

A Guide to the Nutritional Assessment and Treatment of the Critically III Patient

Mark S. Siobal, BS, RRT, FAARC Jami E. Baltz, RD, CNSC



Produced by the American Association for Respiratory Care Copyright© 2013 by the American Association for Respiratory Care

Supported by an eduction grant from



Table of Contents

| Foreword |
|---|
| Executive Summary |
| Nutritional Overview6 |
| Nutritional Assessment |
| Malnutrition |
| Nutritional Support18 |
| Determining Nutritional Requirement |
| Clinical Practice Recommendations for Nutritional Support |
| Summary 40 |
| References |
| Acronyms |

Foreword

As a key component to the interdisciplinary health care team, respiratory therapists must be cognizant of all of the variables that impact the care of the critically ill patient in the intensive care unit. This team includes nurses, pharmacists, physicians, dietitians, nutritionists, physical therapists, and respiratory therapists.

Proper nutritional assessment and treatment is a key component in the management of these patients. Malnutrition of the critically ill patient (and especially those on mechanical ventilation) is not a rare event; it happens fairly often. Malnutrition can lengthen time spent in the ICU, extend hospital length of stay, and for the mechanically ventilated patient, it can delay or impede the weaning process, which brings with it other associated risks. Therefore, it is the obligation of all bedside clinicians to be sure that critically ill patients be assessed for nutritional adequacy and that appropriate intervention is taken. In short, this intervention should be a multidisciplinary effort. All disciplines play an important role in managing the nutritional needs of the critically ill patient as there are several factors that must be considered beyond the patient's caloric intake. This guide provides these considerations in a thoughtful and comprehensive manner.

This guide not only covers nutritional assessment and management of the adult critically ill patient, but

also discusses specific patient populations where malnutrition is more prevalent. Obese patients, pediatric patients, and the elderly population are such classifications that are presented in this guide. Proper nutrition is key to everyone but carries greater importance in the critically ill or mechanically ventilated patient.

The American Association for Respiratory Care is grateful for the unrestricted grant from GE Healthcare that allowed us to write and publish this document. It provides a balanced review of the literature for the diagnosis, treatment, and management of the nutritional needs of the critically ill patient. This document should be in the hands of all respiratory therapists at the bedside who are managing patients in the ICU and on mechanical ventilation.

1. Kaliston RRT

Thomas Kallstrom, MBA, RRT, FAARC Executive Director/Chief Executive Officer American Association for Respiratory Care

Executive Summary

Introduction

The purpose of this guide is to provide an overview of the important considerations regarding nutritional assessment and treatment that the health care team must address to ensure patients are provided with appropriate nutritional support. The goal of this work is to review a broad list of topics that covers the nutritional support and care process to provide the health care team with a broad understanding of the nutrition assessment and treatment process for the hospitalized critically ill patient.

Overview

Appropriate nutrition is essential for improving outcomes in the health care environment. Hospitalized patients have high rates of malnutrition. Unmet nutritional needs and malnutrition lead to increased morbidity and mortality, decreased quality of life, prolonged duration of mechanical ventilation, and increased length of hospital stay, all of which contribute to the higher cost of health care. Critically ill patients and those patients with respiratory failure require special attention to prevent muscle wasting and to avoid overfeeding and complications associated with nutritional care. A functional nutrition support system should include an interdisciplinary team approach for assessment and treatment, which incorporates an evaluation of nutritional risk, standards for nutritional support, an appropriate assessment and reassessment process, proper implementation, route of support based on patient condition, and a means of measuring nutrient requirements to determine if target goals are being met

Interdisciplinary Approach

The Society of Critical Care Medicine recognizes the value and importance of a multidisciplinary team approach to nutritional care as a means to improve clinical outcomes. Each discipline in an intensivist-led interdisciplinary team, which includes dietitians, nurses, pharmacists, respiratory therapists, speech pathologists, and physical therapists, can contribute to improved outcomes and reduced health care costs.

Nutritional Risk and Assessment

Assessment of nutritional status is performed to identify patients at higher risk for malnutrition-related complications. Patients with moderate or severe malnutrition are likely to have longer ICU and hospital length of stay and higher risk of death. After the initial assessment, the primary goals of nutritional support are to maintain lean body mass in at-risk patients and to provide continuous evaluation of the nutrition care plan. Minimized risk of malnutrition can be achieved by prompt initiation of nutritional support, proper targeting of appropriate nutrient quantities, and promotion of motility through the gastrointestinal tract.

A registered dietitian or other trained clinician gathers information to examine the patient's nutritionrelated history and physical findings, anthropometric physical measurements, biochemical data, and medical tests and procedures, and then screens the patient for other nutrition-associated conditions such as malnutrition, obesity, and the risk of refeeding syndrome.

Route of Nutritional Support

Enteral nutrition (EN) is the preferred route of nutritional support. EN should be started within the first 24-48 hours after admission in patients who are incapable of volitional intake. Gastric or small bowel feeding is acceptable in the ICU setting. Enteral feeding tube placement in the small bowel should be done in patients at high risk for aspiration or whose intolerance to gastric feeding is demonstrated. Holding enteral feeding for high gastric residual volumes (GRV) in the absence of clear signs of intolerance and demonstrated risk of aspiration may result in an inappropriate cessation of EN and cause a calorie deficit over time. The definition for high GRV should be determined by individual institutional protocol; but use of GRV up to 500 mL has not been shown to increase the risks of regurgitation, aspiration, or pneumonia.

The decision to initiate parenteral nutrition (PN) is influenced by the patient's nutritional risk, clinical diagnosis and condition, gastrointestinal tract function, and duration of anticipated need. PN in a previously healthy patient should be considered when EN is not feasible for the first 7–14 days after hospital admission. Patients with evidence of moderate-to-severe malnutrition where EN is not an option should receive PN within the first few days following admission.

Nutritional Considerations During Critical Illness

The general goals of nutritional care in all patients, including those with respiratory disorders and critical illness, are to provide adequate calories to support metabolic demands, to preserve lean body mass, and to prevent muscle wasting.

Nutritional support during critical illness attenuates the metabolic response to stress, prevents oxidative cellular injury, and modulates the immune system. The stress response to critical illness causes wide fluctuation in metabolic rate. The hyper-catabolic phase can last for 7–10 days and is manifested by an increase in oxygen demands, cardiac output, and carbon dioxide production. Caloric needs may be increased by up to 100% during this phase. The goal is to provide ongoing monitoring and support with high-protein feedings while avoiding overfeeding and underfeeding. Nutritional modulation of the stress response includes early EN, appropriate macro- and micronutrient delivery, and glycemic control.

Determination of Nutritional Requirements

Nutrient requirements can be calculated by over 200 different equations. Predictive equations use traditional factors for age, sex, height, weight, and additional factors for temperature, body surface area, diagnosis, and ventilation parameters. Additional data such as injury-stress, activity, medications received, and obesity have been added to improve accuracy. Several predictive equations were developed with a focus on specific patient populations and medical conditions.

Predictive equations have varying degrees of accuracy. Error rates can be significant and result in underand overestimation of caloric needs that impact outcomes. Some equations are unsuitable for use in critically ill patients, while others have been validated with improved accuracy. Due to the extreme metabolic changes that can occur during critical illness, energy needs should be measured using indirect calorimetry (IC) in patients not responding to nutritional support,

have complex medical conditions, and are ventilator dependent.

Indirect calorimetry relies on accurate determination of oxygen consumption (VO2) and carbon dioxide production (VCO₂) using precise measurements of inspired and expired fractions of oxygen and carbon dioxide. The abbreviated Weir equation uses the measured VO₂ and VCO₂ to determine resting energy expenditure (REE). The respiratory quotient (RQ), the ratio of VCO2 to VO₂, can then be calculated. The RQ was once thought to be a means to determine nutritional substrate use, but this assumption has never been substantiated and use of the RQ measurement is of limited clinical value. Measured values of RQ between the physiologic ranges of 0.67-1.3 should be used as a way to validate test quality. Values of RQ outside of this range invalidate the results due to technical measurement errors and should be repeated.

Clinical Practice Recommendations

Several clinical practice guidelines are available to guide nutritional support. The Society of Critical Care Medicine and the American Society of Parenteral and Enteral Nutrition (SCCM/ASPEN), the European Society for Clinical Nutrition and Metabolism (ESPEN), the Academy of Nutrition and Dietetics (AND), and the Canadian Clinical Practice Guidelines for Nutritional Support (CCPG) have developed best practice recommendations based on the interpretation of available evidence, consensus agree-ment, and expert opinion.

The following present summaries of some of the best-practice recommendations from the various organizations:

- Nutritional support should be initiated early within the first 24–48 hours in critically ill patients.
- Primary goals of nutritional support and care are to: preserve and maintain lean muscle mass; provide continuous assessment, reassessment, and modification to optimize outcome; monitor the patient for tolerance and complications such as refeeding syndrome; prevent protein energy malnutrition by giving higher protein content while providing adequate total calories; monitor nutrition goals and target achievement rate of > 50% within the first week; and prevent accumulation of a caloric deficit.

- Indirect calorimetry should be used when available or when predictive equations are known to be inaccurate.
- Current EN practice recommendations are to: preferentially feed via the enteral route; initiate EN within 24-48 hours; reduce interruptions of EN for nursing care and bedside procedures to prevent underfeeding; maintain head of bed (HOB) elevation to reduce aspiration risk; accept GRV up to 500 mL before reducing or stopping EN in the absence of clear signs of intolerance; use motility agents to improve tolerance and reduce GRV; and promote post-pyloric feeding tube placement when feasible.
- Current PN practice recommendations are to: only use PN when enteral route is not feasible; use PN based on the patient's nutritional risk classification for malnutrition; delay PN up to seven days if the patient is in Nutritional Risk Class I or II; initiate PN early if the patient is in Nutritional Risk Class III or IV; convert to EN as soon as tolerated to reduce the risks associated with PN.

- Use of trophic or "trickle feeding" and permissive underfeeding may be beneficial.
- Use of pharmaconutrients and immunonutrition: omega-3 fatty acids (fish oils) may be beneficial in acute respiratory distress syndrome (ARDS) patients; utilize high omega-3 fatty acid to omega-6 fatty acid ratios. The use of arginine, glutamine, nucleotides, antioxidants, and probiotics may be beneficial in specific patients. The use of arginine should be avoided in patients with severe sepsis.

