, many individuals are reliant on nutrition support to meet their nutritional needs after a TBI.

Nutrition Support and TBI

Nutrition support is needed when the patient is intubated, significantly impaired neurological status or is experiencing significant dysphagia. Early initiation of feeding is correlated with better outcomes including reduced infectious complications, positive effects on hormone profile (cortisol, thyroid stimulating hormone (TSH), free thyroxine, etc.), reduced risk of malnutrition, GCS recovery, and decreased the length of stay. 14,33–38 In addition, patients fed
earlier rather than later show a tendency towards less morbidity and mortality and may reduce relative risk of disability. Härtl and colleagues reported a two-fold increased likelihood of death in patients who were not fed within five days after injury and four-fold increase after seven days. Furthermore, Härtl et al. found that the amount that they were fed in this first five days was also related to death with every 10-kcal/kg decrease in calorie intake being correlated to a 30% to 40% increase in mortality. RDs need to be part of the treatment team early to advocate for nutrition support in patients who are metabolically compromised to avoid complications associated with delayed nutrition. While there is substantial evidence to support the initiation of early nutrition support, there are conflicting reports of whether enteral or parenteral nutrition produces better outcomes in TBI patients. Many studies have found that both routes are equally effective at meeting nutritional goals.

**Enteral Nutrition**

ASPEN recommends enteral nutrition be provided if the gastrointestinal tract is functioning for most patients who have undergone trauma, including TBI. Early enteral nutrition (within 24-48 hours) is beneficial as it supports the integrity of the gut by maintaining the intestinal barrier, stimulating blood flow, inducing release of trophic endogenous agents (e.g. cholecystokinin, gastrin, bombesin, and bile salts) and reduces infectious morbidity and ICU length of stay.

A review of patients in the ICU with TBI suggests that as high as 94% of individuals required enteral nutrition. There are many ways in which to administer enteral nutrition including nasogastric (NG), nasojejunal (NJ), percutaneous endoscopic gastrostomy (PEG), or percutaneous endoscopic jejunostomy (PEJ). The location and type of the tube depends on tolerance to enteral nutrition and the anticipated length of time the patient will require enteral
When it is determined that the patient cannot take nutrition orally an NG tube will be placed for nutrition. This is usually the first attempted route of nutrition as this can be completed at bedside by a nurse, physician, or dietician and placement for this route can be easily confirmed by portable abdominal X-ray. An NG tube placement requires that the prolonged supine position be avoided, and the head of the bed should be maintained at 30-45 degrees during feeding to minimize the risk of aspiration.

A post-pyloric (NJ) feeding tube may be indicated if the patient has to be in a prolonged supine position due to this high risk of aspiration during enteral nutrition or if vomiting and abdominal distention occur. This is reserved for these patients as tubes placed post-pyloric must be done so under x-ray and may require justification for insurance to cover these tubes as an outpatient due to additional equipment needed. Post-pyloric feeding effectively meets nutrition requirements, reduce gastric residue, and improves gastrointestinal dysfunction that is typically seen with TBI patients.

Many patients with TBI have altered gastrointestinal function after injury which can lead to feeding intolerance. Lee reported TBI was associated with twice the incidence of intolerance to gastric feeding compared to other trauma diagnoses. It has also been suggested that 50% to 80% of patients with head injuries exhibit delayed gastric liquid emptying compared to those who did not. Altered gastric motility and emptying may occur as a result from the injury itself due to damage in the autonomic central nervous system or nerves. Individuals who have undergone a trauma show decreased bowel sounds, gastroparesis, lack of cardiac response to tracheal stimulation and high gastric retention. Many of these complications are related to vagal dysfunction related to peripheral autonomic dysfunction. It has been suggested that increased intracranial hypertension, especially in TBI patients, can lead to the suppression of
vagal activity and when this brain-gut link is altered this can result in gastric dysrhythmias and intolerance to enteral nutrition. 52,53 An increased ICP level and the severity of the TBI is related to the level of intolerance to tube feeds. 54 Intolerance to tube feeding frequently results in decreased delivery of enteral nutrition and increased length of ICU stay. 19

