The Association of UK Dietitians

Traumatic Brain Injury in Critical Care Practice Handbook for Dietitians

Working party from Major Trauma Group of the British Dietetic Association 2020 -2022

Dedications:

This handbook is dedicated to all the dietitians who worked in an intensive care setting during the COVID 19 pandemic.

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1. Abbreviations

AKI	Acute kidney injury
ALA	Omega-3 Alpha-Linolenic Acid
ASPEN	American Society for Parenteral and Enteral Nutrition
ARP	Arginine Rich Peptides
ATP	Adenosine Triphosphate
BAPEN	British Association for Parenteral and Enteral Nutrition
BDA	British Dietetic Association
BG	Blood Glucose
BMI	Body Mass Index
CKD	Chronic kidney disease
CRRT	Continuous Renal Replacement Therapy
CT	Computed Tomography
CVVH	Continuous Veno-Venous Haemofiltration
DGE	Delayed Gastric Emptying
EFFORT	The Effect of higher protein dosing in critical ill patients
EN	Enteral Nutrition
ESPEN	European Society for Parenteral and Enteral Nutrition
GCS	Glasgow Coma Score
GI	Gastrointestinal
GRV	Gastric Residual Volumes
IBW	Ideal Body Weight
IC	Indirect Calorimetry
IGC	Intensive Glucose Control (IGC)
KD	Ketogenic Diet
LOS	Length of stay
MAMC	Mid Arm Muscle Circumference
MDT	Multidisciplinary Team
MEE	Measured energy expenditure
MUAC	Mid Upper Arm Circumference
MUST	Malnutrition Universal Screening Tool
NGT	Nasogastric Tube
NICE	National Institute for Health and Care Excellence
NJT	Nasojejunal Tube
NNC	Non nutritional calories
OGT	Orogastric Tube
PCS	Post-Concussion Syndrome
PEN	Practice based Evidence in Nutrition
PN	Parenteral Nutrition
PSH	Paroxysmal Sympathetic Hyperactivity
REE	Resting Energy Expenditure
RCT	Randomized Controlled Trial
RRT	Renal Replacement Therapy
TBI	Traumatic Brain Injury
UUN	Urinary Urea Nitrogen
VBF	Volume Based Feeding
VRII	Variable Rate Intravenous Insulin Infusion

2. Traumatic Brain Injury in Critical care: Practice Handbook for Dietitians Introduction

BDA Trauma Handbook Working Party 2020-2022

This handbook has been written to provide guidance and support for Dietitians involved in the management of patients with Traumatic Brain Injury (TBI) in a critical care unit. The purpose of the handbook is to aid clinical judgement and help in ensuring that timely and appropriate nutrition support is provided to patients with TBI in critical care.

The handbook will focus on nutritional aspects of dietetic care including screening and anthropometry, enteral feeding, nutritional requirements and immune-modulating enteral formulas. The recommendations and guidance will be amalgamated with recent evidence including material from the Practice Based Evidence (PEN) database of the British Dietetic Association (BDA).

Experience from practising dietitians has been collated from a Trauma Workshop at the BDA Critical Care Study Day in November 2019. This has been included in the handbook to provide information regarding current dietetic practice for patients with TBI in critical care. Case studies are also provided to assist with the development of knowledge and skills for Dietitians working in this clinical area.

Please note all information in this handbook is for educational purposes only and not for commercial use.

3. Nutritional Screening for Patients with Traumatic Brain Injury in the Critical Care Unit

Amy Carter

Recommendation

Nutritional screening can be used in the critical care setting to identify patients with TBIs who will require dietetic intervention. This could be carried out by a Dietitian or where applicable by other Health Care Professionals, using a clinically appropriate screening tool.

Rationale

It is vital to identify critical care patients with TBI who are malnourished or at risk of malnutrition due to the hypermetabolic and hypercatabolic effects of TBI and potential complications due to altered gastrointestinal motility[1].

Some international guidelines advocate the use of nutritional screening tools on the critical care unit. The American Society for Parenteral and Enteral Nutrition (ASPEN)[2] summarises the importance of nutritional screening stating that: “based on expert consensus, determination of nutrition risk (e.g., nutritional risk screening, NUTRIC score) should be performed on all patients admitted to critical care for whom volitional intake is anticipated to be insufficient. High nutrition risk identifies those patients most likely to benefit from early EN [Enteral Nutrition] therapy”.

The NUTRIC screening tool which is recommended by ASPEN[2] is the first nutritional risk tool specifically recommended for critical care patients. The NUTRIC–S has since been developed which may be superior in terms of mortality predictions and can be used in settings where APACHE II is not available[3].

The British Association for Parenteral and Enteral Nutrition (BAPEN)[4] advises that most patients in typical critical care units are at risk of malnutrition, and this is captured in the Acute Disease Effect score on the MUST in view of ‘no or unlikely dietary intake for >5 days’.

NICE[5] recommends that all hospital inpatients on admission should be screened and Nutrition support should be considered in any of the following situations:

- a BMI of less than 18.5 kg/m^2
- unintentional weight loss greater than 10% within the last 3–6 months
- a BMI of less than 20 kg/m^2 and unintentional weight loss greater than 5% within the last 3–6 months.
- have eaten little or nothing for more than 5 days and/or are likely to eat little or nothing for the next 5 days or longer
- have a poor absorptive capacity, and/or have high nutrient losses and/or have increased nutritional needs from causes such as catabolism.

However, it is important to consider that ESPEN[6] recommend that every critically ill patient staying for more than 48 hours on critical care should be considered at risk of malnutrition and that a general clinical assessment should be performed to assess malnutrition in critical care, until a specific tool has been validated for use on critical care.

Summary of Experience from Dietitians in Trauma Workshop 2019

53% of Dietitians working in Trauma reported that nutritional screening takes place in patients with TBI in critical care. Of these, 80% report using the Malnutrition Universal Screening Tool (MUST) and in Trusts where nutritional screening was not used, 80% had a blanket referral system in place for the Dietitians.

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4. Anthropometry in Patients with Traumatic Brain Injury in Critical Care

Amy Carter

Recommendation

A dietetic assessment should be performed to determine risk of malnutrition which may include physical examination, general assessment of body composition, weight history preadmission, muscle mass or strength where possible.

Rationale

Head injury increases the risk of weight loss, muscle wasting, and malnutrition due to hypercatabolism and hypermetabolism[1]. The preservation of lean body mass in patient with TBI in critical care is challenging due to multifaceted consequences of injury. These include decreasing muscle protein synthesis due to inactivity and immobility, alongside multiple mechanisms which lead to deterioration of lean body mass and include calcium-dependent proteolysis, ATP-dependent proteolysis, lysosomal proteolysis, and free radical oxidative activation[2]. A prospective cohort study found that during and after the critical care stay, 40% of patients lost more than 10kg of their body weight[3].

In view of complex and compounded factors that influence dietetic assessment and monitoring, anthropometric measurements are key to the assessment and monitoring of patients with traumatic brain injury in critical care. An accurate weight for TBI patients in critical care is required for several reasons including calculating nutritional requirements, fluid balance monitoring, ventilation settings and the calculation of drug doses. A simple visual estimate of patients' body weight has been shown to be inaccurate in critical care with an error as much as 20% against measured values[4]. However, the process of weighing a critical care patient presents unique challenges and often requires cautious interpretation, due to a number of potential inaccuracies including weighing with equipment on the bed.

Badjatia et al[5] summarises that few recommendations can be made regarding monitoring nutritional status and response to nutritional interventions for patients with acute brain injury in critical care, recognising that while body weight is the most commonly used indicator in assessing nutrition status in a non-critical care population, it is more likely a marker of fluid balance than of nutritional status in critical care and not a reliable measure of responsiveness to nutritional therapy.

The role of anthropometry in nutritional assessments is supported by recent research[6] which found that bedside anthropometry, especially the mid arm muscle circumference (MAMC) is efficient in identifying patients with nutrition depletion with significant influence on outcome at 3 months. Interestingly, the study reported unfavourable outcomes were significantly more frequent in patients who had at least 15% fall in mid arm circumference, or 10% fall in MAMC (68.8 vs. 38.2%, $p = 0.04$) at 2 weeks, highlighting the role of anthropometry in nutritional assessment and monitoring.

An important consideration for the clinical assessment of patients with TBI on critical care is Paroxysmal Sympathetic Hyperactivity (PSH). This is defined as a "syndrome, recognised in a subgroup of survivors of severe acquired brain injury, of simultaneous, paroxysmal transient increases in sympathetic (elevated heart rate, blood pressure, respiratory rate, temperature, sweating) and motor (posturing) activity"[7]. In patients with PSH, body weight can decrease by 25-29% during the acute period and energy expenditure has been noted to be up to 3 times in patients with PSH[8]. Caldwell et al.[9] conducted a case series and reported that all 4 patients identified with persisting PSH had clinically important percentage weight loss with nutrition and hydration requirements markedly above their estimated requirement for slow weight gain, despite adjustment for brain injury and recommended the careful monitoring of nutrition, hydration and mineral supplementation.

The role of anthropometry in critical care patients is supported by the European Society for Parenteral and Enteral Nutrition[10] (ESPEN) who advise:

"a general clinical assessment should be performed to assess malnutrition in the critical care, until a specific tool has been validated. General clinical assessment could include anamnesis [Pt recollection], report of unintentional weight loss or decrease in physical performance before critical care admission, physical examination, general assessment of body composition, and muscle mass and strength, if possible. Such loss in muscle is associated with a prolonged hospital stay and interferes with quality of life and functional capacity. A definition of acute critical illness-associated malnutrition still needs to be developed"

Summary of Experience from Dietitians in Trauma Workshop 2019

70% of Dietitians asked who are working in Trauma reported that they had access to bed scales and 53% of the Dietitians reported that their patients are weighed on admission to critical care. 25% of the Dietitians advised that they use alternative anthropometric measures when undertaking nutritional assessments. Examples of alternative anthropometric measures could include ulna, mid upper arm circumference (MUAC), tricep skinfold and hand grip strength.

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5. Initiation of Enteral Feed

Emma Service

When to Start Feeding

Recommendation

Enteral feeding can safely be initiated in an individual with TBI within 24-48 hours of injury, once haemodynamic stability has been achieved.

Rationale

PEN[1] suggest although there is inconsistency in proving the benefit of early versus late feeding in TBI patients, it is generally considered safe to commence enteral nutrition (EN) within 24-48 hours of injury once the patient is haemodynamically stable. Delivering EN in a haemodynamically unstable patient requiring vasopressor support has been associated with non-occlusive bowel necrosis[2].

Initiating early EN versus delayed EN in critical care patients has been associated with a trend towards a reduction in mortality, reduced infectious complications and improved nutritional intake [3,4,5]. Starting nutritional therapy early in individuals with TBI can help preserve skeletal muscle mass, vital organ function and cerebral metabolic homeostasis[6].

Summary of Experience from Dietitians in Trauma Workshop 2019

Data collected from 15 dietitians working with critically ill TBI patients in the UK demonstrated that 100% of dietitians reported that all patients have EN started within 48 hours of admission to critical care, provided they are haemodynamically stable.