Appropriate nutritional support in hospitalized patients and the prevention of malnutrition can improve outcomes and reduce health care costs. The nutritional care plan should utilize the team approach and be supported by organizational standards with policies and procedures that are based on the best available evidence. The health care team's proper implementation, continuous assessment, and monitoring of the nutrition care plan are key elements for success.

Nutritional Overview

The Importance of Appropriate Nutrition

Appropriate nutrition is essential for health and healing. In hospitalized patients, malnutrition is a common problem affecting both adult and pediatric populations. Rates of malnutrition have been observed in 15–60% of hospitalized patients. ^{1,2} Critically ill patients are at high risk for malnutrition-related complications. The resulting detrimental effects of malnutrition include increased morbidity and mortality, decreased functional quality of life, prolonged duration of mechanical ventilation, and increased length of hospital stay, all which contribute to higher health care costs.³

Critical illness associated with respiratory failure requires special attention to prevent catabolic or destructive metabolism.4 Nutritional therapy in this setting requires maintenance of adequate calorie and protein intake to prevent muscle wasting and avoid overfeeding and complications associated with nutritional care.5 Malnutrition is a risk factor for the onset of respiratory failure and can worsen further after respiratory failure is established. Nutritional support can affect respiratory muscle strength, endurance and function, carbon dioxide production, and immune system response. To ensure successful support and recovery from respiratory failure, the nutritional care plan must also consider other important aspects, such as fluid and electrolyte balance, micronutrient requirements, and acid-base status. Recovery from respiratory failure requires a regimented nutritional support process that includes a comprehensive assessment of risk, proper implementation, ongoing reassessment of caloric requirements, tolerance of treatment monitoring, and avoiding the development of complications.4

Importance of Interdisciplinary Collaboration

The role of health care team members in providing expertise regarding nutritional support has evolved around interdisciplinary collaboration. Registered dietitians and physicians complete specialized training programs to attain the Certified Nutrition Support Clinician (CNSC) credential and are increasingly involved in nu-

trition support organizations such as the American Society of Parenteral and Enteral Nutrition (ASPEN).⁶

Respiratory therapists have traditionally maintained the responsibility and technical expertise in performing metabolic measurements by indirect calorimetry assessments, especially in the mechanically ventilated critically ill patient. Clinical practice guidelines developed by the American Association for Respiratory Care (AARC) maintain an evidence-based framework for nutritional assessments using indirect calorimetry for patients receiving mechanical ventilation.⁷

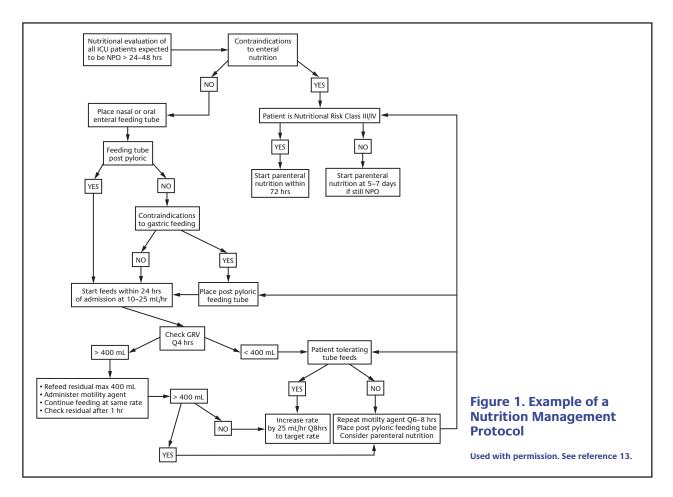
Speech pathologists aid in the assessment of post-extubation dysphagia. Detection of swallowing dysfunction that is common after prolonged mechanical ventilation can help prevent the detrimental impact and risks associated with aspiration and poor nutrition among patients with or without neurologic dysfunction. ^{8,9} Post-extubation dysphagia is associated with longer hospitalization in survivors of critical illness with neurologic impairment.

Critical care organizations such as the Society of Critical Care Medicine (SCCM) recognize the importance of an intensivist-led multidisciplinary team consisting of nurses, dietitians, pharmacists, respiratory therapists, and physical therapists. ¹⁰ Each discipline provides expertise pertinent to nutritional support and care, contributes to improved outcomes, and reduces costs.

The future and ongoing challenge to the evolution of health care is to facilitate the team approach toward best practices and therapeutic efficacy. Appropriate nutritional assessment and treatment protocols require devoted resources toward diagnosis, intervention, and monitoring. The integrated health care delivery team trained in nutritional assessment and treatment will be better equipped to optimize and ensure health care resources are maximized. ¹¹

Importance of Adequate Nutritional Assessment and Treatment

Nutritional deficits related to chronic disease and acute illnesses are frequently found in patients admitted to the ICU. Many patients who cannot resume oral food ingestion within the first few days of admission are prone to losing body mass due to poor nutrient intake and are at risk for developing an acute and pro-



longed inflammatory process. Patients in the ICU for more than 48 hours need nutritional assessment and support maintained constantly throughout their period of critical illness and hospitalization. Many critically ill patients experience severe gastrointestinal motility disorders and can experience dysphagia following extubation, which may increase the risks for aspiration. Complications associated with critical illness can have serious consequences that can be diminished with early recognition and intervention. The promotion of effective nutrition can only be achieved with a standardized nutritional support protocol that incorporates regular assessments of gastrointestinal function and tolerance of parenteral and enteral feeding. 12

In critically ill patients unable to take nutrition by mouth, EN through the gastrointestinal tract is the preferred route. PN by intravenous access is another alternative. Use of an evidence-based nutritional management protocol increases the likelihood that patients receive nutrition via the enteral route (see Figure 1).

A standardized approach targeting gastric or postpyloric feeding tube placement when indicated, gastric decompression and monitoring for high residual volumes, and use of bowel motility agents can shorten the duration of mechanical ventilation and reduce the risk of death. Clinical outcome benefits from improving the rate of EN can be significant when adjusted for nutritional risk of moderate-to-severe malnutrition at baseline.¹³

Development and maintenance of a best-practice nutritional support program reduces costs and improves outcomes. Maintenance of nutritional support requires continuous monitoring of the appropriate route of administration and the adequacy of usage in order to minimize costs and reduce waste. It Insufficient calorie intake is associated with an increase in mortality risk. The reasons for failure to achieve recommendations for best clinical practice include lack of sufficient nutritional support services to monitor adherence, inadequate training in nutritional support, and restricted use of nutrient formulations that show improved outcomes secondary to their higher cost or disagreement about the supporting evidence. Is

Nutritional Assessment

Nutritional Risk Assessment

Assessment of nutritional status is performed to identify patients at higher risk for malnutrition-related complications. Obesity is a risk factor for increased morbidity in the ICU with complications such as prolonged ventilation, infections, poor wound healing, and pressure ulcers. ¹² There is an increased understanding that acute and chronic inflammation are key risk factors in the pathophysiology of disease or injury associated with malnutrition. ¹⁶ Patients determined to have a nutritional status of Class III (moderate malnutrition) or Class IV (severe malnutrition) (see Table 1) ¹⁷ are more likely to have longer ICU and hospital length of stay and higher risk of death. ¹³

Table 1. Nutritional Risk Classification for Malnutrition

Class I Normal, no nutrition compromise,

nutritionally stable.

Class II Mild malnutrition, mildly compromised, somewhat nutritionally unstable with a few

nutrition-related problems or indicators that affect health status.

Class III Moderate malnutrition, moderately

compromised, several nutritionrelated problems or indicators that directly affect health status, and the patient may be medically unstable.

Class IV Severe malnutrition, severely

compromised, overt nutritional deficiencies or malnutrition, many nutrition-related problems or indicators that have profound effect on health status, the patient is considered medically and nutritionally unstable.

Used with permission. See reference 17.

Nutrition risk assessment should encompass two necessary elements. The initial assessment should establish the presence or estimate of lean body mass loss prior to ICU or hospital admission. The goal of preventing further loss of lean body mass can be achieved when acute illness is promptly controlled and with the formation of an adequate nutritional support process. Additionally, the safe provision of nutritional support requires a continuous evaluation of the risks of nutritional care. Minimized risk can be achieved by prompt initiation of nutrition, targeting the appropriate nutrient quantities, promoting motility through the gastrointestinal tract, and averting serious life-threatening complications such as refeeding syndrome. Patients found to be at higher risk for nutrition-related problems should receive specialized nutritional support. Development of nutritional assessment and care protocols designed for the specific needs of critically ill patients are required to minimize the reduction of lean body mass until discharge. Nutritional care from admission to hospital discharge is essential to reducing risk of nutrition-related complications and promoting recovery¹² (see Figure 2).

Standards for Nutritional Support

Nutritional support standards for adult acute care have been developed to guide the nutrition support process. These standards are designed to optimize the development and performance of a competent nutritional care plan (see Figure 3).¹⁸

Components of a nutritional support program should include the following: 18

Organization

A nutritional support service or interdisciplinary team approach with established policies, procedures, and a performance improvement process should be initiated for each admitted patient.

Nutritional Care Process

The process for nutritional care should identify atrisk patients using a screening process that is formalized and documented. Regulatory agencies such as The Joint Commission (PC.01.02.01 – EP 4) require that a nu-

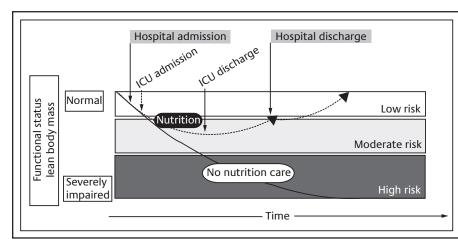
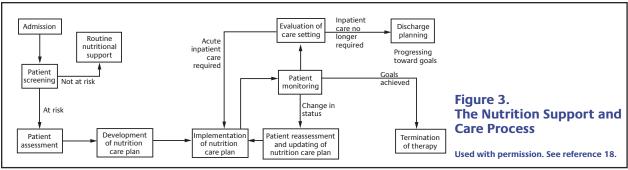


Figure 2. Time Course of Increasing Nutritional Risk Assessment and Support in Critical Illness

Used with permission. See reference 12.



tritional screening be completed when the patient's condition warrants within the first 24 hours after admission. Identified nutritionally at-risk patients should undergo a formal nutritional assessment that includes subjective and objective criteria, classification of nutritional risk, requirements for treatment, and an assessment of appropriate route of nutrition intake.