Gastric motility issues typically last in the majority of patients with a head trauma for the first two weeks post injury. 14,51 Kao et al. reported patients with TBI were able to tolerate full enteral nutrition by day 16. 50 Common complications related to tolerance to tube feedings include interruptions for various medical interventions and unintentional removal of the feeding tube. 39 This can impede adequate nutrition in these patients, and with the extent of increased metabolic needs this places these patients at further risk for malnutrition. 39 Many individuals assess tolerance to enteral nutrition by measuring gastric residuals, which is the most common feeding intolerance. 53 Diarrhea is another common feeding intolerance for individuals receiving enteral nutrition. This is influenced by the type and amount of fiber in formula, osmolality of formula, delivery mode, EN contamination, medications (antibiotics, proton-pump inhibitors, prokinetics, glucose lowering agents, nonsteroidal anti-inflammatory drugs (NSAIDs), laxatives, and sorbitol-containing preparations), and infectious etiologies, including Clostridium difficile (C.Diff). 19 The RD can facilitate improved tolerance to enteral nutrition by recommending a slower advancement rate to goal feeds, changing the formula, transitioning to a continuous enteral nutrition schedule from intermittent, or advancement of the tip of the tube, if applicable. When other source of intolerance cannot be identified the introduction of prokinetic agents may be indicated.

Providing prokinetic agents such as erythromycin or metoclopramide can help control these gastrointestinal disorders in TBI patients by speeding up gastric emptying and movement
in the upper GI tract, resulting in reduced gastric residuals.\textsuperscript{43,55,56} Dickerson and colleagues suggest that the combination of these medications is the more effective in improving gastric feeding than metoclopramide alone. \textsuperscript{43} However, patients with TBI are typically less responsive to prokinetic therapy with an estimated three-fold probability of failure to metoclopramide therapy. \textsuperscript{43} Additionally, in some instance barbiturates may be used to reduce ICP, which can severely hinder gastrointestinal motility and is commonly refractory to motility agents. \textsuperscript{57} Failure to respond to metoclopramide may also be influenced by low serum albumin concentration, early initiation of enteral nutrition and prokinetic therapy, and high ICP. \textsuperscript{43} If all interventions have been exhausted to improve tolerance of enteral nutrition, parenteral nutrition may be indicated.

\textit{Parenteral Nutrition}

According to ASPEN guidelines, if the patient has a functioning gastrointestinal tract enteral nutrition is recommended, however some patients are unable to tolerate tube feeds and parenteral nutrition is indicated to meet nutrition needs. \textsuperscript{19} After a TBI, parenteral nutrition provides significantly higher nitrogen intake which is recommended in critically ill patients to promote lean body mass and prevent catabolism. \textsuperscript{42} However, those receiving parenteral nutrition also resulted in increased nitrogen excretion; therefore, nitrogen balance was similar to those who were fed enterally. \textsuperscript{41,42} Parenteral nutrition may also help decrease the risk of aspiration pneumonia. \textsuperscript{58,59} However, results concerning the impact of parenteral nutrition on infection risk varies with many studies reporting no difference in serum albumin levels, weight loss, incidence of infection, or clinical outcome between parenterally and enterally fed patients. \textsuperscript{40–42} Still, the studies that were available frequently compared early initiation of parenteral nutrition compared to late initiation of enteral nutrition, which may not be an accurate depiction of the positive
effects of parenteral nutrition. Furthermore, hyperglycemia that is common with parenteral nutrition and can lead to worse neurological outcomes.40,42,60

Early parenteral nutrition may be indicated in individuals who receive high dose barbiturates due to reduced gastrointestinal motility which results in frequent intolerance to enteral nutrition. One study reported that all patients receiving barbiturate therapy failed nasogastric feeding within the first 48 hours of full induction into a barbiturate coma. 57 Additionally, only 25% of these patients tolerated post-pyloric feedings within 48 hours. Therefore, early parenteral nutrition may be indicated in these patients.

Transition to Oral Diet

When a safe oral nutrition program is determined by the interdisciplinary team, which frequently involves a texture modified diet, patients can begin weaning from nutrition support. The average time for the patient to safely consume their first oral meal after injury is about three months.7 Buchholz originally describes a two-phase algorithm for weaning patients from tube feeding to oral nutrition: Preparatory Phase and Weaning Phase. 61

The Preparatory Phase consists of stability from a medical and nutritional standpoint, a swallowing assessment by an OT or SLP, and the transition from continuous tube feeding to an intermittent feeding schedule.61 Intermittent tube feedings provide full nutrition needs over several 20-60 minute time periods every 4-6 hours through a gravity bag, pump or syringe.62 This phase continues until a patient is cleared for an oral diet, which is determined by an OT or SLP.