Refeeding risk

As TBI patients are admitted as an emergency, they are normally eating and drinking well prior to admission and are often low risk of refeeding syndrome. However, caution should be taken in those with a low BMI, weight loss and/or alcohol and substance misuse. High refeeding risk patients should start EN cautiously and have close monitoring.

For additional information and guidance please also refer to the Refeeding section of the PENG Pocket Guide to Clinical Nutrition[7].

Feeding Protocols

Recommendation

A robust feeding protocol should be available to the clinical critical care team that incorporates strategies to optimise delivery of EN. The protocol should be initiated as soon as it is deemed safe for the patient to start EN and should include guidance on type of feed, feeding rate, refeeding risk, management of gastric residual volumes (GRV) and post pyloric feeding. All TBI patients on EN should be seen by a specialist dietitian in a timely manner.

Rationale

Feeding protocols should be standard practice in the critical care setting and have been shown to improve the likelihood of nutritional goals being met and earlier commencement of EN[3,4,5.] However, caution should be taken as protocols do not allow for differences in patients, feed interruptions or clinical conditions. Use of feeding protocols are not sufficient to prevent nutritional deficits, therefore a specialist dietitian should provide an individualised nutritional support plan in those on critical care for longer than 48 hours[5.]

Summary of Experience from Dietitians in Trauma Workshop 2019

All 15 dietitians surveyed (as above in 'When to start feeding' section) report their units use a starter feeding protocol on critical care for TBI patients. Some dietitians note exclusions to the use of protocols including those at risk of refeeding, those on high dose inotropes and where EN is contraindicated. During working hours (Monday to Friday) 65% of the dietitians surveyed see their patients within 48 hours of admission, with 85% of those being seen within 24 hours.

Other considerations

Hypocaloric feeding

Due to endogenous energy production that occurs early in critical illness, overfeeding should be avoided. Permissive underfeeding may be beneficial. Hypocaloric EN with protein delivered to target does not confer major differences in outcome when compared to standard feeding[8,9,10].

Maximising EN delivery

International observational data shows only 58% of energy and 53% of protein requirements are met via EN in critically ill head injured patients predominantly due to feed interruptions[11]. Volume based feeding (VBF) can be considered to maximise delivery around interruptions to EN, adjusting the rate to meet targets[12,13]. Additionally, reducing fasting time for procedures can also improve EN delivery[14].

Further reading suggestion

For additional information and guidance please also refer to the critical care section of the PENG Pocket Guide to Clinical Nutrition[15.]

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6. Route of Feeding

Emma Service

Enteral Nutrition versus Parenteral Nutrition

Recommendation

Despite lack of consistent data on the optimal route of feeding in TBI patients, the use of enteral nutrition (EN) is generally recommended over parenteral nutrition (PN). However, where EN is contraindicated or does not meet nutritional requirements, exclusive or supplemental PN should be considered to optimise nutritional delivery. PN should be considered on a case-by-case basis.

Rationale

Poor quality studies in critically ill TBI patients specifically leave the question unanswered as to whether EN or PN is preferable[1]. However, international guidance in critical care patients suggests reduced cost and reductions in infectious morbidity favour the use of EN over PN[2,3,4].

ESPEN[4] suggest PN should not be started until all strategies to maximise EN tolerance have been attempted. Historically, it was suggested exclusive PN should be withheld for the first 7 days of admission in patients at low nutritional risk but commenced as soon as possible in patients with reasons to delay EN and at high nutritional risk[3,5]. More recently, ASPEN now suggest there is no significant difference in clinical outcomes between early exclusive EN or PN during the first week of critical illness. Neither route is superior to the other therefore either is acceptable[6].

Although EN is indicated in most cases, energy and protein targets can be difficult to achieve in practice, particularly in TBI patients with delayed gastric emptying (DGE) or those experiencing frequent interruptions to feeding. Supplemental PN should be considered to help meet nutritional targets on an individual basis[4] perhaps after other strategies such as prokinetics and post-pyloric feeding have failed. Optimal timing of when to start supplemental PN is not clear. It has been suggested starting 4 days after critical care admission could reduce nosocomial infections[7]. However, ASPEN have found no clinical benefit in providing SPN early in the critical care so recommend not initiating until after day 7[6].

Summary of Experience from Dietitians in Trauma Workshop 2019

Data collected from 15 dietitians working with critically ill TBI patients in the UK demonstrated 100% of dietitians reported PN is only used if EN is contraindicated or following the failure to establish EN. The reasons stated for EN failure were high gastric aspirates; ileus; the inability to pass a nasogastric tube (NGT) and the inability to pass a nasojejunal tube (NJT) in those with delayed gastric emptying (DGE). 36% of these dietitians noted that PN would not be started until after 7 days in those at low nutritional risk.

Gastric versus Post Pyloric Feeding

Recommendation

In practice, we suggest gastric feeding should be used as first line in TBI patients, unless contraindicated. Post-pyloric feeding is commonly associated with time delays and is considered less physiological when compared to gastric feeding.

Nonetheless, the post-pyloric route should be considered in patients at high risk of intolerance to gastric feeding; when gastric feeding has failed; or in those at high risk of aspiration.

Rationale

There is no consistent international guidance for the optimal route of enteral feeding in critical care patients. No research to date demonstrates a significant difference between the gastric and post-pyloric feeding routes in terms of patient mortality, ventilator days, or length of stay in critical care.

ESPEN and ASPEN recommend gastric access should be used as the standard approach to initiate EN[3,4]. Post-pyloric feeding is only recommended where gastric feeding intolerance is not resolved or for those at high risk of aspiration[3,4].

On the contrary, Canadian practice guidelines recommend the use of post-pyloric feeding over gastric feeding, if feasible, due to the reduced risk of pneumonia in critically ill patients[8].

PEN recommends initiating post-pyloric feeding for critically ill TBI patients. Post-pyloric feeding specifically in TBI patients has been associated with reduced incidence of ventilator-associated pneumonia and in the total number of complications[9]. Caution should however be taken with this suggestion, due to small sample sizes and blinding bias.

Despite recommendations for post-pyloric feeding in TBI patients, in practice it is difficult to establish at bedside without appropriate placement methods and expertise. Awaiting endoscopic placement of NJTs usually results in time delays to feeding. Bedside NJT placement can be useful to help facilitate the provision of EN, avoid long gaps in feeding and avoid PN. However, this requires training, protocols and engagement of medical and nursing staff[10,11].

Summary of Experience from Dietitians in Trauma Workshop 2019

All 15 dietitians surveyed (as above in 'EN v PN' section) reported that gastric feeding was the first line route for TBI patients. It was reported that only 28% of major trauma critical care units have the ability to place bedside NJTs. Others reported either relying on endoscopic placement or/and difficulty and delays in getting tubes passed.

Post-Pyloric feeding was reported to be used only for those with high gastric residual volumes despite prokinetics; persistent emesis and those at high risk of aspiration.

Feeding patients with a basal skull fracture

Recommendation

TBI patients with a suspected or confirmed basal skull fracture or complex maxillofacial injuries should be considered a contra-indication to blind insertion of a NGT initially. It is safer to pass an orogastric tube (OGT) in these patients. When and how a NGT is passed should be discussed with the neurosurgical team.

Rationale

Blind NGT insertion in those with suspected or confirmed basal skull fracture or complex maxillofacial injuries should be avoided to avoid intracranial placement[12,13]. An OGT is recommended in these patients and where a NGT is required it should be placed under direct vision or ideally delayed until imaging has confirmed it is safe to pass.

Summary of Experience from Dietitians in Trauma Workshop 2019

Dietitians working with critically ill TBI patients indicate that OGTs are used for confirmed or suspected basal skull fracture in TBI patients. The timeframe for changing to a NGT remains uncertain and is normally guided by the neurosurgeons, although dietitians have indicated an OGT is generally required for at least the first 10 days of critical care admission. Some patients then have the NGT placed blind at bedside and others have guided insertion, this is unit and team dependent.

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7. Energy Requirements

Karen Jackson, Rachel Ball

The following is a collection of guidelines to aid calculation of the energy needs of critical care patients with a TBI. The information provided below is for patients with an isolated TBI and may differ for trauma patients with more complex polytrauma injuries.

Clinical judgement is required when applying any of the energy calculations (indirect calorimetry, ventilator derived, predicative or weight-based equations) to your patient as their clinical condition changes. Under and overfeeding critical care patients results in worse outcomes including increased mortality and morbidity[1]. It is important to be aware that there is relatively little validation for ventilator derived, predictive and weight-based equations despite all being commonly used in practice. Indirect calorimetry (IC) is recommended as providing the best estimate of resting energy expenditure (REE) for ventilated and non-ventilated patients.

Mechanically ventilated non-obese critically Ill TBI adult patients

The energy needs of individuals with TBI are highly variable as the condition results in a hypermetabolic state that increases systemic and cerebral energy requirements. In the absence of indirect calorimetry, ventilator derived, predictive and weight-based equations may be used to guide estimation of energy calculations[2].

Recommendation 1

Although evidence is limited where IC is not available, REE using VO^2 or VCO^2 directly obtained from the ventilator has been shown to have greater accuracy than predictive or weight-based equations[3].

Indirect Calorimetry:
REE Day 1-3: provide up to 70% of MEE[3]
REE Day 3-7: 80-100% of MEE[3]

VCO^2 or VO^2 equation:

REE: VCO2 x 8.19
VO2 calculated from pulmonary artery catheter can also be used
Or:
REE: 3.941 * VCO2(L/min)/RQ + 1.106 * VCO2(L/min) * 1440

Alternatively, the Penn State 2003b equation has been shown to be the most accurate predictive equation for TBI patients to estimate REE in the absence of IC[2].

Step 1: Calculate the Mifflin-St. Jeor equation
For men: REE = (kg x 10) + (cm x 6.25) - (age x 5) + 5
For women: REE = (kg x 10) + (cm x 6.25) - (age x 5) -161

Step 2: Input into the Penn State 2003b equation
REE = (REE from Mifflin-St. Jeor x 0.96) + (VE [minute volume in L/min] x 31) + (Tmax [maximum body temperature in last 24hrs] x 167) – 6212

Where this not possible, the following weight-based equations may be used as a baseline energy measurement:

ESPEN[4]: 20-25kcal/kg/day

ASPEN[5]: 12-25kcal/kg during initial 7-10 days of critical care admission

Jacobs[6]: Patients with moderate-severe injury: 25-30kcal/kg/day

or 120% to 140% of predicted REE (as measured by Harris-Benedict equation).

(A recommendation for general trauma patients and not specific to TBI patients).

Severe head injury (GCS score <8) 30kcal/kg/d (~140% of MEE) in non-pharmacologically paralyzed patients.

Rationale

Baseline-predicted REE in TBI patients can range from 100-200% (based on Harris-Benedict equation) and is influenced by changes in metabolic function related to increased oxygen consumption because of changes in stress hormones. Hyperventilation, fever, seizures, posturing and the use of paralytics, barbiturates, and/or coma-inducing agents in early management can either increase or decrease energy expenditure[7].