Development of a Nutritional Care Plan

The nutritional care plan should include clear objectives, use a multidisciplinary approach, have defined goals, select the most appropriate route, select the least costly substrate formulation for the patient's disease process, and include a process for reassessment of adequacy and appropriateness.

Implementation Process

The ordering process for the nutritional care plan should be documented before administration occurs. The appropriate nutritional access device should be inserted by a qualified health care professional using standardized procedures with appropriate placement confirmed and placement and/or adverse events documented. Enteral and parenteral formulations should be

prepared accurately and safely using established policies and procedures. Parenteral formulation should be prepared in a sterile environment using aseptic techniques. Additives to formulations should be checked for incompatibilities and prepared under direct supervision of a pharmacist. All nutritional formulations should be labeled appropriately and administered as prescribed while monitoring patient tolerance. Protocols and procedures should be used to reduce and prevent the risks of regurgitation, aspiration and infection, and a process for Sentinel Event review should be established.

Monitoring and Re-evaluating the Nutritional Care Plan

Establish the frequency and parameters for monitoring the nutritional care plan based on the patient's degree of nutritional risk. Standard procedures for monitoring and re-evaluation should be established to determine whether progress toward short- and long-term goals are met, or if realignment of goals are necessary.

Transition of Therapy Process

Assess achievement of targeted nutrient intake to ensure that at least 60% of estimated requirements are

being met before nutritional support is transitioned between parenteral, enteral, and oral intake. Maintain continuity of care when transitioning between levels of care or changes in the care environment. Termination of nutritional support should follow protocols that take into account ethical and legal standards and the patient's advance directives. 18

Nutritional Assessment

The nutritional assessment process includes the collection of data to determine the nutritional status of an individual. A registered dietitian or physician trained in clinical nutrition gathers data to compare various social, pharmaceutical, environmental, physical, and medical factors to evaluate nutrient needs. The purpose of nutrition assessment is to obtain, verify, and interpret data needed to identify nutrition-related problems, their causes, and significance. This data is then used to ensure adequate nutrition is provided for the recovery of health and well-being.19

Food/Nutrition-related History

Past dietary behaviors can be identified in the nutritional assessment to determine the individual's pattern of food consumption. Assessment of dietary history should include:

- Appetite
- Weight history (loss, gain)
- Taste changes
- Nausea/vomiting
- Bowel pattern (constipation, diarrhea)
- · Chewing, swallowing ability
- Substance abuse
- Usual meal pattern
- Diet restrictions
- Food allergies or intolerances
- · Medications, herbal supplements
- Meal preparation, ability to buy/obtain food
- Activity level
- Knowledge/beliefs/attitudes
- Nutrient intake

The registered dietitian may use a 24-hour recall or a usual daily intake recall, a food diary or food record, or a food frequency questionnaire. The 24-hour recall or food frequency questionnaire employ retrospective data that can be easily used in a clinical setting. The 24-hour recall is a commonly used technique incorporated into the patient interview in which the individual states the foods and the amount of each food consumed in the previous 24 hours. Accuracy of the recall is dependent on the patient's memory, the perception of serving size, and the skill of the interviewer to elicit complete information. The 24-hour recall may underestimate usual energy intake. Food frequency questionnaires (FFQ) collect information on both the frequency and amount consumed of specific foods.²⁰ The FFQ can help to identify eating patterns; however, intake of nutrients may be overestimated. In food diaries or food records, dietary intake is assessed by prospective information and contains dietary intake for three to seven days. These methods provide the most accurate data of actual intake but are very labor intensive and time consuming to analyze. Therefore, they are typically used in the research or outpatient

Anthropometric Measurements

Anthropometrics refers to the physical measurements of the body. The measurements are used to assess the body habitus of an individual and include specific dimensions such as height, weight, and body composition (i.e., skin-fold thickness, body circumference including points at the waist, hips, chest, and arms).16

Height and weight

Height and weight can be assessed by asking the patient or caregiver, or by taking a direct measurement. When recording data, note the date and whether the height and weight were stated or measured. Once these two measurements are obtained, a more useful number (the body mass index [BMI]), can be calculated. BMI is defined by weight and height measurements where:

Using pounds and inches:

BMI = Weight in pounds / (Height in inches) 2 x 703

Using kilograms and meters:

BMI = Weight in kilograms / (Height in meters)²

BMI can have a strong correlation between body fat and risk of disease. This number is a useful tool for determining the BMI category: underweight, healthy weight, overweight, obese, or morbidly obese.

BMI categories

A healthy weight may be confirmed by a BMI of between 18.5 and 24.9 for adults or a BMI-for-age between the 10th and 85th percentiles for children. A BMI of 25.0

| BMI (kg/m²) | Classification | Risk of Comorbidities |
|-------------|------------------------|------------------------------|
| <16.0 | Severe underweight | Severe |
| 16.0-16.9 | Moderate underweight | Moderate |
| 17.0-18.5 | Mild underweight | Average |
| <18.5 | Underweight | Low, but risk of other |
| | | clinical problems increases |
| 18.5-24.9 | Normal weight | Average |
| 25.0-29.9 | Overweight (pre-obese) | Increased |
| 30.0-34.9 | Obese Class I | Moderate |
| 35-39.9 | Obese Class II | Severe |
| ≥40.0 | Obese Class III | Very Severe |

to 29.9 indicates excessive weight in the adult. BMI-forage in children suggestive of excessive weight is between the 85th and 95th percentiles. Obesity is defined as a BMI greater than 30 in the adult and greater than the 95th percentile in boys and girls aged 2 to 20 years. Adults who are categorized as underweight have a BMI of less than 18.5, while underweight children score in the bottom 10th percentile for BMI-for-age. See Table 2.²¹

Body composition

Body composition measures body fat, muscle mass, and bone density. Body weight variations in individuals of similar height differ in the proportion of lean body mass, fat mass, and skeletal size. Several common measurements, which include skin-fold thickness, circumference measurements, and more high-tech measurements like bioelectrical impedance analysis (BIA) or dual-energy X-ray absorptiometry (DXA) scans, can be used to determine body fat to body mass, intracellular water to extracellular water ratios, and bone density.

Skinfolds

Skinfold thickness measures subcutaneous fat with the assumption that it comprises 50% of total body fat. Usually, the triceps and subscapular skinfolds are the most useful for evaluation. Skinfold thickness measurements are limited by reliability due to proper equipment and technique of the examiner and have limited practical application in the acute care setting.

Arm muscle area

The triceps skinfold (TSF) measurement, along with mid-upper arm circumference (MAC), is used to calculate the arm muscle area (AMA). The MAC is measured halfway between the acromion process of the scapula and the tip of the elbow. The results indicate muscle stores available for protein synthesis or energy needs. Changes over time in AMA will show whether the patient has been deprived of protein or calories. AMA is one of the markers of nutritional status and can be a predictor of mortality.²³

Waist circumference

An alternative to BMI, waist circumference can be a more accurate predictor of excess body fat and risks associated with obesity.²⁴ The measurement of waist circumference has been correlated with visceral fat;²⁵ and the distribution of body fat, specifically as visceral fat, which is deposited in the abdominal region, is correlated with obesity-related health risks.²⁶ (See Figure 4.)

According to the U.S. Department of Health and Human Services (HHS), the following individuals are at increased risk for developing chronic diseases:

- Women with a waist circumference of more than 35 inches
- Men with a waist circumference of more than 40 inches.

However, the World Health Organization, due to recent research findings, has recommended lower thresholds for waist circumference for Asian populations. ²⁶ Therefore, those at increased risk for developing chronic disease include:

- Asian women with a waist circumference of more than 31 inches
- Asian men with a waist circumference of more than 35 inches.

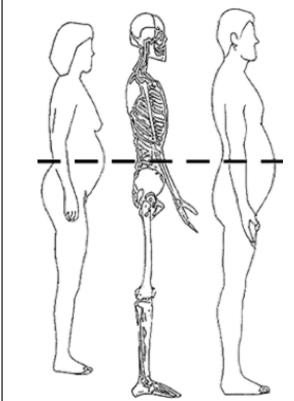


Figure 4. Measuring-Tape Position for Waist (Abdominal) Circumference in Adults

Photo courtesy of the National Heart, Lung, and Blood Institute

Other body assessment tools

More accurate measurements of body composition include the more advanced techniques of bioelectrical impedance analysis (BIA), low-density X-rays (DXA), computed tomography (CT) scan, and magnetic resonance imaging (MRI). These methods are very accurate and noninvasive; however, they are not necessarily ideal in the clinical setting, are expensive, and time consuming.

Biochemical Data

Laboratory values of particular significance used in assessing nutritional status include serum proteins and lymphocytes. An individual's protein stores may indicate the degree of nutritional risk. Protein-energy malnutrition (PEM) may be reflected in low values for albumin, transferrin, transthyretin (prealbumin), retinol-binding protein, and total lymphocyte count. Blood levels of these markers indicate the level of pro-

Table 3. Common Biomarkers of **Nutritional Status and Inflammation**

Used with permission. See reference 28.

| Biomarker | Normal Range |
|----------------------------|---------------|
| Albumin | 3.5–5g/dL |
| Transferrin | 200–400 mg/dL |
| Prealbumin (Transthyretin) | 18–50 mg/dL |
| Retinol-binding protein | 3.0-8.0 mg/dL |
| C-reactive protein | 0-1.0 mg/dL |
| | |

tein synthesis and thus yield information on overall nutritional status. However, inadequate intake may not be the cause of low protein values. Certain disease states, hydration level, liver and renal function, pregnancy, infection, and medical therapies may alter laboratory values of circulating proteins.²⁷ It is important to note that a nutritional disorder diagnosis cannot be made from one single laboratory value but should be utilized with other assessment data to determine the nutritional status of the patient. The majority of laboratory values used in nutritional assessments lack sensitivity and specificity for malnutrition.²⁸ See Table 3.