The Weaning Phase is the progression from therapeutic feeds to full oral nutrition and eventually discontinuing enteral nutrition. This involves providing supplemental tube feedings based on amount of meal eaten or nocturnal tube feeds. 61,63 For example, if a patient consumes
50% of a meal, only 50% of the goal tube feeding for that meal period would be given. (Appendix A) Once 75% of the patient’s nutritional needs are met through solid foods for a minimum of three days enteral nutrition can be discontinued. It is the RD’s role to provide oral nutrition supplements and counsel the patient on therapeutic diets to encourage adequate intake. During the weaning phase, the patient is monitored closely for aspiration, dehydration and to ensure that feeding and swallowing strategies as well as texture modifications to enable the patient to safely consume adequate oral intake.

Long-Term Nutrition Support

When long-term enteral access is expected based on functional/mental status and severity of dysphagia, a percutaneous endoscopic gastrostomy (PEG) or jejunostomy (PEJ) tube may be indicated. If the patient remains mechanically ventilated, an early intervention of combined tracheostomy and PEG placement, within the first seven days of injury, reduces ICU and hospital length of stay and shortens the course of mechanical ventilation compared to late intervention. If the patient is able to be extubated but is unable to initiate a diet due to swallowing difficulties or neurological status a PEG tube may be indicated due to the length of time necessary to regain swallowing function. Additionally, most skilled nursing facilities and inpatient rehab centers will deny admission when the patient is medically stable for discharge if they have a nasogastric or jejunal feeding tube. Consideration for insurance approval should also be taken when considering PEG tube placement Medicare guidelines state that the patient must have a permanent (defined as greater than three months) impairment of structures that normally allow food to meet the stomach or small intestine, such as severe dysphagia. Many other insurance providers mirror these guidelines although coverage may vary between insurance policies.
Insurance coverage generally continues if the patient is taking small bites of food by mouth for therapeutic purposes in an effort to transition to a full oral diet.

**Immunonutrition and TBI**

There is growing interest in using immunonutrition to modulate the inflammatory response to the TBI and to facilitate the reduction of a secondary injury. A TBI increases the levels of inflammatory cytokines, including interleukin-6 (IL-6), which is a key regulator of the inflammatory response. Elevated IL-6 have been related to poor outcomes for patients with TBI.

In response to the increased demand for immunonutrition, many enteral nutrition companies have created immune-modulating formulas that are available for patients with TBI in addition to use in patients who are admitted due to trauma or surgery. Many of these formulas are high protein to support the nutritional needs for healing and peptide-based to help with tolerance and digestion. Formulas marketed as immune-modulating are typically supplemented with arginine, glutamine, and omega-3 fatty acids. Immune-modulating formulas containing omega-3 fatty acids, arginine, and glutamine decrease IL-6 levels in patients with TBI. However, immunonutrition may have no impact on mortality or length of stay.

**Omega-3 Fatty Acids**

Long-chain polyunsaturated fatty acids (PUFA), particularly omega-6 and omega-3 fatty acids, are found in large quantities in the central nervous system (CNS). The human brain is made up of 60% lipid by dry weight and docosahexaenoic acid (DHA) comprises 50% of neuronal membrane phospholipids. Although, omega-3 fatty acids show promise in helping with TBI, most studies have been conducted in mice and no clinical trial data is available in humans presently. Fatty acids have an indirect role as metabolic precursors for eicosanoids.
which play a role in platelet aggregation, neurotransmitter release, vascular function, infection responses, inflammatory activity, and immune system activity. 

21,75 Alpha-linolenic acid, an essential fatty acid, is a precursor for DHA and eicosapentaenoic acid (EPA); however, because this conversion process is inefficient in obtaining DHA and EPA, intake through the diet is recommended. 

21 EPA and DHA are both critical for eyesight and brain function. DHA is the most abundant in the CNS and is shown to have neuroprotective functions. 72,73 Omega-3 fatty acids are commonly found in flaxseed, fish and seafood, soybean and canola oils. 21 Some immune-enhancing enteral formulas, as well as the lipids that are compounded in parenteral nutrition may contain supplemented omega-3 fatty acids. 21

Following a TBI, DHA and EPA are converted to neuroprotectins and resolvins which then prevent cell death and upregulate tissue repair. 21,76 Research using mice models suggests an intravenous DHA bolus in the first hour of trauma may significantly improve neurological outcome. 73 This acute bolus rapidly changed the lipid mediator profile at the site of the injury and promotes a resolving mediator profile as early as three hours after administration. 73 However, this effect may be lost if the administration is not given within four hours of injury. 73 Omega-3 fatty acid supplementation for at least four weeks after injury may counteract long-term learning disability related to the injury. 77–80 A case report reported by Lewis et. al. reports that providing a substantial amount (the patient received 9,756 mg EPA, 6756 mg DHA and 19,212 mg total omega-3 fatty acids through PEG) provided early in treatment may have positive effects on recovery. 81 Although research suggests that omega-3 fatty acid supplementation may be beneficial following a TBI, more studies in humans are lacking thus more research is needed.
Zinc