Given the degree of variability in REE, The Penn Nutrition TBI knowledge pathway[2] suggests that there is insufficient evidence to recommend the use of predictive or weight-based equations for determining REE in critically ill patients and advise using IC. ASPEN (2016)[8] and ESPEN (2019)[4] likewise recommend IC as the gold standard method for calculating energy requirements.

When IC is not available, ventilator-derived carbon dioxide consumption (VCO^2) has been proposed. Stapel et al (2015)[3] compared using this method to IC and concluded that this method was more accurate and precise than the frequently used predicative equations. Large errors of inaccuracy did not occur, and the inaccuracy rate was significantly lower than that of predictive equations. However, limited equations were included within the comparison (Penn State equation and ESPEN 25kcal/kg) and there is limited evidence to support the use of this method.

Koekkoek et al (2020)[1] also compared VCO^2 and IC to measure REE in their small prospective study but found significant bias, low precision and low accuracy rates. They concluded that although predictive equations are inaccurate, they may predict energy expenditure better than the VCO^2 method. This method may become more accurate as and when new techniques for CO^2 breath measurement are developed.

There has been several studies[9,10] demonstrating the poor accuracy rates of REE using predicative and weight-based equations when compared to measured energy expenditure (MEE), with reported accuracy of only 35 to 45%. If used in the absence of IC, careful decision-making is required to select the most appropriate predictive or weight-based equation dependent on the patient's clinical condition, time in the patient's critical illness journey and the accuracy of factors required to calculate the equation.

Penn[2] recommend using the modified Penn State equation (2003b) as providing the most accurate estimate of REE in individuals with TBI. Nonetheless, this equation is associated with a +/- 15% error in 15% of individuals and requires additional external validation to confirm this observation.

Walker and Hauberger (2009)[11] demonstrated that using the modified Penn State equation had the smallest margin of error for a general critical care population: within 10% of the measured REE in 71% of occasions measured. However, it is important to note that 15% of individuals had REE values greater or less than 15% of measured REE.

Frankenfield and Ashcraft (2012)[12] concluded that the modified Penn State equation predicted resting metabolic rate accurately 72% of the time in a mixed cohort of TBI and stroke patients. However, they did not use any other equations as comparison, and this has not been externally validated.

Morbitzer et al[13] demonstrated that the Penn State equation had the weakest correlation with measured energy expenditure in the TBI population underestimating REE.

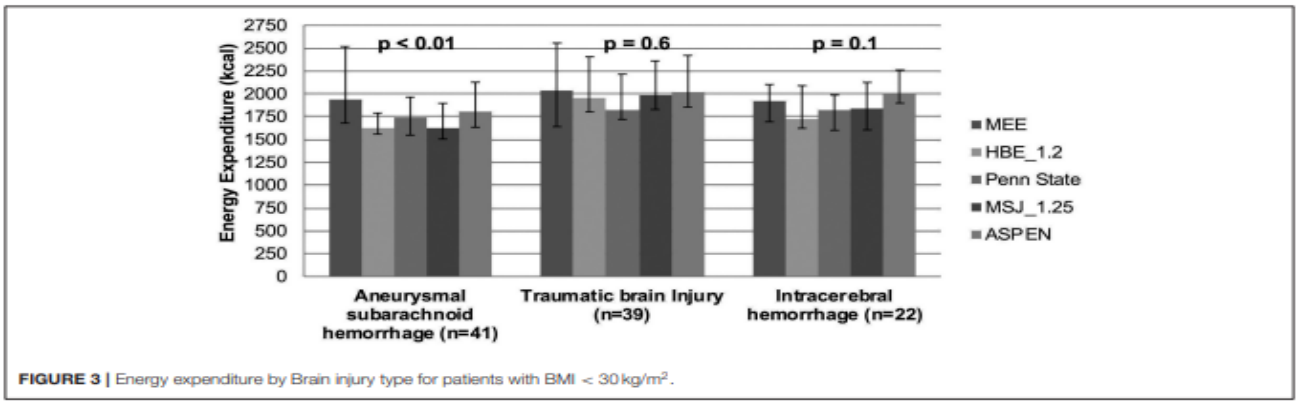

FIGURE 3 | Energy expenditure by Brain injury type for patients with BMI < 30 kg/m^2.

Replicated from Orbiteer et al[13]

It is of note that dietitians specifically working with patients with TBI report rapid and significant weight loss using any of the above equations which result in a low calorie/kg/body weight provision. In everyday practise the provision of a high calorie/kg/body weight feeding prescription (as recommended by Jacob[6]) is adopted to prevent this deterioration.

Mechanically ventilated obese critically Ill TBI adult patients (BMI >30kg/m^2)

Estimating REE in an obese patient presents a significant challenge not only on account of the comorbidities and metabolic anomalies associated with obesity but deciding upon the optimal dose of enteral nutrition to avoid the harms of overfeeding which are even greater than in the normal weight patient.

A systematic review by Kee et al (2012)[14] reported that critically ill obese patients with a BMI >30kg/m^2 compared to those with a BMI <30kg/m^2, had a greater risk of experiencing malnutrition due to clinician's misinterpretation of obese patients having adequate nutrition stores secondary to high BMIs. Critically ill obese trauma patients are less capable of mobilizing fat stores than their lean counterparts[15].

As with non-obese patients, the REE of individual obese patients is associated with high variability and monitoring for under- and overfeeding is essential.

Recommendation 2

Energy provision for obese patients should not exceed 65-70% of target calculated by IC[8]. Hypocaloric feeding is a general recommendation as for obese critically ill patients as there is no specific guidelines for obese critically ill TBI patients.

Rationale

IC is recommended for calculating energy requirements as ventilator derived, predictive and weight-based equations are less accurate than in non-obese patients[2,4,8].

Where IC is not available. The following predictive equations may be used to calculate REE:

Obese Adults <60years old: Use Penn State 2003b

For men: REE = (kg x 10) + (cm x 6.25) - (age x 5) + 5
For women: REE = (kg x 10) + (cm x 6.25) - (age x 5) -161

(REE from Mifflin-St. Jeor x 0.96) + (VE [minute volume in L/min] x 31) +
(Tmax [maximum body temperature in last 24 hrs] x 167) – 6212

Obese adults >60 years old: Use Modified Penn State 2011

For men: (wt [kg] x 10) + (ht [cm] x 6.25) – (age x 5) + 5
For women: (wt [kg] x 10) + (ht [cm] x 6.25) – (age x 5) -161

REE = (Mifflin x 0.71) + (Tmax x 85) + (Ve x 64) - 3085

Alternatively, ASPEN[8] guidelines suggest using the following weight-based equations:

Patients with a BMI 30-50kg/m^2 11-14kcal/kg actual body weight

Patients with a BMI >50kg/m^2: 22-25kcal/kg of ideal body weight

(IBW – calculated as recommendation 4)[8]

These equations have been validated and are reasonable predictors of 65% measured REE[16].

ESPEN[4] recommend using adjusted body weight to calculate REE when IC is not available:

Adjusted body weight: (actual body weight - ideal body weight) x 0.33 + ideal body weight

Up to the publication of this handbook, there were no clinical trials to evaluate different energy intakes and outcomes in critically ill adults with obesity. Data from observational studies recommend hypocaloric high protein feeding (50-70% of target energy requirements) to decrease the risk of co-morbidities[8]. However, this recommendation is controversial due to a lack of randomized, high-quality studies to support any benefits of hypocaloric feeding in this population.

A prospective cohort study[17] comparing IC with weight-based equations in an ICU population of patients with BMI >30kg/m^2 found weight-based equations underestimated REE. The study was a small study, the authors only used weight-based equations as recommended by ASPEN[8], the patients were at low nutritional risk and at a relatively low severity of illness.

Frankenfield et al (2012)[18] reviewed the accuracy of predictive equations in the extremes of BMI and found accuracy (percentage of estimates falling within 10% of measured REE) in the morbidly obese group was highest for the modified Penn State equation (76%). They recommend using this equation for obese critically ill patients with a BMI >30kg/m^2 and over 60 years old. The equation validated by Frankenfield in a critical care population with 18% trauma patients, found the equation to be accurate in 74% of occasions measured; however, in those with a BMI > 40kg/m^2, the accuracy was reduced to 68%.

Although a degree of underfeeding may be beneficial in this population, provision of adequate nutrient intake to prevent malnutrition and loss of lean body mass is also important. Improved outcomes such as decreased length of stay have been associated with hypocaloric feeding of obese critically ill patients but an association between negative energy balance and increased infections has also been reported.

Mechanically ventilated underweight critically ill adult patients

Assessing the energy requirements of underweight critically ill patients is as challenging as at the lower extremes as at the upper extremes of body weight. The malnutrition status of critically ill patients is a strong predictor of mortality. A large body of evidence exists associating malnutrition (when BMI <20kg/m^2) with increased mortality. There are currently no specific energy recommendations for the underweight critically ill patient.

Recommendation 3

In the absence of IC, ventilator derived, predictive and weight-based equations can be used to calculate energy requirements but may require further modification given the importance of matching energy intake with the needs of the severely underweight patient.

Rationale

Ahmed et al (1999)[19] demonstrated in a small prospective trial of underweight patients (body weight of <50kg) that commonly employed formulae often underestimated REE when comparing to measured energy expenditure (MEE) using IC. Although this trial was in non-critically ill patients, the study used the same weight-based equations that are frequently used in critical care. The authors suggested using an empirical equation of 30-32 kcal/kg.

In a population of 42 critically ill underweight patients, Campbell et al (2005)[20] evaluated that prediction equations either significantly over- or underestimated energy needs compared to IC. The study highlighted the limitations of using prediction equations in this patient group.

Frankenfield, Ashcroft and Galvan (2013)[21] reviewed the accuracy of the Penn State equation (2003b) for the underweight group (<20.5kg/m^2), and found it was accurate 63% of the time. However, below a BMI of 20.5kg/m^2, the accuracy rate dropped to 58%.

ASPEN guidelines (2016)[8] recommended aiming >80% of estimated energy needs by 48–72 hours in patients with high nutrition risk. In the updated ASPEN guidelines[5], the recommendation changed to make no distinction between energy provisions based on patient's nutritional risk. The recommendation changed following no emerging RCT evidence to support or refute the original recommendation and no difference in outcomes based on high versus low energy provision.

How to calculate ideal body weight

Recommendation 4

Ideal body weight (IBW) can be calculated by using either of the following methods:

Calculating IBW
As per recommendation 4 in Energy Requirements (refer to the relevant section for evidence):

Ideal body weight (IBW) can be calculated by using either of the following methods:
Hamwi:
Women: IBW (kg) = 45.5 + 2.3 x (height - 60in)
Men: IBW (kg) = 50.0 + 2.3 x (height - 60in)
ESPEN:
IBW (kg) = 2.2 x BMI + 3.5 x BMI x (height – 1.5m)

Rationale

The ESPEN[4] equation is referenced from Peterson et al (2016)[23] and can be used to calculate either BMI or body weight at any target BMI value. The authors of the study claim a mean empirical accuracy of 0.5-0.7% and the equations aligns with BMI values for both women and men and avoids over- and underestimation issues at the upper and lower extremities of the height spectrum.