Albumin

Comprising the majority of protein in plasma, albumin is commonly measured. The half-life of albumin is 14-20 days, which reduces its usefulness for monitoring the effectiveness of nutrition in the acute care setting. However, the general availability and stability of albumin levels from day to day make it one of the most common tests for assessing long-term trends and provides the clinician with a general idea of baseline nutritional status prior to a procedure, insult, or acute illness. Albumin levels often reflect the metabolic response and severity of disease, injury, or infection and can be a useful prognostic indicator. Albumin synthesis is affected by nutrition and also by inflammation. During an inflammatory state, the production of albumin diminishes. The effect of inflammation and hypoalbuminemia has been linked with increased morbidity, mortality, and longer hospitalization.²⁹

Transferrin

The transport protein for iron (transferrin) has a half-life of 8-10 days and, therefore, can be a better indicator of improved nutritional status than albumin. However, lack of iron influences its values along with a number of other factors, including hepatic and renal disease, inflammation, and congestive heart failure.³⁰

Transthyretin and retinol-binding protein

Transthyretin, also called prealbumin, and retinolbinding protein have a half-life of just two to three days and 12 hours, respectively. Each responds to nutritional changes much quicker than either albumin or transferrin. However, a number of metabolic conditions, diseases, therapies, and infectious states influence their values.30 Levels of transthyretin and retinol-binding protein are influenced by many factors other than nutritional status. Similar to albumin, their use is limited in the setting of stress and inflammation. Because these conditions are so common among the critically ill, visceral protein markers are of limited usefulness for assessing nutritional deficiency but are of greater importance in assessing the severity of illness and the risk for future malnutrition.

Total lymphocyte count

The immune system may be compromised by a lack of protein. Two laboratory values, white blood cells and percentage of lymphocytes, have been used as measures of a compromised immune system. However, many non-nutritional variables influence lymphocyte count; therefore, their usefulness in assessing nutritional status is limited.31

Biomarkers of inflammation

Biomarkers of inflammation are important values to measure along with serum proteins. The presence of inflammation affects the nutritional status of the patient. The inflammatory response increases the catabolic rate and causes albumin to leak out of the vascular compartment. Inflammation triggers a chemical cascade that causes a loss of appetite or anorexia, therefore decreasing dietary protein intake and further catabolism.²⁸

One of the most common biomarkers of inflammation used in clinical practice is C-reactive protein (CRP). The production of CRP increases with infection and inflammation along with pro-inflammatory cytokines (i.e., IL-1a, IL-1b, IL-6, TNF) while the production of albumin and prealbumin decreases.³² Other biomarkers of inflammation include prolactin, cholesterol, hyperglycemia, and ferritin. 33-35

Other Tests and Procedures

Creatinine-height index

Because the rate of creatinine formation in skeletal muscle is constant, the amount of creatinine excreted in the urine every 24 hours reflects skeletal muscle mass and can indicate muscle depletion. However, it requires an accurate urine collection and normal renal function. Other factors that influence creatinine excretion that can complicate interpretation of this index include age, diet, exercise, stress, trauma, fever, and sepsis.30

Nitrogen balance (protein catabolism)

Nitrogen balance reflects skeletal muscle, visceral or organ, blood cell, and serum protein stores. Because nitrogen is a major byproduct of protein catabolism, its rate of urinary excretion can be used to assess protein adequacy. The amount of nitrogen excreted in the urine is typically measured as the 24-hour urinary urea nitrogen (UUN). If there is a positive urinary nitrogen balance, protein metabolizing is sufficient, and nitrogen is excreted in the urine. A UUN value less than zero indicates a negative nitrogen balance, which indicates that the patient needs a higher protein intake. Theoretically, by increasing exogenous protein, loss of endogenous protein is reduced. However, because of invalid 24-hour urine collections, alterations in renal or liver function, large immeasurable insensible losses of protein from burns, high-output fistulas, wounds, ostomies, and inflammatory conditions, nitrogen balance calculations are generally negative and do not accurately reflect nutrition status.30

Pulmonary function

Pulmonary function test results may change with malnutrition. Weakness of the diaphragm and other muscles of inspiration can lead to a reduced vital capacity and peak inspiratory pressures. The strength and endurance of respiratory muscles are affected, particularly the diaphragm. Respiratory muscle weakness can affect the ability to cough and clear secretions, which may impact rates of pulmonary complications. Dietary antioxidants are thought to protect tissue from oxidant injury or stress, due to their ability to stabilize reactive molecules. Oxidative stress contributes to airflow limitation; therefore, antioxidant vitamins provide pulmonary antioxidant defense.36

Nutrition-focused physical findings

The nutritional-focused physical assessment is the evaluation of body systems, oral health, suck/swallow/breathing ability, and appetite, conducted by the Registered Dietitian or another member of the health care team as part of the nutritional assessment. 19 Physical examination can reveal observable signs of nutrition deficiencies where high cell turnover occurs, like the hair, skin, mouth, and tongue. 16 Signs of weight loss, including loss of lean body mass and subcutaneous fat, should be investigated. Special attention should be given to fluid retention as this can mask weight loss. ¹¹ Other physical findings such as skeletal muscle depletion can be clinical indicators of inflammation or signs of systemic inflammatory response.

Patient History

Interviewing the patient or the caregiver to determine past and current eating practices can be helpful. The patient's medical record can also reveal additional information regarding social, pharmaceutical, environmental, and medical issues. Much of this data can give insight into a patient's nutritional status. The patient's social history indicates marital status, employment, ed-

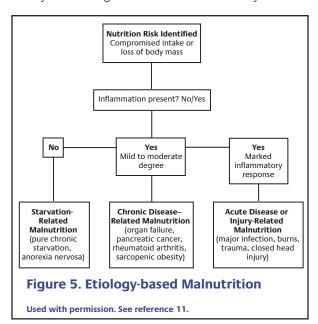
ucation, and economic status. Drug-nutrient interactions may be identified from the prescribed medications that lead to potential nutrient deficiencies. Environmental issues could point out the difficulties the patient has in procuring, storing, and/or preparing food. The education acquired by the heath care provider could determine the potential for understanding and applying nutrition counseling. The economic status of the patient may drive certain food choices. Much of the information can be helpful to raise suspicion and guide the investigation further into revealing the nutritional status of the patient.

Malnutrition

Once a nutritional assessment is completed, the degree and severity of malnutrition (if present) can be determined. Malnutrition is characterized by deficient, excess, or unbalanced nutrient intake. Malnutrition syndromes can be associated with acute or chronic inflammation. Etiology-based diagnosis of malnutrition falls into three categories: "starvation-related malnutrition," when there is chronic starvation without inflammation (e.g., secondary to anorexia nervosa), "chronic disease-related malnutrition," when inflammation is chronic and of mild-to-moderate degree (e.g., organ failure, pancreatic cancer, rheumatoid arthritis, or sarcopenic obesity), and "acute disease or injuryrelated malnutrition," when inflammation is acute and severe (e.g., major infection, burns, trauma, or closedhead injury).37 See Figure 5.11

Identification of two or more of the following is recommended for a diagnosis of malnutrition:

- Insufficient nutrient intake < 50–75% of estimated energy requirements over time
- Loss of weight
- Loss of muscle mass
- Loss of subcutaneous fat
- Localized or generalized fluid accumulation that may mask weight loss or loss of lean body mass



 Diminished functional status measured by handgrip strength.

Malnutrition is a major contributor to increased morbidity and mortality, decreased functional quality of life, prolonged duration of mechanical ventilation, increased length of hospital stay, and higher health care costs.^{3,11}

Undernutrition and Protein Energy Malnutrition

Undernutrition is a nutritional deficiency resulting from the lack of nutrient intake. Undernutrition suppresses immune function and is often a precursor of disease progression and/or worsening infection. Buring critical illness, proteolysis (muscle protein breakdown) increases, which can cause dietary protein needs to more than double. Failure to meet this increased protein requirement can lead to a state of protein energy malnutrition, which can be characterized by weight loss and muscle wasting. Difference of the suppression of the state of the state of the suppression of t

Overnutrition, Obesity, and Metabolic Syndrome

Overnutrition in the obese patient can lead to fluid overload, hyperglycemia, fatty liver deposits and liver dysfunction, and the need for prolonged ventilator support. 40 Obese individuals have a higher incidence of inflammation-associated chronic diseases, greater susceptibility to infection, 41,42 and have an increased risk of mortality. 43,44 Obesity-induced inflammation is an important contributor to the development of insulin resistance and hyperglycemia. 45 Obesity increases the risk and prevalence of asthma in both adults and children.46 Sarcopenic obesity is obesity associated with a decline in muscle strength and mass in elderly patients, which may further reduce physical activity and result in additional weight gain.47 The additional weight loading of the chest wall increases the work of breathing, reduces lung volume, decreases functional residual capacity, and can result in atelectasis, hypoxemia, and hypercapnia. Obese patients have a high

| Hypophosphatemia | Hypokalemia | Hypomagnesemia | Vitamin/Thiamine Deficiency | Sodium Retention |
|--|---|---|---|---|
| Impaired oxygen, transport and delivery, hypoxia Impaired cardiac function Impaired diaphragm contractility Respiratory failure Paresthesias Weakness Lethargy Somnolence Confusion Disorientation Restlessness Encephalopathy Areflexic paralysis Seizures Coma Death | Nausea Vomiting Constipation Weakness Paralysis Respiratory compromise Rhabdomyolysis Muscle necrosis Alterations in myocardial contraction Electrocardiograph changes ST-segment depression T-wave flattening T-wave inversion Presence of U-waves Cardiac arrhythmias Atrial tachycardia Bradycardia Atrioventricular block Premature ventricular contractions Ventricular tachycardia Ventricular fibrillation | Weakness Muscle twitching Tremor Altered mental status Anorexia Nausea Vomiting Diarrhea Refractory hypokalemia and hypocalcemia Electrocardiograph changes Prolonged PR Widened QRS Prolonged QT ST depression Peaked T-wave T-wave flattening Cardiac arrhythmias Atrial fibrillation Torsade de pointes Ventricular arrhythmi Ventricular tachycard Tetany Convulsions Seizures Coma Death | Encephalopathy (eg, Wernicke- Korsakoff encephalopathy) Lactic acidosis Death | Retention Fluid overload Puomonary edema Cardiac decompensation |

prevalence of obstructive sleep apnea and are prone to developing obesity hypoventilation syndrome. 48,49