Zinc is an essential trace metal strongly associated with neuronal and immune system health due to its role as an antioxidant. 4,82 Zinc works to inhibit anti-inflammatory responses that would typically cause oxidative stress, commonly seen in zinc deficiency. 4,82 Zinc is also integral in the brain function across the lifespan by regulating gene expression as managing many key enzymes in neuronal metabolism. 82 Given the significant influence on brain function, it is suggested that alterations in zinc status can be responsible for many neurodegenerative disorders such as Alzheimer’s disease and mood disorder as well as neuronal damage associated with TBI, stroke and seizures.4

Following a TBI the development of zinc deficiency is common. Along with zinc deficiency, there is evidence of continued cell death for four weeks following the injury.4,9 Patients with TBI typically have elevated urinary zinc losses that persist for weeks following injury and result in reduced serum zinc levels.4 These urinary zinc losses are proportional to TBI severity.83 Providing patients who have had a TBI with 22-25 mg/day of zinc improves the rate of neurologic recovery and protein metabolism. 9,83 One study found that zinc supplementation (22 mg/d) compared to those receiving adequate zinc (2.5 mg/d) resulted in lower mortality rates (12% vs 26%, respectively) as well as significantly improved GCS scores in as early as two weeks after initiation. 9 Another found that prolonged zinc supplementation (25 mg/d) reduced physiological responses to stress and anxiety, improved cognitive behavior, and prevent impairment of spatial learning and memory. 83 Zinc supplementation is an inexpensive therapy and should be considered after a TBI due to the positive effects on neurologic recovery and lack of negative effects. 83 Long-term zinc supplementation should be used with caution due to its link with copper deficiency.
Glutamine

Glutamine is a conditionally essential amino acid that plays a large role in cell maintenance through numerous functions. These functions include acting as a substrate for protein synthesis, as an anabolic precursor for muscle growth, acid-base balance in the kidney, substrate for ureagenesis in the liver, substrate for hepatic and renal gluconeogenesis, an oxidative fuel for intestine and cells of the immune system, inter-organ nitrogen transport, precursor for neurotransmitter synthesis, precursor for nucleotide and nucleic acid synthesis and as precursor for glutathione production. 21,84–86 A study in mechanically ventilated patients with TBI concluded that enteral nutrition supplemented with glutamine and probiotics reduces infection rate, decreases the occurrence of pneumonia, stress ulcers, and shortens the length of ICU stay. 87,88

Although, there are many potential benefits to immunonutrition, it may be contraindicated for patients who are critically ill, including those with renal or liver disease. Rathmacher and colleagues found that blood urea was elevated in individuals who were receiving arginine and glutamine due to stimulation of ureagenesis. 89 As such, consideration should be given for patients who have comorbidities such as renal or liver disease prior to initiating immunonutrition. Despite the association between low glutamine concentration and poor outcome, early supplementation did not improve outcomes. 90

Conclusion

Early nutritional intervention to provide adequate nutrition is crucial for the survival and recovery of a patient following a TBI. The RD plays a major role in determining and guiding the Medical Nutrition Therapy. Specifically, the route of nutrition (oral vs. enteral vs. parenteral), as well as troubleshooting nutrition-related complications associated with TBI. This review of
literature demonstrates that more research is needed to determine the best evidence-based nutritional support recommendations for treating TBI.

**Take Home Messages**

As a result of this review of literature, many of these recommendations will be adapted to my current practice. Per my hospital’s policy I will calculate estimated energy needs using Harris-Benedict equation and select a stress factor of at least 120-150% of basal energy needs due to hypermetabolism seen after a TBI. I will use ASPEN guidelines to determine protein needs. Due to the metabolically compromised nature of this population, I will advocate for early initiation of enteral nutrition and will select a product that has immunological benefits such as Impact 1.5 peptide or Pivot 1.5. Additionally, I would like to incorporate checking zinc levels and providing supplementation when appropriate into my recommendations. Through this review I have become more aware of signs of intolerance as well as interventions to alleviate these and promote adequate nutrition and positive outcomes in this population.