It has been argued that the use of IBW is justified on the grounds that lean body mass can change significantly during critical illness. Therefore, IBW may be more accurate than actual body weight. However, in practice there is a huge variation in which weight is used: adjusted body weight, ideal body weight, or actual weight[11].

Calorie provision initially post injury

Recommendation 5

Optimal nutrition support in critical illness continues to be a matter of debate, whether to provide trophic feeding or full nutrition in the early phase of critical illness is controversial as both under- and overfeeding can be deleterious in critically unwell patients.

Current recommendations post critical care admission suggest initial energy provision should be 70-80% of MEE requirements[4,8]. Delivery can be increased to 80-100% after day 3-7 for IC calculations, or day 7 for those using alternative methods[3].

Rationale

Hypercaloric feeding or overfeeding is defined as providing too many calories (>110% of the calculated or measured energy target). To avoid overfeeding, hypocaloric feeding or permissive underfeeding in low nutritional risk patients should be considered in the early phase of critical illness.

Several trials[24,25,26] have demonstrated that early full energy provision has poorer patient outcomes resulting in increased length of stay, ventilation duration, infection rates and critical care and hospital mortality. This may possibly be due to the metabolic consequences of lower nutritional provision. Recommendations suggest targeting actual energy expenditure during the initial 72 hours of acute illness should be avoided. Coupled with endogenous energy production (possibly amounting to between 500-1400kcal/day), full early feeding could result in overfeeding[27].

An alternative hypothesis has been suggested why feeding to goal may be harmful in the early phase of critical illness. Enteral nutrition's impact on autophagy has been suggested as a possible mechanism for this harm. Autophagy is a cellular repair process that plays a role in both homeostasis and disease recovery. Autophagy is inhibited by increased insulin, glucose, and nutrients. Consequently, disruption of autophagy by enteral nutrition could be a mechanism of harm[28].

Several trials[29,30] with permissive underfeeding have shown a reduction in hospital mortality. However, a follow up systematic review and meta-analysis[31] and post hoc-analysis[32] by the same authors could not confer any difference in outcomes between normocaloric versus hypocaloric diets in critically ill patients. Rice et al[33] in a randomised open label trial concluded that trophic enteral feeding in the first six days of ventilation resulted in similar clinical outcomes as early full-energy enteral nutrition.

It is of note that in most hypocaloric studies both the intervention and full feeding groups frequently receive lower than the recommended caloric intake, and both often groups represent hypocaloric groups. Dependent on the trial, high energy provision has ranged from 17-30kcal/kg/day whilst low energy 5-9kcal/kg/day or 200-1100kcal/day making it difficult to ascertain what is high or low energy provision.

Currently, there has been no clear benefit demonstrated between hypocaloric versus isocaloric nutrition in the early phase of critical illness. Singer et al[34] (TICACOS trial using MEE) demonstrated that patients in the IC group that achieved higher mean energy intakes were associated with a trend towards improved hospital mortality (P=0.01). More recent meta-analyses have shown no difference between normocaloric versus hypocaloric energy provision in critically ill patients[35.]

The ASPEN[5] guideline analysis of clinical trials ranging from 2001-2020 of early high energy versus early low energy provision examined several RCTs to determine impact on clinical outcomes. No significant differences were found between the higher versus lower energy intake groups for several clinical outcomes to include risk of bacteraemia (P=0.67), risk of infection (P=0.84), mean ICU length of stay (P=0.65), mean ventilator days (P=0.90) and ICU mortality (P=0.25) amongst other outcomes.
The trials included in their review were for the general critical care population and not specific to critical care patients with TBI.

A prospective study[36] specifically in TBI critically ill patients showed a significant relationship between the amount of early nutrition therapy provided and the risk of death. Optimal energy and protein intake predicted the mortality risk after two weeks, with a 30%–40% decrease in mortality for every 10kcal/kg/day increase in energy intake, reaching a plateau at approximately 25 kcal/kg/day.

It is important to have a good understanding of the actual feed delivery to your patients on your critical care unit. Many nutritional trials fail to achieve their target nutrition goals and this often materialises in everyday practise. Regular monitoring and nutrition audits are recommended to ensure the patient's nutritional goals are being achieved.

Recommendation 5 does not apply to patients at high nutritional risk.

Calories from non-nutritional sources

Recommendation 6

Non-nutritional calories (NNC) should be considered when calculating energy provision to avoid calorie overload and a positive energy balance[1].

Rationale

Non-nutritional calories can result in a large calorie overload and overfeeding. Weijs et al[26] reported in a study among an ICU patient population extensive and frequent overfeeding from NNC.

Overfeeding is a potential cause of multiple metabolic disorders i.e., hypertriglyceridemia, hyperglycaemia, hepatic steatosis, and infectious morbidity. Sources of NNC include glucose/dextrose infusion, sedative agents i.e. propofol, trisodium citrate used in continuous veno-venous hemodiafiltration (CVVH) and acetates.

Sources of fat intake should be checked to ensure the daily fat intake is not exceeded to prevent fat overload. Patients with TBI and multiple trauma can receive significantly higher doses of propofol. It is important to be aware of the fat provision between a 1% and 2% propofol solution (a 1% solution cumulatively can deliver double the amount of fat).

Exceeding a cumulative glucose intake should be avoided to prevent de novo lipogenesis[37], hyperglycaemia, enhanced CO^2 production, and increased insulin requirements[4]. For all critical care patients, the amount of glucose or carbohydrates should not exceed 5mg/kg/minute[4].

Source of NNC	Calorie Provision	Example	Ingredients
Propofol 1% & 2%	1.1kcal/ml	20mlsx1.1x24hrs=528kcals	Soya bean oil, purified egg phospholipid, glycerol, sodium hydroxide
Dextrose 5%/10%/20%/50%	4kcal/g[25]	1L 5% dextrose (5g per ml) 4x5x10 = 200kcal 1L 10% dextrose (10g per ml) 4x10x10 = 400kcal 1L 20% dextrose (20g per ml) 4x20x10 = 800kcal 1L 50% dextrose (50g per ml) 4x50x10 = 2000kcal	

The caloric load from citrate dialysis is more difficult to determine as it depends on several factors: the concentration of trisodium citrate, the infusion rate, the blood flow in the filter and the filtration fraction into the ultrafiltrate over time. Bousie et al[38] analysed the impact of NNC calories in mechanically ventilated critically ill patients, noted a significant difference between the total NNCs received for patients on citrate (CVVH) versus those not requiring CVVH (P0.001). CVVVH may continue over a period of days as the patient develops acute kidney injury, oliguria, or complications of renal insufficiency after ICU admission.

There can be a large variability in NNC delivered to individual patients and underlines the importance of individual patient monitoring for the most relevant sources of NNCs.

Non-Mechanically ventilated TBI patients

Recommendation 7

As with ventilated patients, the energy needs of individuals with TBI in non-ventilated patients is highly variable.

Rationale

REE for non- ventilated patients should be calculated using indirect calorimetry (IC). In the absence of IC, weight-based or predictive equations can be used. However, these equations are not specifically for TBI patients. Alternatively, PENG have produced energy recommendations expressly for the TBI population.

The following energy calculations maybe used in self ventilating patients:

Ireton-Jones:
IJEE= 629 – 11 X A + 25 X W - 609 X O
(A = age; W = weight; O = obesity (1 for present, 0 for absent))

Modified Penn State:
REE (kcal/day) = weight in kg (20) – age in years (3) + male sex (197) + body mass index in kg/m^2 (25.9) + mean heart rate in beats/min (9.4) + 89

ESPEN[4]:
20-25kcal/kg/day

ASPEN[5]:
12-25kcal/kg/day initial 7-10 days

PENG:
BMI 18.5-29.9kg/m^2
REE x PAL: 29/kcal/kg or 24kcal/kg (rehab patients)

BMI > 30kg/m^2
REE (using Mifflin-St Jeor) x PAL
Mifflin-St Jeor:
For men: REE= (kg x 10) + (cm x 6.25) - (age x 5) + 5
For women: REE= (kg x 10) + (cm x 6.25) - (age x 5) -161

PAL	Description
1.00-1.10	In bed and immobile
1.10-1.20	In bed and/or sitting out
1.20-1.25	Limited mobility
1.25-1.40	Sedentary

Of the predictive equations that are available in the spontaneously breathing population, the modified Penn State equation has been shown to be the most accurate if BMI is ≥20.5kg/m^2; accurate in 71% of patients compared with 44% for Ireton-Jones and 42% for Mifflin-St Jeor[39]. However, this equation has not been validated and should not be used at present.

Summary of Experience from Dietitians in Trauma Workshop 2019

The questionnaire was completed by attendees provided some oversight into the practices of estimating energy requirements from dietitians in working with TBI patients in ICU.

No ICU dietitians participating in the questionnaire had access to IC and predominantly used either predictive equations or weight-based equations to estimate REE.

Table: Summary of energy calculations used by dietitians for Critical Care TBI patients

	Penn State	Mifflin St Joer	Ireton Jones	ASPEN/ESPEN	Henry (Oxford)	Other
Energy Calculation used:						
Ventilated patient BMI < 25kg/m^2 Day 1-7	41%	11%	3%	34%	11%	
Ventilated patient BMI < 25kg/m^2 Day 7 onwards	49%	10%	6%	25%	10%	
Ventilated patient BMI 25-30kg/m^2 Day 1-7	46%	13%	0%	33%	8%	
Ventilated patient BMI 25-30kg/m^2 Day 7 onwards	50%	10%	5%	25%	10%	
Ventilated patient BMI >30kg/m^2 Day 1-7	48%	12%	4%	20%	4%	12%
Ventilated patient BMI >30kg/m^2 Day 7 onwards	52%	10%	5%	19%	0%	14%
All dietitians accounted for non-nutritional calorie provision in their energy provision						

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8. Protein Requirements

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The following recommendations are taken from clinical guidelines from specialist societies to aid the calculation of protein requirements for TBI patients in critical care.

Introduction

The metabolic response to acute TBI is marked by excessive catabolism with the rapid breakdown of lean body tissue to liberate substrates for energy metabolism. Coupled with decreased protein synthesis resulting from subsequent immobility as well as documented challenges in nutrient delivery, TBI patients are at significant risk of subsequent protein-energy malnutrition[1,2].

It is suggested that approximately 16% of total body protein is lost in the first 21 days of admission, with 67% of that protein loss coming from skeletal muscle alone[3]. Whilst nutrition support cannot completely eliminate this muscle wasting, recent studies in critical illness suggest that provision of protein is more closely linked to positive outcomes than the provision of total energy; with higher protein intakes being associated with improved nitrogen balance, Glasgow Coma Score (GCS), decreased length of stay (LOS) and reduced infection rates[4,5]. Determination of individual protein requirements for TBI is difficult, as the extent of hypermetabolism and catabolism is largely dependent on the severity of the head injury as well as the ventilation status, sedation, and presence of posturing[6].