The metabolic syndrome consists of a grouping of risk factors that have shown to be strongly associated with an increased risk for cardiovascular disease and the development of type-2 diabetes mellitus. Metabolic risk factors for metabolic syndrome consist of: hyperlipidemia, hypertension, hyperglycemia, a proinflammatory state, and a prothrombotic state. The predominant underlying risk factors include abdominal obesity and insulin resistance. 50 Obesity hypoventilation syndrome, obstructive sleep apnea, and congestive heart

failure are associated with the development of metabolic syndrome. $^{51-56}$

Refeeding Syndrome

Refeeding Syndrome is a term used to describe the complex metabolic and clinical disturbances that occur after the reinstitution of nutrition to patients who are severely malnourished or starved.⁵⁷

Clinical manifestations of refeeding syndrome are related to the resulting electrolyte and vitamin deficiencies cause by starvation and malnutrition, and the

subsequent abnormalities that develop once nutritional support is initiated. Refeeding after a period of malnutrition and starvation increases the basal metabolic rate, which results in major alterations in macronutrient metabolism. This leads to hypophosphatemia, hypomagnesemia, hypokalemia, and thiamine deficiency and can cause hyperglycemia during refeeding, decreased excretion of sodium and water, and an expansion of fluid compartments. The development of refeeding syndrome can result in severe cardiovascular and pulmonary complications. Cardiac arrhythmias and death have been seen in chronically malnourished patients receiving aggressive parenteral nutrition and early carbohydrate administration. Other significant complications include confusion, coma, and seizures. Congestive heart failure, pulmonary edema, diaphragm and intercostal muscle weakness, decreased tissue oxygen delivery, and increased carbon dioxide production can cause respiratory failure and can make weaning from mechanical ventilation more difficult.58,59 See Table 4.58

Factors that aid in the identification of patients at risk for refeeding syndrome include:57,60

- $BMI < 16-18.5 \text{ kg/m}^2$
- Unintentional weight loss >10-15% within last 3-6 months
- Little or no nutritional intake for >5-10 days
- A history of alcohol abuse or drugs, including insulin, chemotherapy, antacids, or diuretics
- Low levels of phosphorous, potassium, or magnesium prior to feeding
- Uncontrolled diabetes mellitus (diabetic ketoacidosis)
- Abused/neglected/depressed elderly adults
- Bariatric surgery
- Dysphagia
- Malabsorption (short bowel syndrome [SBS], inflammatory bowel disease [IBD], cystic fibrosis (CF), persistent nausea/vomiting/diarrhea, chronic pancreatitis)
- Chronic disease conditions (tuberculosis, HIV,
- Prolonged hypocaloric feeding or fasting
- Unconventional/eccentric diets.

Nutritional Support

The two routes of nutritional support are enteral and parenteral. Enteral nutrition (EN) is provided via the gastrointestinal tract, either by mouth or through a feeding tube. Parenteral nutrition (PN) is an intravenous solution composed of nutrients infused through an IV line that bypasses the gastrointestinal tract. Determination of the most appropriate route is influenced by the patient's nutritional risk, clinical diagnosis and condition, gastrointestinal tract function, and duration of anticipated need. See Figure 6.4

Parenteral Nutrition

PN provides nutrition to patients who are unable to digest or absorb sufficient nutrition via the gastroin-

testinal tract. These patients may include those with an obstruction, severe malabsorption, bowel hypomotility (ileus), or bowel ischemia (see Table 5). EN is the preferred modality over PN as it has been shown to have cost, safety, and physiologic benefits. EN may reduce disease severity, complications, and length of stay, and improve patient outcome.¹⁰

Administration of PN requires insertion of a central venous catheter or peripherally inserted central catheter (PICC). Due to the risks of catheter-related complications and infection, current recommendations suggest that PN should only be used if early EN is not feasible for the first 7–14 days following ICU admission, especially in patients who were previously

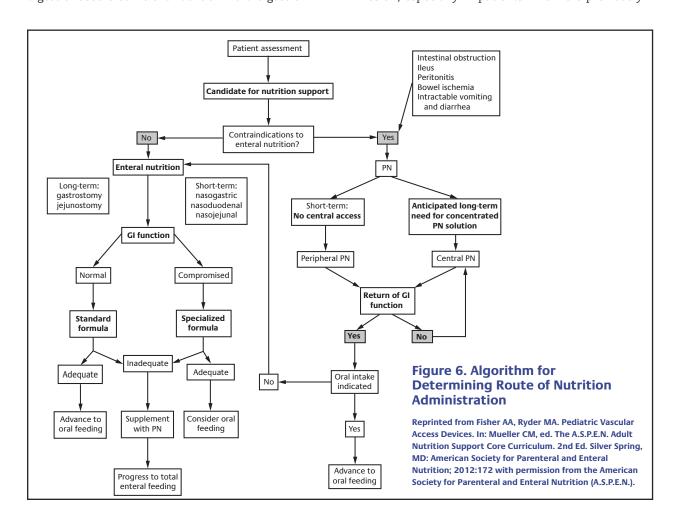


Table 5. Contraindications to Enteral Nutrition Support

- Nonoperative mechanical GI obstruction
- Intractable vomiting/diarrhea refractory to medical management
- Severe short-bowel syndrome (< 100 cm small bosel remaining)
- Paralytic ileus
- Distal high-output fistulas (too distal to bypass with feeding tube)
- · Severe GI bleed
- Severe GI malabsorption (eg, enteral nutrition failed as evidenced by progressive deterioration in nutritional status)
- Inability to gain access to GI tract
- Need is expected for < 5–7 days for malnourished adult patients or 7–9 days if adequately nourished
- Aggressive intervention not warranted or not desired

GI, gastrointestinal.

Reprinted from Fisher AA, Ryder MA. Pediatric Vascular Access Devices. In: Mueller CM, ed. The A.S.P.E.N. Adult Nutrition Support Core Curriculum. 2nd Ed. Silver Spring, MD: American Society for Parenteral and Enteral Nutrition; 2012:173 with permission from the American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.).

healthy prior to hospitalization. ¹⁰ In a recent multicenter, randomized controlled trial, later initiation of PN was associated with shorter length of ICU stay, fewer infections and other complications, and fewer days on mechanical ventilation when compared with early initiation. ⁶¹ Patients with evidence of moderate-to-severe malnutrition where EN is not an option should receive PN within the first few days following admission. ¹⁰

Parenteral nutrition formulations

PN is customized to individual patient needs for nutrients, electrolytes, vitamins, and trace elements by specially certified pharmacists through a process called compounding. Manual and automated compounding devices are available, but numerous cases of parenteral compounding errors in ordering, transcribing, compounding, and infectious complication have been reported. To address these problems, preparation of PN can be outsourced to specialized compounding pharmacies. Standardized, premixed, and commercial products are available. To improve the safe administra-

tion of parenteral nutrition, standardized procedures for ordering, labeling, nutrient dosing, screening orders, administering, and monitoring are recommended.⁶⁵

Enteral Nutrition

Short-term EN is typically administered via a nasally or orally inserted small bore weighted tip feeding tube called a "Dobhoff" tube. The weighted tip helps the tube travel past the stomach and through the pyloric valve into the duodenum and jejunum. Initial placement is performed with a guide wire inserted into the tube. Complications during insertion can include soft-tissue trauma and hemorrhage, esophageal perforation, and placement into the lungs. Definitive verification of tube placement is determined by chest radiograph.

Percutaneous endoscopic gastrostomy (PEG) or jejunostomy tubes placed surgically through the abdominal wall should be considered for long-term enteral feeding when nutritional support is expected for at least four weeks. ⁶⁶

Enteral nutrition formulations

Numerous EN formulations are available with various products designed for specific disease states such as renal failure, gastrointestinal disease, diabetes and hyperglycemia, hepatic failure, acute and chronic pulmonary disease, and immunocompromised states. See Table 6.

Unfortunately, most of these specialty products lack strong scientific evidence to promote routine use because of inconsistent, inconclusive, or unavailable clinical trial results. ^{67,68} Until clinical evidence becomes available, standard formulas should be used for the majority of patients requiring enteral feeding. In the critically ill, at-risk patient, evaluation of the nutritional needs, physical assessment, metabolic abnormalities, gastrointestinal (GI) function, and overall medical condition should be used to identify the enteral formula that will meet the individual patient requirements and determine product selection. ^{66,69}

Feeding Tube Placement — Gastric versus Post-Pyloric

There is an ongoing controversy in clinical practice regarding post-pyloric versus gastric feeding tube placement. Generally, in the intensive care unit it is preferred to place the feeding tube in the post-pyloric position due to the assumption that delayed gastric emptying results in a predisposition to bleeding, regurgitation, reflux, and aspiration. The clinical condi-

Table 6. Common EN Formulations Marketed for Patient and Disease-specific Conditions

| Enteral Formula Type | Description | Products | |
|----------------------|---|--|--|
| Standard Formulas | Meant to match nutrient requirements for healthy individuals Concentrations vary from 1.0-2.0 kcals/mL May or may not include soluble/insoluble fiber | Osmolite ¹ Jevity ¹ Promote ¹ TwoCal HN ¹ Nutren ² Isosource ² Fibersource ² Replete ² | |
| Diabetic | Lower carbohydrate with increased fat Contain more complex carbohydrates Concentrations vary from 1.0–1.5 kcal/mL | Glucerna ¹ Glytrol ² Diabetisource AC ² | |
| Renal | Generally lower in protein, calorically dense, and lower in potassium, magnesium and phosphorus May vary in protein, electrolytes, vitamins, and minerals depending on renal replacement therapy | Nepro ¹ Suplena ¹ Novasource Renal ² Renalcal ² | |
| Liver | Increased amounts of branched chain amino acids (BCAA) with decreased aromatic amino acids (AAA) Calorically dense, low in total protein, sodium, and fat-soluble vitamins and minerals | Nutrihep ² | |
| Pulmonary | Calorically dense, low in carbohydrates, and high in fat (COPD) Calorically dense, high omega-3 to omega-6 fatty acid ratio, antioxidants, (ARDS) | Pulmocare (COPD) ¹ Nutren Pulmonary (COPD) ² Oxepa (ARDS) ¹ | |
| Immune Modulating | Key ingredients include arginine, glutamine, nucleotides, and omega-3 fatty acids | Pivot 1.5 ¹ Impact ² | |
| Bariatric | Designed for critically ill morbidly obese patients Very high in protein Contains omega-3 fatty acids | Peptamen Bariatric ² | |
| Pediatric | Formulated for pediatric nutrient needs and conditions | Pediasure ¹ Elecare ¹ Nutren Junior ² Peptamen Junior ² | |