*BMC Anesthesiology*. 2017;17(1). doi:10.1186/s12871-017-0369-4

68. McCarthy MS, Martindale RG. Immunonutrition in critical illness: What is the role? 


APPENDIX A

Case Study Comparing Various Energy Needs Equations

Patient Description

A 70-year-old male is admitted to the neurosciences ICU after being found unconscious after a fall. The patient was transferred to the unit intubated, mildly sedated on Precedex for agitation, which was quickly weaned and a nasogastric tube was placed. A CT scan of the head showed bilateral traumatic subdural, subarachnoid and intraparenchymal hemorrhages with a GCS of 7T (E1V1T5). Patient is status-post decompressive hemi-craniotomy for management of cerebral edema. The patient’s respiratory rate is 16 breaths/min, tidal volume of 4.4 L, and minute ventilation is 7.04 L/min. Maximum temperature in the past 24 hours of 37.2 degrees Celsius. He is now 24 hours following injury and the RDN receives a consult for tube feeding management.

Anthropometric Data:

Height: 177.8 cm / 70”  Weight: 77.3 kg / 170 lbs  IBW: 75.5 kg / 102% IBW  BMI: 24.5 kg/m²

Body Surface Area: 1.95 m²

RECOMMENDATIONS:

Estimated Energy Needs:

- Harris Benedict: \( RMR = 1543 \text{ kcal} \times SF (1.2-1.5) = 1852-2315 \text{ kcal} \)
- Simplified Equations: \( (25-30 \text{ kcal/kg}) = 1933-2319 \text{ kcal} \)
- Mifflin-St. Jeor: \( RMR = 1542 \text{ kcal} \times SF (1.2-1.5) = 1850-2315 \text{ kcal} \)
- Ireton-Jones: \( RMR= 1884 \text{ kcal} \times SF (1.2-1.5) = 2260-2826 \text{ kcal} \)
- Penn State: \( RMR = 1706 \text{ kcal} \times SF (1.2-1.5) = 2047-2559 \text{ kcal} \)
- Swanimer: \( RMR= 1770 \text{ kcal} \times SF (1.2-1.5) = 2124-2655 \text{ kcal} \)

Protein Needs: 93-155 g protein (1.2-2.0 g/kg)
Enteral Nutrition Needs:

RD recommends an initiation of an immune-modulating formula (such as Impact® 1.5 or Pivot® 1.5) at 20 mL/hour with advancement of 10 mL every 8 hours until a goal rate of 55 mL/hour (1320 mL/day) to provide 1980 kcal (25.6 kcal/kg), 124 g protein (1.6 g/kg), and meets 100% of recommended dietary intake of vitamins and minerals. Both formulas provide 1500 kcal/L, 94 g protein/L and each provides glutamine, 3.7g/L (Pivot®) -4.9 g/L (Impact®) of DHA+ EPA, and 13g/L (Pivot®) – 18.7 g/L (Impact®) of arginine. The RD also recommends checking a baseline zinc level and supplementing with 22-25 mg/day.

It has been 48 hours after the initiation of enteral nutrition and the patient has reached his goal rate. He is exhibiting signs of gastric intolerance to tube feeds with abdominal distention and thick white secretions have been suctioned from endotracheal tube thought to be tube feeding.

RECOMMENDATIONS:

• Assessment of bowel function – may consider bowel regimen to stimulate regular bowel movements and improve gastric emptying.

• A trial of metoclopramide or erythromycin to facilitate gastric emptying may be indicated.

• Advancement of the tip of the tube into the jejunum may be indicated as a result of delayed gastric emptying and to reduce risk for aspiration.

• If none of the above are successful, consider initiation of parenteral nutrition.
It has been three months after injury and the patient has been stable on an intermittent regimen via PEG tube. His current regimen is 500 mL at breakfast, 500 mL at lunch and 250 mL at dinner of immune-enhancing formula. He has made significant neurological improvements and is now cleared to start a pureed diet with mildly thickened liquids.

RECOMMENDATIONS:

• Start a calorie count on the patient to assess daily energy and protein intake.

• Provided intermittent tube feedings based on oral intakes at meals as follows:
  
  o If patient consumes ≤25% of meal, provide full feeding
  
  o If patient consumes 50% of meal, provide 50% of goal tube feeding
  
  o If patient consumes ≥75% of meal, hold tube feeding

• Once patient is able to consume ≥75% of estimated energy needs based on calorie count results for at least 72 hours.