Protein needs are most frequently assessed in the literature using nitrogen balance studies, but there is a significant lack of large, randomized prospective trials which relate specifically to clinical outcomes in critically ill patients, and TBI specifically. ASPEN (2016)[7] notes the use of nitrogen balance or non-protein calorie to nitrogen ratio (NPC: N) (70:1–100:1) is of limited value in the critically ill patient[8] and that the use of serum protein markers (albumin, prealbumin, transferrin, CRP) to determine the adequacy of protein provision should not be used, as they have not been validated in critical care.

As such weight-based equations may be used to monitor the adequacy of protein provision by comparing the amount of protein delivered with that prescribed, but it is important to remember that patients with a TBI frequently present with other injuries as well and therefore calculated requirements need to account for the heterogeneity of this population group.

Recommendation 1

Patients with a TBI admitted to ITU should be provided with 1.5-2.5g protein/ per kg actual body weight/ per day if BMI is 20-30kg/m^2

Rationale

There is a lack of high-quality evidence which relates to protein requirements specific to the TBI population and recommendations vary greatly throughout the literature.

ASPEN 2016[7] and PEN nutrition[9] recommend a protein range of 1.5-2.5g protein/per kg actual body weight/per day for critically ill TBI patients, acknowledging the higher values for patients with concurrent injuries e.g. burns and polytrauma.

Both sources cite Dickerson et al (2012)[5]; a prognostic study which aimed to determine the amount of protein required to achieve nitrogen equilibrium or a positive nitrogen balance with specific considerations given to TBI. Despite a noted variability in results, relating to individual differences in protein catabolism, there was a general trend toward improved nitrogen balances with higher protein intakes. This was reflected in 54% of participants achieving nitrogen equilibrium or positive nitrogen balance when administered 2g protein/per kg actual body weight/per day or greater, compared with 38% for those who

received 1.5-1.99g protein/per kg actual body weight/per day and 29% for those who received 1-1.49g protein/per kg actual body weight/per day ($p < 0.001$).

There was no significant difference in nitrogen balance between patients with and without TBI at similar protein intakes, but it must be noted that the study only evaluated a single point within the first 5 days to 14 days post-injury. Several authors have shown that nitrogen excretion increases for up to 4 weeks post TBI[10-15] and therefore the single reading of nitrogen balance in this study, may not reflect the overall picture during admission.

Dickerson et al (2012)[5] also determined that protein supplementation above 2.5g protein/per kg actual body weight/per day showed minimal clinical benefit, with ureagenesis being greater when protein intake exceeded this amount. It does however suggest that in the absence of renal or hepatic failure, short-term administration of higher protein doses is unlikely to cause serious adverse effects. This finding is corroborated by Hoffer and Bistrian (2012)[16] who conducted a systematic review of the clinical literature pertinent to the optimum and safe upper limit of protein provision in adult critical illness. The review strongly suggests that 2.0-2.5g protein/per kg actual body weight/per day (BMI <30kg/m^2) is safe and could be optimum for most critically ill patients; based on nitrogen balance and protein turnover improving with increasing protein levels up to the highest level studied (2.5g protein/ per kg actual body weight/per day). Dickerson et al (2012)[5] conclude by highlighting the individual nature of protein requirements secondary to differences in catabolic response to trauma for each patient and recommends the pertinence of close monitoring.

ESPEN[17] cites Dickerson et al (2017)[18] acknowledging that higher protein intakes reaching 1.5-2g protein/per kg actual body weight/per day may be considered in head trauma (due to the large protein losses), but it does not go so far as to make a recommendation of protein requirements in TBI, stating that most of the smaller studies referenced in the literature are not comparable in terms of patient selection, calorie and protein intake, timing and route of administration and therefore valid and reliable conclusions cannot be drawn.

The Canadian Critical Care Practice guidelines[19] acknowledge several smaller studies[20-23] which demonstrate improved outcomes for critically ill patients receiving higher protein diets but note that these studies are not comparable in their outcomes, and they are not all specific to TBI. They conclude that further randomised trials are required to make strong conclusions about the dose of protein in critically ill patients.

The disparity in evidence and the contradictory findings that currently relate to protein requirements for TBI patients highlight the urgent need for more well-designed clinical trials to identify protein requirements in this patient population.

Recommendation 2

Patients with a BMI of 30-40kg/m^2 should be provided with 2.0g protein/per kg ideal body weight (IBW)/per day and patients with a BMI >40kg/m^2 should be provided with up to 2.5g protein/per kg ideal body weight (IBW)/per day

Calculating IBW
As per recommendation 4 in Energy Requirements (refer to the relevant section for evidence):

Ideal body weight (IBW) can be calculated by using either of the following methods:

Hamwi:
Women: IBW (kg) = 45.5 + 2.3 x (height - 60in)
Men: IBW (kg) = 50.0 + 2.3 x (height - 60in)

ESPEN:
IBW (kg) = 2.2 x BMI + 3.5 x BMI x (height – 1.5m)

Rationale

There are no specific guidelines for protein requirements in the obese, critically ill traumatic brain injury (TBI) patient and limited high-quality evidence which relates specifically to protein requirements for the obese, critically ill patient.

From the available clinical guidelines from speciality societies, it is only ASPEN (2016)[7] that makes specific recommendations for obese critically ill patients based on evidence from observational studies and expert consensus, but this is not specific to patients with a TBI. ASPEN (2016)[7] suggests using hypocaloric, high protein feeding to minimise losses in lean body mass, support in mobilising adipose stores and reduce the risk of overfeeding and its associated complications. In the absence of requirements specific to TBI and obesity, it is most appropriate to use the available evidence specific to obese critically ill patients.

It is well understood that obese patients have a greater number of underlying co-morbidities than their lean counterparts and are therefore considered at an increased risk of complications. Despite an often-presumed excess energy store, an early small-scale study of 17 trauma patients (7 obese; 10 non-obese) by Jeevanandam et al (1991)[24] suggested that the obese patient mobilised relatively more protein and less fat when compared with non-obese subjects; meaning a higher percentage of energy needs were achieved from protein metabolism rather than fat metabolism in the obese cohort. Whilst it may be considered that some degree of weight loss in obese patients is beneficial in terms of increasing insulin sensitivity, reducing the risk of comorbidities and supporting nursing care; this needs to be carefully considered against the risk of exacerbating protein deficits and sarcopenia commonly seen in the obese population. Moisey et al (2013)[25] demonstrated that the presence of sarcopenia was associated with worsened outcomes in a study of 149 elderly trauma patients (47% of the cohort were obese), with mortality rates higher in those who were sarcopenic (32% vs 14%). The study also found that the presence of sarcopenia was associated with greater ventilator and critical care days.

The idea of hypocaloric high protein feeding for the obese individual has resulted from several studies which show the possibility of achieving a positive nitrogen balance without overfeeding calories. Both Choban et al (2013)[26] and Dickerson et al (2017)[18] cite the same studies, 2 RCTs[27,28], and a small number of observational studies[29-32] which use hypocaloric, high protein regimens for obese, hospitalised patients (but not necessarily critically ill). Average protein intakes in these studies ranged from 90 to 140g per day, from a range of PN and EN (or both) sources. Differences in results across the studies ranged from significant improvements in clinical outcomes (LOS, antibiotic therapy, and days of mechanical ventilation) to positive clinical outcomes, to no difference in outcomes (mortality, LOS). It must be noted that outcome measures were not consistent between the studies, and they were not of sufficient statistical power or quality to determine a specific amount of protein provision as optimal regarding nitrogen balance.

It is important to note that a study by Alberda et al (2009)[20] cited in Choban et al 2013[26] demonstrated that lower protein intakes in combination with hypocaloric feeding in the obese class II (BMI of 35–39.9kg/m2) cohort, were associated with a greater mortality rate. This is considered ‘permissive underfeeding’ and should not be confused with hypocaloric, high-protein feeding, where optimal protein intakes are preserved.

Based on the evidence that provision of protein at a dose of 2.0g protein/per kg IBW/per day was insufficient in achieving neutral nitrogen balance when BMI is greater than 40kg/m^2 and that clinical outcomes are at least equivalent in obese patients receiving hypocaloric, high protein feeding when compared with eucaloric, high-protein feeding[26,28,29]; both Choban et al (2013)[26] and Dickerson et al (2017)[18] conclude that protein provision of 2.0–2.5g protein /per kg IBW/per day should guide protein requirements as sufficient to achieve net protein anabolism. ASPEN (2016)[7] advises that these protein recommendations are adjusted in line with nitrogen balance studies to achieve nitrogen equilibrium if possible.

The PEN Nutrition Knowledge Pathway[33] for protein requirements in obese critically ill patients makes the same recommendation for hypocalorically fed obese patients (in the absence of severe renal or hepatic dysfunction) of 1.2g protein/per kg actual body weight/per day or 2.0-2.5g protein/kg IBW/ per day, to

achieve nitrogen balance during underfeeding. PEN cites the observational studies from the ASPEN (2016)[7] guidelines but notes specifically that in a systematic review by Hoffer and Bistrian (2012)[16] which was conducted to determine safe upper limits of protein intake in critically ill patients, none of the studies included was conducted specifically to determine protein requirements in obese critically ill patients.

The PEN Nutrition Knowledge Pathway[33] suggest the adequacy of provision of protein can be monitored by the frequent collection of 24- hour urine specimens for urinary urea nitrogen (UUN), and the calculation of nitrogen balance is as follows:

Nitrogen balance (g/day) = (24 hours protein intake/6.25) – [Total urea nitrogen (gm) + 2 gm]
OR
Nitrogen balance (g/day) = (24 hours protein intake/6.25) – [Urinary urea nitrogen (gm) + 4 gm]

It is however important to note that due to the catabolic and metabolic processes following acute critical illness, a positive nitrogen balance and protein anabolism is very difficult to achieve.

In contrast to ASPEN (2016) [7] and The PEN Nutrition Knowledge Pathway[33], ESPEN (2019) guidelines[17] recommend that protein delivery should be 'guided by urinary nitrogen losses or lean body mass determination (using CT or other tools)' or that if these methods are not available then protein intake can be calculated using 1.3g protein/per kg adjusted body weight/per day. They acknowledge the dated and small-scale nature of the currently available evidence as summarised in Dickerson et al, (2017)[18], noting that many of the trials were performed more than 20 years ago in less than 50 patients. The authors also raise concerns that using BMI alone as a classification for obesity could lead to a heterogeneous group on the basis that BMI doesn't differentiate for body composition, leading to extremes of muscle mass within the same BMI category. It is not clear if nitrogen loss is higher in patients who are overweight and obese compared to 'healthy' weight patients when adjusted for actual lean body mass.

ESPEN (2019)[17] advise that adjusted body weight is calculated as:

(actual body weight - ideal body weight) x 0.33 + ideal body weight.

Conversely, ASPEN (2016)[7] does not recommend using an adjusted body weight due to a lack of validation studies and a variable definition in the literature[34].

Again, recommendations throughout the literature are both limited and conflicting, thus highlighting the imperative need for further high calibre studies across the critical care setting and specific to the TBI population.