LEGEND: Abbott Nutrition¹, Nestle Health Science²

tion of the patient generally dictates the placement of the feeding tube. Patients who are at high risk for aspiration and delayed gut motility should be considered for post-pyloric small bowel access. Per ASPEN guidelines, these patients include those who have sustained severe blunt and penetrating torso and abdominal injuries, severe head injuries, major burns, undergone major intra-abdominal surgery, had a previous episode of aspiration or emesis, had persistent high gastric residuals, are unable to protect the airway, require pro-

longed supine or prone positioning, or are anticipated to have multiple surgical procedures.⁴

Post-pyloric feeding access can be difficult and may delay the introduction of EN. The repeated attempts of placement and using more advanced modalities such as fluoroscopy to determine placement can increase costs of providing care.⁷³

Multiple studies have not shown a significant difference in improved clinical outcomes with post-pyloric feeding tube placement. Meta-analysis of clinical out-

comes of several small sample size studies have evaluated mortality, incidence of pneumonia, and reducing aspiration risk. 74-76 The only clinical outcome that has been shown to have an improvement with post-pyloric feeding is an increase in the volume of targeted nutrient delivery.76

Current recommended practice is to target postpyloric feeding tube placement but not to delay gastric feeding unless clear signs of intolerance, aspiration risk, and high gastric residual volume are evident.

Gastric Residual Volume

The practice of measuring GRV is a standard nursing practice used to determine tolerance of gastric tube feedings. It is assumed that high GRV is correlated with an increased risk of reflux, aspiration, and pneumonia. However, little evidence exists in the literature correlating GRV with these risks.⁷⁷ GRV has not been shown to be a marker of aspiration. 78,79 Aspiration occurs in critically ill patients whether GRV is low or high, but aspiration risk may increase with high GRV.80 It has also been shown that GRV does not correlate with gastric emptying.81

The practice of checking GRV is time intensive, and small-bore feeding tubes often occlude during the process. 82 In fact, the practice of checking GRV may result in inappropriate cessation of EN and cause a decrease in nutrient delivery and accumulation of calorie deficit over time. Caloric deficit in already at-risk mechanically ventilated patients may increase complications and morbidity.83

In the absence of other signs of intolerance (such as emesis and abdominal distension), the most recent ASPEN/SCCM clinical practice guidelines for holding or reducing enteral nutrition is a GRV of 500 mL. 10,84 This highly controversial recommendation is supported by several studies that show that a higher tolerable GRV was not associated with an increase in adverse events such as regurgitation, emesis, and aspiration.85-88 Higher GRV in combination with prokinetic agents to promote bowel motility have been shown to improve nutrient volume administered and reduce the time to reach target goals without increasing complications.86-89 Two prospective studies compared routine GRV monitoring to not checking GRV and also found no difference in adverse events. 89,90 Therefore, the controversial practice of tolerating a higher GRV of 500 mL is supported by current evidence.

Regardless of the acceptable GRV used by an institution, the following practices have been proven to reduce the risk of aspiration: 10,84

Head of bed elevation to 30°

- · Use of bowel motility agents such as metoclopramide
- Post-pyloric or small-bowel feeding tube placement when indicated.

Trophic Feedings

Low-dose, "trickle," or trophic feeding is the practice of feeding minimal amounts (10-30 mL/hr) of EN with the primary goal to maintain gut function and integrity despite not meeting daily caloric needs. It is most often used in preterm infants on PN⁹¹ or in adult patients with impaired enteral feeding tolerance or gut function. EN stimulates organs of digestion to function in their normal capacity and to assist in the digestion and absorption of nutrients. It also prevents passage of bacteria across the GI tract into the systemic circulation, reducing infection rates, enhancing immune function, and preserving GI mucosal structure and function.30 Trophic feeding may also reduce the development of a postoperative ileus. 10,84,92 Studies in mechanically ventilated patients with respiratory failure or ARDS show that trophic feedings resulted in fewer episodes of gastrointestinal intolerance but resulted in similar clinical outcomes compared to early advancement to full enteral feeding.93,94

Stress Prophylaxis

Critically ill patients are at risk of GI bleeding from gastric or duodenal ulcers due to increased gastric acidity and decreases in the gastric mucosal barrier. EN can improve mucosal blood flow and reverse the production of inflammatory mediators that cause gastropathy. EN may provide stress prophylaxis and help to reduce the use of acid-suppressive therapy in the ICU.95 Additional randomized controlled trials and protocols are needed to investigate this further.

Nutrient Requirements and Distribution

The purpose of a nutritional assessment is to determine a nutrition care plan with the primary goal of meeting the nutritional requirements of the patient. This includes determination of total energy, protein, carbohydrate, fat, and micronutrient needs.

Carbohydrate requirements

Carbohydrates are the primary fuel source for the body. It is recommended that approximately 45-65% of total calories come from carbohydrates. A minimum daily amount of 100-150g/day is necessary to provide adequate glucose to the brain. If consumed in insufficient amounts, an accumulation of ketone bodies develops as a result of excessive fat and protein catabolism, and acidosis occurs.⁹⁶

Protein requirement

Amino acids or proteins are essential to maintaining or restoring lean body mass. Because illness usually increases protein catabolism and protein requirements, the recommended dietary allowance (RDA) of 0.8 g/kg per day is generally insufficient for critically ill patients. Based on the assessment of the protein catabolism rate, protein intake may need to be doubled or even tripled above the RDA (1.5 to 2.5 g/kg/day). Ideally, approximately 20% of a patient's estimated calorie needs should be provided by protein. Higher percentages of protein may be needed in patients with "wasting syndrome" or cachexia, elderly persons, and persons with severe infections. However, whenever high protein intakes are given, the patient should be monitored for progressive uremia or azotemia (rising BUN > 100 mg/dl).96 Too much protein is harmful, especially for patients with limited pulmonary reserves. Excess protein can increase O2 consumption, REE, minute ventilation, and central ventilatory drive. 97 In addition, overzealous protein feeding may lead to symptoms such as dyspnea in patients with chronic pulmonary disease.

Fat requirements

The remaining calories (20–30%) should be provided from fat. A minimum of 2–4% is needed to prevent essential fatty acid deficiency. Fat intakes in excess of 50% of energy needs have been associated with fever, impaired immune function, liver dysfunction, and hypotension.⁹⁶

Vitamins, minerals, and electrolytes

The dietary reference intakes (DRI) provide the recommended optimal level of intake for vitamins, minerals, and electrolytes. The primary goal is to prevent nutrient deficiencies as well as help reduce the risk of chronic diseases. Some nutrients may need to be supplemented above the DRI for certain disease states, therapies, or conditions. ⁹⁶

Fluid requirements

Fifty to sixty percent of body weight consists of water. Fluid requirements are estimated at 1 ml/kcal/day or 20–40 ml/kg/day. Depending on a patient's medical condition, fluid restriction may be warranted. Additional fluid may be required for excessive fluid losses (urinary, fecal, blood, wound, emesis) and with excessive insensible losses (fever). 96

Nutrition Support and Respiratory Function

Patients with acute and chronic respiratory failure may present with or have the potential to develop nutrition-related complications. Nutrition support plays a significant role in treatment as further deterioration can have a direct effect on respiratory function, further decline, and poor outcomes. 98 Specific nutrition recommendations exist for intervention and treatment of acute and chronic respiratory failure.

Respiratory consequences of malnutrition may include the following:⁹⁹

- Loss of diaphragmatic and accessory muscle mass and contractility
- Ineffective cough
- Decreased maximum expiratory pressure and maximum inspiratory pressure
- Decreased FVC or FEV₁
- Reduced production of surfactant 100
- Fluid imbalance
- Congestive heart failure
- Decreased lung compliance, atelectasis, and hypoxemia
- Decreased hypoxic and hypercapnic response^{101–103}
- Increased CO₂ production
- Increased incidence of hospital-acquired infections¹⁰⁴
- Decreased lung clearance mechanisms
- Increased bacterial colonization
- Emphysematous changes to lung parenchyma^{105,106}

Chronic Obstructive Pulmonary Disease

Disease-related malnutrition is common in patients with chronic obstructive pulmonary disease (COPD). Between 30–60% of inpatients and 10–45% of outpatients with COPD are at risk for malnutrition. Malnourished COPD patients exhibit a higher degree of gas trapping, reduced diffusing capacity, and a diminished exercise tolerance when compared to patients with normal body weight, adequate nutrition, and comparable disease severity. The underlying mechanism between malnutrition and COPD is thought to be from a variety of contributing factors (see Figure 7).

Malnutrition may be responsible for the respiratory muscle wasting, which intensifies the progression of COPD or may simply be a consequence of disease severity. Similarly, long-term caloric malnutrition is associated with the loss of body weight that includes an extensive loss of lung tissue and reduction in diffusion

capacity. Emphysematous-like changes are found to occur in persons with chronic anorexia nervosa and those who die of starvation. 105,106

In COPD patients with acute respiratory failure, malnutrition may have detrimental effects, especially in weaning from ventilatory support. 110 Malnutrition is associated with a decrease in diaphragmatic muscle strength, 111 a decrease in ventilatory drive, 102 reduced surfactant production, 112 and an increased risk of nosocomial pneumonia. 104,107 Protein energy malnutrition is common in COPD patients. 113 Early and aggressive nutritional support in COPD patients can produce significant improvements in several functional outcomes including respiratory and limb muscle strength.