Other considerations

Underweight Populations

Whilst it is noted that predictive equations are less accurate in underweight patients (BMI <20kg/m^2), there are currently no specific recommendations for protein requirements in underweight critically ill patients. As per PENG Requirements Guideline Group Consensus Opinion (2018)[35] it is recommended to start at 1.5g protein /per kg actual body weight/per day and monitor regularly.

Timing

The optimal timing of protein intake is unclear and there are no specific recommendations to guide practice. Whilst some studies[36,37] have demonstrated improved survival from early higher protein intakes (e.g. >1-1.2g protein/kg/day by day 3-4) another study[38] found that a larger amount of protein administered during days three to five was associated with higher mortality. It is likely optimal protein targets will change over the course of a critical care admission and therefore high protein intake is only

beneficial if not associated with overfeeding. In line with energy targets, it seems sensible to increase protein intake to the goal over the first 7–10 days of critical stay (see energy section for evidence).

Feed Delivery

It is important to consider wider factors that can impact protein provision in TBI including delayed gastric emptying/intolerance, fasting for procedures and tube displacement. An observational study conducted by Chapple et al (2016)[39] evaluated the nutrition support practices in 1,045 patients with TBI from 341 different critical care units internationally. The study demonstrated that nutritional deficits were commonplace in the TBI population, with patients on average receiving just 53% of the protein requirements in the first 12 days of admission post-injury. Whilst there was no association between energy and protein intake and mortality, a greater deficit was associated with a greater length of stay both in critical care and the wider admission.

Anthropometrics

Another important point to consider is that equations that incorporate a patient's weight are reliant on accurate weights being obtained, and this can be a challenge for patients within the critical care setting. Ultrasound is emerging as a tool to measure muscle mass and monitor changes in muscle tissue and it can be relatively easily conducted at the bedside of a critical care patient. It is possible to use Computed tomography (CT) scanning to obtain an accurate quantification of skeletal muscle and adipose tissue depots. However, this is unlikely to be used routinely in practice due to its exposure risk and cost[7].

Continuous Renal Replacement Therapy (CRRT)

For patients requiring CRRT relevant renal guidelines should be followed[40,7,41]. It is suggested that protein requirements may be increased up to a maximum of 2.5g protein/per kg actual body weight/ per day[42]. There is no guidance specific to the obese population currently.

CKD/AKI

For patients with CKD/AKI refer to the renal section in PENG 2018[35] to guide protein requirements as caution is advised in this cohort.

Exercise Programmes

There is some evidence that suggests outcomes are improved when increased protein intakes are coupled with increased physical activity, but both ASPEN (2016)[7] and ESPEN (2019)[17] agree that further evidence is required before recommendations for specific exercise programmes can be made.

Updated literature

The updated ASPEN 2022 critical care guidelines[43] reviewed subsequent literature from 2015-2018[44-47] with a focus on patient outcomes at higher and lower protein intakes. Again, there was a paucity of high-quality evidence, owing to varying study designs and their targeted outcomes, and therefore no updated recommendations for protein provision have been made at this time. There were no trials specifically in patients with burns, obesity, or multi-trauma which met the inclusion criteria.

Upcoming Research

We await the results of the EFFORT Trial[48] (The Effect of higher protein dosing in critically ill patients) designed as a large multi-centre, international RCT. It aims to investigate the effect of prescribing a higher dose (≥2.2g/kg/day) of protein /amino acids compared to a low dose (≤1.2g/kg/day) on clinical outcomes in critically ill patients considered at high nutritional risk. Once complete, the study will be one of the largest of its kind and will be a significant addition to the literature to inform and guide health care practices in the future.

Several other clinical trials are also currently testing higher versus lower protein doses in critically ill patients. These are listed in the ASPEN 2022 guidelines[43] supporting appendix. With the publication of these studies, it is hopeful there will be a greater amount of high-quality evidence to better inform optimal protein provision and consequently help to improve the outcomes of patients who are critically ill.

Summary of experience from Dietitians in Trauma Workshop 2019

A questionnaire was completed by attendees to provide insight into the practices of estimating protein requirements for critically ill TBI patients at a range of BMIs.

	Ventilated patient BMI < 25kg/m^2	**Ventilated patient BMI 25-30kg/m^2**	**Ventilated patient BMI >30kg/m^2**
Initial target (e.g. N or g/kg)	n=11 81% of dietitians use 1.2-1.5gP/kg as their initial target 9% starting at 1.0gP/kg for days 1-3 and increasing to 1.3gP/kg for days 3-5 9% using a range of 1.2-2.0gP/kg with the top end used for high CRP (>150) as per local policy.	n=9 78% using the same requirements as for BMI <25kgm^2 11% using 1.3gP/kg if BMI>28.5kg/m2 11 % using 1.5gP/day/IBW	n=10 40% using 2.0-2.5gP/kg IBW 10% using 1.5-2.0gP/kg 10% using 1.5-2.0gP/kg/IBW 10% using 1.5-2.0gP/kg at 75% 10% using 1.2-1.3gP/IBW 10% using 1.2-1.3gP 10% using 1.2gP/kg at 75% *Note IBW as calculated using Hamwi or using BMI 25
Stated reasons for target changes	Comments: Obesity, renal function, weaning, 'depends on what's wrong', CRP, changes in clinical condition e.g. pressure damage No specific targets stated	No specific targets stated	Comments: Targets dependent on renal function, if the patient is on/off the vent, aiming higher range due to lower muscle mass pre adm. No specific targets stated
Unstable renal function / CRRT target	Unstable renal function target (n= 4): Range 1.0-1.3gP/kg CRRT target (n=9): Range 1.4-2gP/kg	Unstable renal function target- n= 0 CRRT target (n=2): Range 1.5-1.7gP/kg	Unstable renal function target- n= 0 CRRT target (n=4): Range 1.3gP/kg -2.5gP/kg IBW

Parameters that might affect targets (e.g anthropometry, renal function, skin integrity)?	Comments: Renal, feed availability, presence of other clinical conditions e.g. liver disease, caution in elderly with CKD, pressure damage, aim for 2.0gP/kg if polytrauma	Nil noted	Comments: Difficult to meet high protein requirements with available resources
Other comments (e.g. frequency of review and recalculation, products available to help meet requirements)	Continuous monitoring and review every 1-2 days. Recalculate requirements after 7 days and then as clinically indicated Some sites using protein modules	Nil noted	Nil noted

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9. Type of feed

Emma Service

Polymeric v Peptide Formula

Recommendation

For most critically ill TBI patients, a whole protein (polymeric) feed is suitable. Peptide formulas could be considered in those with poor absorption or GI complications but rarely as first line.

Rationale

International guidance suggests standard polymeric formula is appropriate and well tolerated in most critical care patients[1,2,3]. No clear benefit on outcomes (mortality; infections; length of stay; diarrhoea or energy/ protein intake) has been shown with the use of peptide-based formula and furthermore, it incurs a higher cost.

Gastrointestinal (GI) intolerance is common in critically ill TBI patients. In clinical practice pre-digested peptide-based formulas, are commonly used by dietitians to promote GI tolerance and improve diarrhoea. However, studies comparing this to polymeric formula are lacking. There is no indication that peptide enteral feeds are better tolerated than polymeric in terms of GI tolerance in general critical care patients, nor have they been proven to improve nutritional adequacy[4]. Furthermore, Carterton et al (2021) demonstrated that peptide formulas do not significantly improve GI tolerance of EN or diarrhoea in critically ill TBI patients[5].

Summary of Experience from Dietitians in Trauma Workshop 2019

Data collected from 15 dietitians working with critically ill TBI patients in the UK demonstrated that 100% of patients received a polymeric feed on the protocol. Fourteen of these dietitians continue a polymeric feed rather than peptide based on initial assessment.

80% of the 15 dietitians use a high protein enteral feed or modular protein supplement.

Fibre v Non fibre

Historically there has been insufficient data to promote the use of fibre (soluble or insoluble fibre) in enteral feeds in critically ill patients[6]. A recent systematic review in 2021 showed EN formulas with fibre may help reduce the incidence and severity of diarrhoea and GI complications[7]. However, specific critical care groups were not analysed and therefore it is difficult to extrapolate this benefit specifically to the TBI population.

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10. What are the most effective motility agents for TBIs in Critical Care?

Marta Alves

Recommendation

Even though there is limited evidence with the majority of studies being observational, it is generally accepted that for TBI patients the use of dual prokinetics (combination of metoclopramide and erythromycin) is more effective than just single prokinetic therapy[1-3].
Evidence: Grade C

Rationale

When to initiate prokinetics

A paper from 2007[4] suggests that approximately 50% of all intubated and ventilated patients may experience delayed gastric emptying (DGE). Furthermore, several studies show that DGE or issues with feed intolerance (also associated with vomiting and diarrhoea) in TBI patients is higher than other critically ill patients with no head injury[5-6].

The measurement of gastric residual volumes (GRVs) for assessment of gastrointestinal dysfunction is a common practice in critical care as it may help identify intolerance of enteral nutrition (EN) during initiation and progression[7-9]. The majority of international guidelines suggest that EN should only be delayed when a GRV is above 500 ml in a 6-hour period. In this situation, and if examination of the abdomen does not suggest an acute abdominal complication, the use of prokinetics should be considered.

Even though more studies and stronger evidence is required, one study in tolerance and efficacy of EN in TBI patients induced into barbiturate coma (with the use of Thiopental) suggests that these patients are likely to develop ileus that is refractory to prokinetic agents. However, this type of treatment is generally used for exceptional cases when otherwise intracranial pressure management is not well controlled[10]. Post pyloric feeding or parenteral nutrition should be considered if gastric feeding fails.

What prokinetic/s should be used

In critically ill adults, combination therapy with erythromycin (3-7 mg/kg per day) and metoclopramide (10mg 3-4 times a day), have shown to be more effective, when compared to single prokinetic therapy. Specifically in TBI patients, 3 different studies, comparing the effect of dual prokinetics conclude that combined prokinetics are more effective than using a single dose of either metoclopramide or erythromycin[1-3].

Possible sides effects and when to discontinue them

Both drugs have been associated with higher incidence of watery diarrhoea, QT prolongation and a predisposition to cardiac arrhythmias[7-8]. In addition, the effectiveness of prokinetics is decreased to one third after 72 hours. ESPEN recommendation is to consider discontinuing them after three days.

Summary of Experience from Dietitians in Trauma Workshop 2019

83% of Dietitians working in Trauma reported that their enteral feed policy includes prokinetics use. Of these, the GRVs thresholds differ between Trusts: 16% of them use 250 ml, 25% use 300 ml; 17% 350 ml; 25% between 400-500ml and the remaining 17% > 500 ml.

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11. Does the use of immunonutrient supplementation improve outcomes?

Jen Wetherden

Recommendation

The efficacy of immune-modulating enteral formulas, probiotic supplementation, glutamine supplementation, and omega-3 fatty acid supplements for the treatment of TBI requires further research prior to recommending for standard use in patients following TBI.

Rationale

The gut plays an important role in the body's immune response[1], and there is a well-documented increase in intestinal permeability in trauma patients increasing the risk for gut bacteria translocation[2].

It has been theorised that supplementation with immune modulating nutrients may impart benefits to patients with TBI, such as improving the gut mucosal barrier. Glutamine has demonstrated improvements in the integrity of the intestinal tract, as well as reducing inflammation, and a reduction in apoptosis of mucosal cells. Omega-3 alpha-linolenic acid (ALA) is an essential fatty acid and precursor to other fatty acids essential to the health and development of the nervous system[3]. Arginine is defined as a conditionally essential amino acid; meaning it is made by the body, but runs short during times of metabolic stress (i.e. critical illness). Arginine rich peptides (ARPs) have been shown to act on multiple pathways of injury, including a neuroprotective effect, and consequently considered as a potential therapy[4].

A systematic review in 2021[5] of 27 studies concluded that supplementation with arginine and other nutrients has no effect on overall mortality in critically ill patients, no effect on rate of infectious complications, no effect on hospital length of stay and critical care length of stay. Diets supplemented with arginine and other nutrients may be associated with a reduction in duration of mechanical ventilation in critically ill patients, but the presence of significant heterogeneity limits this extrapolation, and furthermore this is not specific to patients with a TBI. No human studies were identified that have examined arginine as a stand-alone supplement in individuals following TBI.

The use of probiotics in critical care has long been studied, with growing interest in the gut microbiome and its role in the prevention and treatment of disease. There have been several systematic reviews conducted[6,7] but no clear guidance established, and caution recommended among high-risk groups such as critically ill patients, due to the risk of serious infection (bacteraemia, fungemia, sepsis and endocarditis, in particular)[8].

American clinical practice guidelines (based on consensus) suggest the use of an arginine-containing immune-modulating formula or supplementing Eicosapentaenoic Acid (EPA)/Docosahexaenoic Acid (DHA) to enteral feeds in individuals following TBI[2].

Summary of Experience from Dietitians in Trauma Workshop 2019

None of the dietitians working within trauma reported using immune-modulating enteral formulas in adults with TBIs, either as a starter feed in the out of hours regimen, or on initiation on assessment. Dietitians were not questioned on their use of immune modulating stand-alone supplements.

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12. What is the efficacy of a Ketogenic diet in improving outcomes?

Karen Jackson

Recommendation

To date there have been minimal studies on the use of the ketogenic diet (KD) therapy in TBI patients. Majority of the research has been conducted with animal models, less is known about the efficacy of the KD in adults, and even less in the intensive care setting. Therefore, there is currently no recommendation or strong evidence that implementing a KD improves outcomes in individuals with TBI in the critical care and optimal feeding formulation remains unclear.

Rationale

The brain is dependent on a constant supply of energy due to intensive cellular processes and maintenance of electrochemical gradients. TBI combined with critical illness induces increased metabolism and catabolism that can result in poor outcomes[1].

Over the last decade interest has grown using neuroketotherapeutic therapies i.e. the KD and ketone esters for the potential neuro-protective effects in TBI[2]. The protective properties include maintaining energy supply, attenuation of oxidative stress, apoptotic cell death and attenuating acute cerebral damage via modulation of the inflammatory response and decrease on traumatic and ischaemic cerebral damage through the production of cerebral ketone body (KB) metabolism[3,4].

A ketogenic diet is a high fat, low carbohydrate, adequate protein diet. Typically, a ketogenic diet has been used for the chronic stage of pharmacoresistance of epilepsy, and in acute stage of refractory/super-refractory status epileptic critical cases in children and young infants[11]. Attention has now turned to using the KD in adults following case reports that have demonstrated a disease-modifying effect when standard treatments have failed. Feasibility studies in adults with TBI & management of refractory intracranial pressure[12], post-concussion syndrome (PCS)[13] managed on a KD suggest that it may be a potential treatment.

Under normal conditions, the brain utilizes glucose as the main substrate of energy derived from the breakdown of carbohydrates. Post TBI, an adaptive metabolic response by brain astrocytes, the key cells involved in regulating brain metabolism, results in utilisation of alternative substrates such as lactate and ketones[5]. The switch to producing ketones and using them as the key body fuel occurs due to a) small stores of carbohydrates, b) limited protein stores, c) an abundance of tri-acyl glycerol and d) the inability of long-chain fatty acids to cross the blood-brain barrier[6]. Following injury, brain glucose metabolism is impaired, the body decreases insulin secretion and increases glucagon secretion. Glucose becomes a less favourable energy substrate and mobilisation of fatty acids from adipose tissue to the liver for breakdown (endogenous ketosis or ketogenesis) provides an alternative energy supply preserving glucose reserves[7]. Ketones produced by astrocytes from fatty acids and leucine are the only endogenous fuel that can contribute significantly to cerebral metabolism[8].

High KB concentrations have been detected in TBI critical care patients in the fasted state. Correlations of brain KB with glutamate, lactate, pyruvate support the hypotheses that increased cerebral metabolic stress may trigger KB release and act as alternative substrates to glucose for fuel in the early phase of TBI[9,10].

The classic ketogenic diet, in the limited studies available, has been the usual KD of choice in TBI patients as it has already been used in clinical practice for the treatment of medication resistant epilepsy to generate KB from metabolising fat rather than carbohydrate for fuel. From studies and case reports, it is evident that administration and monitoring of the KD is labour intensive, requires clear goals i.e. therapeutic level of ketosis, pathways to identify and manage symptoms i.e. hypoglycaemia and hyperketosis, collaboration of the MDT and employment of person(s) with KD experience.

The safety and tolerability of the KD in adults is unclear particularly in TBI critical care patients. Reporting of adverse events post commencement of the KD are limited due to the limited literature available. Kidney stones, excessive acidosis, excessive hypoglycaemia, gastrointestinal upset i.e. medium chain triglyceride (MCT) related diarrhoea, constipation, nausea and vomiting have been raised as potential side effects.

The evidence to recommend the use of KD in TBI is not substantial enough due to limited human studies, minimal randomized control trials, small sample size studies and most of which being case reports or retrospective studies. These limitations act to impede the claim that KD is a valid option for management of TBI. Further clinical studies in humans with larger sample sizes are required to provide evidence and validate the neuroprotective benefits of ketogenic therapy

Summary of Experience from Dietitians in Trauma Workshop 2019

No dietitians reported that they have been utilising the ketogenic diet as part of their practise to improve outcomes in TBI individuals and only in the limited literature has employing the KD diet been used.

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13. Glycaemic Control in TBI

Emma Service

Recommendation

It is generally accepted that blood glucose should be maintained at <10mmol/l (range 6-10mmol/l) in critically ill TBI patients. Insulin can be administered to resume normoglycaemia.

Rationale

The optimal serum glucose level in TBI patients remains unclear, however it is well documented that hyperglycaemia frequently occurs early and is associated with higher mortality rate[1,2]. Hyperglycaemia can also exacerbate secondary brain injury and result in worse neurological outcomes[3].

Insulin increases glucose utilisation and reduces the damage of hyperglycaemia to brain cells. Close monitoring of blood glucose and frequent adjustment of insulin is required. A variable rate intravenous insulin infusion (VRII) is used commonly to help achieve normoglycaemia in critical care.

In critical care patients, the landmark study by Van den Berghe[4] demonstrated favourable outcomes for mortality and morbidity attributed to intensive glucose control (IGC) (blood glucose 4.4.-6.1mmol/l) in comparison to the control group (blood glucose 10-11.1mmol/l)[4]. However, since a further trial has shown higher mortality (statistically significant) in the IGC group (blood glucose 4.5-6mmol/l) versus conventional targets (blood glucose <10mmol/l), where the IGC group experienced higher incidence of hypoglycaemic episodes[5].

A systematic review and meta-analysis (7 RCTs, 1013 cases) specifically in TBI patients looking at IGC (glucose 4.5-6.1mmol/l) promoted favourable neurological outcomes, lower infection rate and shorter length of stay[6]. It has also been shown IGC therapy can help to reduce intracranial pressure in those with isolated TBI[7].
Besides lowering blood glucose with insulin, there are various strategies clinicians often adopt to help normalise glucose homeostasis in TBI patients:

- Early nutritional support can improve insulin resistance[8].
- Cerebral microdialysis can be used to monitor the metabolic state of the injured brain and might help identify effective glycaemic target ranges[9].
- In severe TBI, moderate hypothermia (32.0°C to 35.0°C) induced within 24 hours of injury is associated with a lower incidence of intracranial pressure (ICP) and decreased levels of glucose compared to the control group (36.5°C to 37.0°C)[10].
- Hypertonic saline or mannitol can decrease raised ICP associated with hyperglycaemia after severe TBI[11].

Summary of Experience from Dietitians in Trauma Workshop 2019

When asked via the Trauma Sub-Group Basecamp, dietitians reported insulin is generally administered in critically ill TBI patients when blood glucose is >10mmol/l via a VRII, with a target of 6-10mmol/l. This does not differ to the treatment of hyperglycaemia in other critical care patients. Tighter glucose control is not adopted due to the risk of hypoglycaemia.

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TBI Case study 1

A 28-year-old male patient, previously fit and well admitted to a trauma unit following RTA (motorbike vs car) with TBI, left frontal lobe contusion and traumatic subarachnoid haemorrhage requiring critical care admission. During our first dietetic assessment the following was identified:

Anthropometry

- weight 48kg on admission (weight measured via critical care bed)
- Height estimated 1.72m (measured using demispan)
- BMI 16.22kg/m^2
- On bed end visual assessment by dietitian, low BMI confirmed

Biochemistry:

- Sodium 138mmol/l, potassium 4.0mmol/l, adjusted calcium 2.2mmol/l,
- phosphate 0.8mmol/l, magnesium 0.9mmol/l, urea 6.5mmol/l, creatinine 48umol/l
- Blood glucose unsupported but stable readings <8.5mmol/l
- CRP 48mg/L, WCC 10.2/L

Clinical information:

Relevant clinical information for dietetic assessment:

- Intubated and ventilated on 35% FiO2
- Sedated with propofol 25mls/hour (1%) + fentanyl + atracurium
- Cardiovascular system supported with 0.2mcg noradrenaline
- Renal function: U.O ~ 30mls/hour
- Bowels open daily type 7 without use of laxatives
- Skin integrity: grazes to face

Dietetic assessment

- 12Fr NGT feeding tube placed and position confirmed by chest x-ray

Questions

1. When would you initiate nutrition?

    - *If clinically stable & inotrope support reducing to commence hypocaloric enteral nutrition as per guidelines – ASPEN/ESPEN.*

2. Would a protocol be followed or would you as dietitian individually assess the patient?

    - *Given low BMI and high propofol contribution to energy provision, individual assessment recommended to not overfeed on calories and underfeed on protein. May require extra modular protein.*

3. How would you calculate nutritional requirements for a patient with a low BMI within the first ten days?

    a. calories

    - *Given that predictive and weight- based equations either significantly over- or*
    - *underestimate requirements in this cohort of patients, careful monitoring is required.*
    - *Initially calculate requirements on actual body weight using Penn State (2003b) but being aware that this may be only 58% accurate or Aspen commencing at 12kcals/kg and building up to 25kcals/kg. When established on feed and biochemically tolerating can recalculate requirements (~day 7-10) on IBW (refer to energy section on IBW) and build up to full requirements using IBW.*

b. protein

- *Calculate @ 1.2-1.5g/kg/ABW and build up to total protein requirement over a period of 7/7 monitoring renal function & actual feed delivery. When established on feed and biochemically tolerating can recalculate requirements (~day 7-10) on IBW.*

c. micronutrients

- *Consider providing micronutrients in addition to formula feed as feed may not provide full micronutrient requirements in smaller volumes. Recommended to check what volume feed is complete in. Consider daily multivitamin until on complete feed.*

4. The patient starts to develop large gastric aspirates (more than the critical care usual cut-off level) and there is evidence of aspiration on chest x-ray. What would be your course of action?