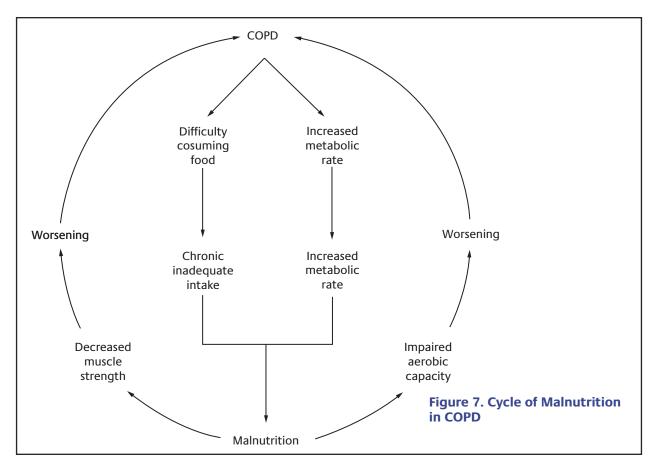
Increased protein intake may improve ventilatory response to CO₂. 102 Several meta-analyses of nutritional support studies have demonstrated improved nutrition related to anthropometric improvements, 114 inspiratory and expiratory muscle strength, exercise tolerance, and quality of life. 107 The most recent Cochrane systematic review found evidence of significant improvements in weight gain, indices of respiratory muscle strength, walking distance, and quality of life in malnourished

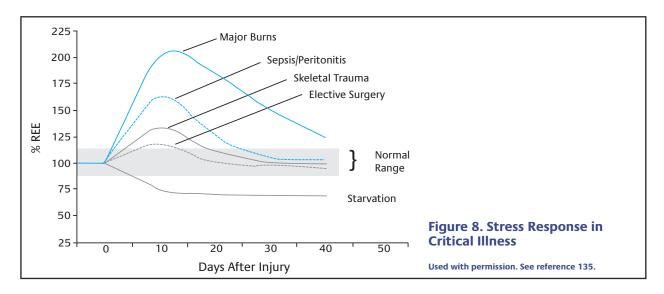
COPD patients who received nutritional supplementation.115

CO₂ is produced with the metabolism of all macronutrients, with the largest amount coming from carbohydrates. It is well known that overfeeding with an excess carbohydrate load increases CO₂ production. However, overfeeding with non-carbohydrate calories can be as detrimental in regards to CO₂ production and the increased work of breathing. 116 A high-fat, reduced carbohydrate nutrition formulation has been marketed in an effort to encourage the benefits of nutrition repletion and weight gain while reducing CO2 production:117 however, several studies have refuted this theoretical benefit, and the practice is not recommended. 118-123

Underlying causes of malnutrition

Underlying causes of malnutrition in COPD patients include increased energy expenditure due to increased caloric cost of breathing, increased systemic inflammation, and the thermogenic effect of medications such as bronchodilators. Also, COPD patients have an inadequate caloric intake caused by dyspnea while eat-





ing, chewing and swallowing difficulties, taste alterations and suppressed appetite from medications, the use of a nasal cannula or tracheostomy, and early satiety.⁹⁹

Psychosocial factors are also underlying causes that contribute to malnutrition in COPD patients. Depression, poverty, difficulty shopping, and tiring easily when preparing food often prevent good nutrition.⁹⁹

Acute Respiratory Distress Syndrome

Acute respiratory distress syndrome (ARDS) is acute pulmonary failure that manifests from inflammatory conditions. Omega-3 fatty acids are metabolized to substances that reduce inflammation and inflammatory mediator production. Enteral supplementation with omega-3 fatty acids may have a beneficial effect in treatment for ARDS. Several studies observed reduced duration of mechanical ventilation, number of days in the ICU, rates of organ failure, and mortality compared to use of standard enteral formulas. 10,124–127

Omega-6 fatty acids are metabolized to proinflammatory substances that influence cytokine production, platelet aggregation, vasodilation, and vascular permeability, and therefore may be harmful. 128–132

Nutritional support high in omega-6 fatty acids should be avoided.¹²⁸ Nutritional supplementation with higher omega-3 to omega-6 fatty acid ratios have been recommended to reduce the risks of inflammatory disorders such as coronary heart disease, diabetes, arthritis, cancer, osteoporosis, rheumatoid arthritis, and asthma.^{129,131} Due to conflicting results from the more recent ARDSNet Trial, the practice of omega-3 supplementation remains controversial.^{133,134}

Nutritional Support During Critical Illness

The general goals of nutritional support in the critically ill patient are to provide the energy and protein necessary to meet metabolic demands and to preserve lean body mass. Nutritional support is also an important therapy in critical illness as it attenuates the metabolic response to stress, prevents oxidative cellular injury, and modulates the immune response. Nutritional modulation of the stress response includes early enteral nutrition, appropriate macro and micronutrient delivery, and meticulous glycemic control.¹²⁷

Stress Response in Critical Illness

Metabolic needs vary during critical illness. The metabolic response to critical illness occurs in three phases: the stress phase, the catabolic phase, and the anabolic phase. The stress phase typically lasts for 24-48 hours and is characterized by hypovolemic shock, hypotension, and tissue hypoxia. See Figure 8. Hypometabolism and insulin resistance is also seen. The primary goal during this time period is resuscitation and metabolic support. Metabolic support may consist of permissive underfeeding. Permissive underfeeding is where patients are fed below their REE, and the primary goal is to support cellular metabolic pathways without compromising organ structure and function. 136 The catabolic phase occurs after resuscitation. In hypercatabolism, increased oxygen demands, cardiac output, and carbon dioxide production are seen. This phase usually lasts 7-10 days, and the goal is to provide ongoing metabolic support with high-protein feedings while avoiding overfeeding. Caloric needs may be increased

Table 7. Consequences of Over- Underfeeding

Overfeeding

Axotemia

Physiologic stress
Respiratory compromise
Prolonged mechanical ventilation
Hyperosmolar state
Hyperglycemia
Hepatic dysfunction
Excessive cost
Immune suppression
Fluid overload

Underfeeding

Increased complications
Immune suppression
Prolonged hospitalization
Respiratory compromise
Poor wound healing
Nasocomial infection
Prolonged mechanical ventilation

Used with permission. See references 139-141.

by up to 100% during the catabolic phase in patients with severe burns. ^{135, 137} As the catabolic phase resolves, the anabolic phase begins and can last for months. Caloric needs may remain elevated during the anabolic phase for repletion of lean body mass and fat stores.

Under- and Overfeeding During Critical Illness

Providing inadequate provision of nutrients can have negative effects on the critically ill patient (see Table 7). Underfeeding can result in a loss of lean body mass, immunosuppression, poor wound healing, and an increased risk of infection. ¹⁴⁰ This can also result in an inability to respond to hypoxemia and hypercapnia, and a diminished weaning capacity. ¹⁴¹ Continual underfeeding in the ICU results in a cumulative caloric deficit, which increases length of stay, days of mechanical ventilation, and mortality. ⁸³

Overfeeding patients can be equally detrimental as well. Excess amounts of nutrients can exacerbate respiratory failure by increasing carbon dioxide. Lacess total calories (not excess carbohydrates) increase $\rm CO_2$ production and, therefore, increase the work of breathing. If under- or overfeeding is suspected, indirect calorimetry is an important tool to help determine accurate energy requirements.

Systemic Inflammatory Response Syndrome

The systemic inflammatory response syndrome (SIRS) underlies many critical illnesses, including sepsis and ARDS. Metabolism in SIRS is characterized by increased total caloric requirements, hyperglycemia, triglyceride intolerance, increased net protein catabolism, and increased macronutrient and micronutrient requirements. Requirements for micronutrients are

also increased in SIRS. Because of the potential high losses of potassium, zinc, magnesium, calcium, and phosphorus, serum levels of these minerals need to be closely monitored and maintained within the normal range. 142

Glycemic Control in Critical Illness

Control of serum glucose levels in non-diabetic patients during critical illness is important due to the adverse effect of hyperglycemia in patient outcomes. Control of hyperglycemia has been shown to reduce morbidity and mortality in hospitalized patients. Hyperglycemia is a normal response to physiologic stress and the inflammatory response related to critical illness. Since hyperglycemia can be caused by enteral and parenteral nutrition, control of hyperglycemia during nutritional support is of critical importance. The stress response to critical illness causes wide swings in nutrient requirements. Therefore, the nutritional support process needs to balance the potential detrimental effects of both under- and overfeeding with glycemic control. Current recommendations are to maintain a target blood glucose goal range of 140-180 mg/dL and to consider a blood glucose value of <70 mg/dL during nutritional support as a treatable hypoglycemia. 143-146

Permissive Underfeeding

Permissive underfeeding is recommended for the critically ill obese patient. Guidelines suggest the goal of EN should not exceed 60–70% of target energy requirements with a high protein goal of 2.0–2.5 g/kg of ideal body weight. ¹²⁷ It is essential to provide adequate protein in these patients to maintain nitrogen balance and lean body mass while encouraging the use of adipose

tissue for fuel. Morbidly obese patients receiving high protein through permissive underfeeding have reduced insulin resistance, lower insulin requirements, better glycemic control, decreased ICU stay, and reduced duration of mechanical ventilation. 147-149

The practice of permissive underfeeding should avoid global starvation of protein and nutrients and the development of protein energy malnutrition. The role of permissive underfeeding where total calories are reduced while compensating with increased protein intake may have generalized benefits in critically ill patients. 150

Permissive underfeeding has been shown to reduce infection, 151 decrease hospital and ventilator days, 151 decrease the incidence of hyperglycemia, 152 and also trends toward decreased mortality. 152-154

Pediatric Critical Illness

Optimizing nutritional therapy in pediatric patients can improve clinical outcomes. As in the adult, the goals of pediatric nutrition support encompass preservation of tissue stores and resolution of disease progress. A recent multicenter international study demonstrated that pediatric intensive care units that used protocols for starting and advancing EN support had a higher percent of target calorie goals administered, reduced 60-day mortality, and lower rates of acquired infections. Also, use of PN was associated with higher mortality. 155 However, the goals of pediatric nutrition are more complicated than those for adult patients. In addition, needs that address the support for appropriate growth and development, and the need for preservation of oral motor skills should be considered. 156 Components of energy requirements in pediatrics consist of basal or resting energy expenditure, activity, growth, gender, maintenance of normal body temperature, and stress factors. Requirements for vitamins and minerals vary based on age, medical status, and size of the child. 157

Immunonutrition

Trauma, surgery, burns, and large wounds

Critical illness is often complicated by systemic inflammation and generalized immunosuppression. Both of these conditions can be responsive to immunonutrition therapy. Immunonutrition using immune modulating nutrition formulations containing omega-3 fatty acids, arginine, glutamine, nucleotides, and antioxidants are used with the goal to modulate the immune system, promote wound healing, attenuate the inflammatory response, and improve organ function.4 The use of these formulas in surgical patients has been shown to decrease risk for infections, reduce length of stay, and reduce mortality. 158 However, caution is advised within the use of arginine in critically ill severe septic patients. 158

Determining Nutritional Requirements

Calculating, estimating, or measuring the number of calories required by an individual determines nutrient requirements. A calorie is a unit of energy equivalent to the amount of potential heat produced or contained in food when released during the metabolic oxidation processes of the body. A calorie is defined as the amount of heat needed to raise the temperature of 1 gram of water by 1 C° (also called a small calorie, abbreviated as cal). A calorie (also called a large calorie, abbreviated as Cal) is defined as the amount of heat needed to raise the temperature of 1 kilogram of water by 1 C°, is equivalent to 1,000 calories and is also referred to as a kilocalorie. Kilocalories (kcal) are used to quantify the energy value of foods.