- *Would you change method of feeding and/or consider use of prokinetics?*
- *Aspiration may not be due to large GRVs. There is limited evidence that large GRVs are a factor of aspiration. However, GRVs may be the result of reduce gastric emptying and may result in patient vomiting which there is evidence that vomiting can cause aspiration. Can follow critical care prokinetic protocol is there is one available and/or discuss with MDT to commence prokinetics.*
- *There is evidence that NJ/post pyloric feeding reduces aspiration risk if it is possible to site a NJ tube.*

5. Whilst trying to re-establish enteral feeding, the patient's blood glucose level is consistently above 15mmol/l and you have yet to meet their nutritional targets. How do you approach this issue?

- *Discuss the issue with the MDT. Consider commencement of insulin and a BG target range.*
- *If the patient is on steroids or septic it may result in hyperglycaemia. Some critical care units now have a higher BG target range for patients on steroids.*
- *Consider reviewing your enteral feeds for lower carbohydrate feeds and 24hour feeding to aid BG control.*

6. When the patient has been established on their initial nutritional target. What factors would you consider for progressing nutrition after ten days and what would your nutritional aims be?

- *To monitor anthropometrics:*
- *MUAC to monitor muscle wasting*
- *Ultrasound of muscle to monitor muscle wasting (if available)*
- *Weight to monitor weight loss*
- *Grip strength – if able*
- *Nutritional adequacy via feed delivery – ensuring calorie, protein & micronutrient targets are met*
- *Changes in clinical status - requiring adjustments of nutritional provision*
- *Changes to activity – to ensure nutritional provision is incorporating changes in activity*
- *Blood glucose - to ensure within range*
- *Adequate fibre – to aid bowels opening*
- *Monitor for signs of malabsorption*
- *Monitor for signs of gastric tolerance with progression of enteral feeding*
- *If extubation possible, working with Speech and Language Therapist to progress diet*

7. The patient has received poor nutritional delivery throughout their 15-day admission. What would your nutritional strategy be considering the patient is still has considerable insulin requirements?

- *Aim to increase feed delivery and select lower carbohydrate feed if possible. Aim for 24hr feeding to aid BG control.*

- *Work with MDT + Diabetes Nurse Specialist to provide insulin regimen with increased enteral feed & discuss BG targets. Patient may be on steroids and/or septic which may impact BG control.*
- *If the patient is on VRII discuss with the MDT commencement of background insulin with VRII to manage spikes in BG control.*

8. The patient's sodium increases to 154mmol/l and urea increases to 14.2mmol/L. The patient has been commenced on diuretics. You have been asked to review the patient in light of the increasing sodium and urea levels. What would be your approach?

    - *It is likely that the patient as a result of diuresis has become intravascularly dry.*
    - *You can suggest that enteral water is provided to lower sodium and urea levels.*
    - *Check if any medications can be changed to provide lower sodium i.e. soluble paracetamol and some IV antibiotics given in sodium as a regular prescription contain high sodium levels.*
    - *Check if possibility to change to lower sodium containing feed but beware that low sodium levels are of lower nutritional value, and you may need to provide large volumes to meet the patients nutritional requirements.*

Case Study 2: Indirect Calorimetry – TBI/polytrauma

A 34-year-old male admitted post polytrauma - bike vs car (hit pothole and went over handlebars and into oncoming traffic). Traumatic cardiac arrest with helicopter emergency services.

Multiple injuries at the time of admission:

1. TBI – Subarachnoid Haemorrhage (SAH), Diffuse Axonal Injury (DAI), Subdural Haematoma (SDH) - managed conservatively with Increased intracranial Pressure (ICP) bolt (removed day 15), GCS persistently E4VtM4
2. Spinal #s C6 right lamina, C7 transverse process, multiple thoracic transverse #s, S1# (in Miami J)
3. Ventilator acquired pneumonia - treated with multiple antibiotics
4. Right hip dislocation (fixed day 1)
5. Unstable pelvic fracture – (fixed day 14)
6. Multiple rib #s managed conservatively, bilateral pneumothoraces resolved
7. Commuted bilateral scapula # and left posterior humeral dislocation post polytrauma

Initial Dietetic Assessment on day 2:

Anthropometry

Estimated at 80kg on admission.
Ulna 27.5cm indicating height 1.78m. BMI therefore 25kg/m^2

Biochemistry (out of range only)

eGFR 46 (32) Urea 15.4 (14.9) creatinine 165 (224)
Na 148 (148), AdjCa2+ 2.13 (low – previously within range)
CRP 151 (102) WBC 9.0 (12.8)

Clinical

Inferior Vena Cava (IVC) filter placed. Maximum Temperature: 36.9^0 c
Current infusions: propofol (2% running at 15ml/hr), fentanyl, rocuronium, norad (0.14mcg)
Intubated and ventilated via ETT: PRVC FiO2 25%, VE: 10.8, SpO2 98%

Dietary

Patient started on standard critical care out of hours protocol and reached target, providing around 1400kcal and 58g protein in 1400ml of a standard polymeric, non-fibre feed
Estimated requirements:
Energy: 25kcal/kg: 2000kcal/day
(Mifflin St Jeor: 1748kcal/day PSU 1963kcal/day)
Protein: (1.5-2.0g/kg) 120-160g/day

Indirect calorimetry performed on day 19 of admission :

REE: 2207kcal (est ~28kcal/kg)

- RQ: 0.90 (i.e. indicates patient is fed on mixed substrate feed and not over/or under fed)
- Variance: VCO2: 4% VO2: 2%

Penn State Equation (PSU): 2018kcal

Day 27: Extubated
Day 31 GCS E4V3M4. Pt 100% NGT fed on oral trials of thin water only.
Day 33: Step down from critical care to trauma ward

Questions:

1) The patient was still sedated with high doses of opiates on day 4, and started experiencing symptoms of delayed gastric emptying (gastric residual volumes of up to 700ml), and bowels had still not opened. Describe the impact of these medications on gut motility.

- *Propofol impairs gastric and colonic contractile activity. Propofol is often used in conjunction with opioids, meaning more inhibitory effects on intestinal motility. Combined with TBI and raised ICPs which induces immense increases in sympathetic activity, which is responsible for several of the peripheral systemic and GI symptoms. Increased ICP may cause changes in GI motility, water, electrolyte absorption*

What is your trust protocol for commencement of prokinetics?

2) When the patient has been established on their initial nutritional target, what factors would you consider for progressing nutrition after ten days and what would your nutritional aims be?

- *Factors: Sedation provision, ventilator requirements, storming, seizures, pyrexia, fluid balance, adequacy of nutritional provision so far. Aims: Weight stability, meeting >90% estimated requirements, feed tolerance*

3) Reflect on the indirect calorimetry measurement of REE versus using the Penn State equation and calories per kilogram.

- *REE higher than PSU – does not include activity factor as well – demonstrates hypermetabolism of TBIs. Using kcal/kg appears more accurate in this case.*

4) What type of feed do you normally commence this type of patient on? What do you consider when choosing fibre vs non-fibre?

- *Discuss any out of hours protocols available and type of provision.*
- *Types of injuries- any GI injuries or surgery. Consider anastomoses. Fibre is safe in majority of critically ill patients – common misconception that fibre is contraindicated when pts is receiving inotropes.*

5) The patient starts experiencing loose stools on critical care. What would you consider and what actions would you take?

- *Stool frequency (overflow?) bowel history, relevant PMHx, stool sample – infection, faecal elastase, drugs (antibiotics, laxatives), overall gut motility*
- *Consider fibre manipulation (adding in, removing or vice versa), fibre dose, semi elemental. Consider feeding styles (bolus feeding)*
- *Consider loperamide if infective cause ruled out.*
- *Consider additional provision of feed to cover for losses (i.e. if stools >350g/day)*

Appendices

Appendix 1: Critical care nutrition guidelines summary for energy and protein requirements

		ASPEN (2016)	ESPEN (2019)	TBI handbook (2022)
Energy	BMI <30kg/m^2	25–30kcal/kg/day Aspen (2021): 12-25kcal/kg in initial 7-10 days	REE from VCO_2 obtained from ventilators: REE = VCO2 x 8.19 REE Day 1-3: 70% of MEE REE Day 3-7: 80-100% of MEE 20-25kcal/kg/day	Moderate-severe injury: 25-30kcal/kg/day
	BMI 30-50kg/m^2 BMI >50kg/m^2	11–14kcal/kg actual body weight/day 22–25kcal/kg ideal body weight/day (Hamwi)	20-25kcal/kg/adjusted body weight/day	11–14kcal/kg actual body weight/day 22–25kcal/kg ideal body weight/day *
Protein	BMI <30kg/m^2	1.5-2.5g/kg actual body weight/day	1.3g/kg	1.5-2.5g /kg actual body weight/day
	BMI 30-40kg/m^2	2.0g/kg/ideal body weight (Hamwi)	1.3 g/kg/adjusted body weight/day	2.0g/kg/ideal body weight (Hamwi)
	BMI >40kg/m^2	2.5g/kg/ideal body weight (Hamwi)		2.5g/kg/ideal body weight (Hamwi)

Note: the above energy and protein requirements may not be specific to TBI patients but also to critical care patients in general.

Ideal body weight estimated from Hamwi equation

- Women: IBW (kg) = 45.5 + 2.3 x (height - 60in)
- Men: IBW (kg) = 50.0 + 2.3 x (height - 60in)

Adjusted body weight as per ESPEN (2019) = (actual body weight - ideal body weight) x 0.33 + ideal body weight

Penn State Recommendations:

BMI <30kg/m^2:

Use Penn State 2003b

1st step- estimated Mifflin St Jeor:

For men: REE = (kg x 10) + (cm x 6.25) - (age x 5) + 5
For women: REE = (kg x 10) + (cm x 6.25) - (age x 5) -161

2st step- estimated Mifflin St Jeor:

(REE from Mifflin-St. Jeor x 0.96) + (VE [minute volume in L/min] x 31) + (Tmax [maximum body temperature in last 24 hrs] x 167) – 6212

Obese adults BMI>30kg/m2 <60years old: Use Penn State 2003b

Obese adults BMI>30kg/m2 >60 years old: Use Modified Penn State 2011

For men: (wt [kg] x 10) + (ht [cm] x 6.25) – (age x 5) + 5
For women: (wt [kg] x 10) + (ht [cm] x 6.25) – (age x 5) -161

REE = (Mifflin x 0.71) + (Tmax x 85) + (Ve x 64) – 3085

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