Calorie or energy needs are fundamental to the recommendations of the nutritional care plan. Macronutrients supply the body's energy requirements. The calorie contribution of the three major macronutrients are: protein = 4 kcal/g; carbohydrate = 4 kcal/g; and fat = 9 kcal/g. Alcohol is the only other calorie source with approximately 7 kcal/g.

Energy needs vary according to activity level and state of health. Energy needs of critically ill patients can be significantly different than normal values. Additional factors such as energy expended in catabolic states may be needed to adjust the estimate in patients with medical conditions such as injury, major wounds, and infection. ¹⁶⁰ Energy needs for obese individuals are less because adipose tissue uses less energy than muscle uses.

The estimated energy requirement is the average dietary energy intake needed to maintain energy balance in an individual. ¹⁶¹ Estimating energy requirements for people according to their age, sex, weight, height, and level of physical activity is accomplished by the use of predictive equations.

Predictive Equations

Numerous equations have been developed to predict caloric requirements. The Harris Benedict Equation (HBE), the most well known predictive equation, was developed in 1919 by comparing measured calories and their correlation to height, weight, age, and gender in normal subjects to estimate the basal meta-

bolic rate (BMR). BMR is defined as the amount of heat produced in a state at rest with complete muscle inactivity during a post-absorptive period 12–14 hours after the last meal. Since BMR as defined by Harris and Benedict is not necessarily reflective of the way nutritional requirements are determined in hospitalized patients, the more relevant terms — resting metabolic rate (RMR) or resting energy expenditure (REE) — are used in clinical practice to predict or measure caloric needs.

Predictive equations use factors validated by the original work by Harris and Benedict and incorporate additional factors such as temperature, body surface area, diagnosis, and ventilation parameters, as shown in Table 8.¹⁶²

Predictive equations have been modified as additional data (such as injury-stress, activity, medications received, and obesity) and have been added to the regression correlation equations. Several predictive equations were developed with a focus on specific patient populations and medical conditions.

Predictive equations have varying degrees of agreement compared to measured calorie requirements. Error rates are not insignificant and, therefore, can have high degrees of under- and overestimation of caloric needs. This variability can result in errors large enough to impact outcomes. 163 Error rates with some equations make them unsuitable as assessment methods of energy expenditure in critically ill patients. A recent systematic review comparing measured calorie requirements show that the Penn State 2003, Ireton-Jones 1992, and Swinamer equations may be useful in critically ill non-obese patients. The Mifflin-St Jeor, Penn State 1998, and Ireton-Jones 1992 equations might also be useful in obese patients. 163 Many of the studies were compared for accuracy and currently, per the Academy of Nutrition and Dietetics (AND), The Mifflin-St Jeor equation was found to be the most reliable, predicting REE within 10% of measured in more non-obese and obese individuals than any other equation, and it also had the narrowest error range. 164 However, error rates can still be significant regardless of the prediction method used. For example, in obese patients, there are no clinical fea-

Table 8. Examples of Commonly Used Predictive Equations

American College of Chest Physicians Equation

25 x weight

If BMI 16-25 kg/m2, use usual body weight.

If BMI > 25 kg/m2, use ideal body weight.

If BMI < 16 kg/m2, use existing body weight for the first 7–10 days, then use ideal body weight.

Harris-Benedict Equations

Men: Resting metabolic rate (RMR) = 66.47 + 13.75(W) + 5(H) - 6.76(A)

Women: RMR = 655.1 + 9.56 (W) + 1.7 (H) -4.7 (A)

Equation uses weight (W) in kilograms (kg), height (H) in centimeters (cm), and age (A) in years.

Ireton-Jones Energy Equations (IJEE) 1992

Spontaneously breathing IJEE (s) = 629 - 11(A) + 25(W) - 609(O)

Ventilator dependent IJEE (v) = 1925 - 10(A) + 5(W) + 281(S) + 292(T) + 851(B)

Equations use age (A) in years, body weight (W) in kilograms, sex (S, male = 1, female = 0), diagnosis of trauma (T, present = 1, absent = 0), diagnosis of burn (B, present = 1, absent = 0), (O) obesity more than 30% above initial body weight from 1,959 Metropolitan Life Insurance tables or body mass index (BMI) more than 27 kg/m2 (present = 1, absent = 0).

Mifflin-St. Jeor

Men: RMR = (9.99 X weight) + (6.25 X height) – (4.92 X age) + 5

Women: RMR = (9.99 X weight) + (6.25 X height) - (4.92 X age) - 161

Equations use weight in kilograms and height in centimeters.

Penn State Equation (PSU 2003b)

RMR = Mifflin(0.96) + VE(31) + Tmax(167) - 6212

Used for patient of any age with BMI below 30 or patients who are younger than 60 years with BMI over 30. This equation was validated in 2009 by the AND Evidence Analysis Library.

Penn State Equation (PSU 2010)

RMR = Mifflin(0.71) + VE(64) + Tmax(85) - 3085

Also known as the Modified Penn State Equation

Used for patients with BMI over 30 and older than 60 years. Validated in 2010 by the AND Evidence Analysis Library.

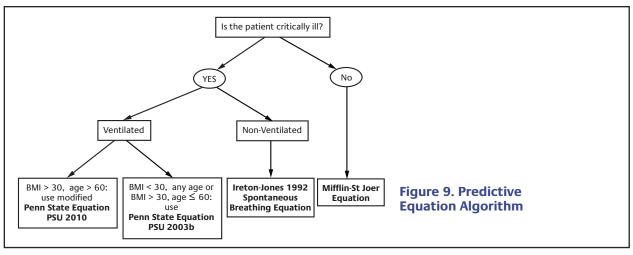
Used with permission. See reference 162.

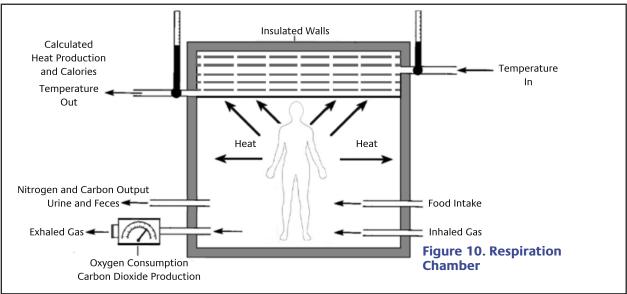
tures that can identify individual patients where a predictive equation is inaccurate. 165

There are more than 200 predictive equations in existence. Many were developed as long as 50–80 years ago and may not reflect body composition, nutritional risks, age, or ethnicity of the populations they are applied to. There is often no consensus on how a predictive equation is selected, and results can vary significantly between clinicians. ¹⁶⁶ Furthermore, there are large segments of populations in whom predictive equations have no validation studies preformed. These groups include the elderly and many non-white

racial groups. The limitations and variability of predictive equations when applied to an individual patient accentuates the need to use a regimented nutritional risk assessment process and sensible clinical judgment when deciding whether to use a predictive equation. Figure 9 provides an example algorithm for using predictive equations.

Kilocalories/kilogram calculation. The American College of Chest Physicians' 1997 equation is a simple and prompt method to estimate daily energy needs of the average adult using a factor of 25–35 kcals/kg. This





method is not necessarily as accurate as predictive equations, as it does not take into account gender, age, stature, and severity of illness. To rapidly estimate the energy needs of the average adult in kcal/day, identify the target goal for weight change and multiply the individual's actual body weight in kilograms times the factor listed as follows:¹⁶⁷

| Goal | Energy Needs (kcal/kg) |
|--------------------|------------------------|
| Weight maintenance | 25 to 30 |
| Weight gain | 30 to 35 |
| Weight loss | 20 to 25 |

To overcome the limitations of predictive equations and estimating formulas, energy needs can be measured at the bedside using calorimetry to measure RMR or REE by calorimetry.

Calorimetry

Calorimeters measure heat released from chemical reactions or physical changes. Calorimetry has been used since the late 19th and early 20th centuries and was adopted as the major method of determining energy needs in individuals. Calculations of calorie requirements by mathematical equation were developed from the use of direct and indirect calorimetry.

Direct calorimetry

Direct calorimeters measure heat. A bomb calorimeter measures the energy value of food by measuring the precise amount of heat liberated as the food is burned in a closed chamber. Another type of direct calorimeter requires that the subject be enclosed in a sealed chamber for extended periods and a precise measurement of heat transfer conducted. The early experimentation

conducted by nutrition scientists led to the development of the respiration chamber and IC.

Respiration chamber

The development of the respiration chamber combined the process of direct calorimetry with measurements of oxygen consumption (VO₂) and carbon dioxide production (VCO₂). See Figure 10.

Around the turn of the century, the correlation of heat production in calories, the rate of VO_2 and VCO_2 , the quantity of nutrients consumed, and the mass of carbon and nitrogen excreted was used to derive the caloric value of oxygen and carbon dioxide. ^{168–172} By simultaneous measurement of the ratio VCO_2 to VO_2 , the respiratory quotient (see Table 9) and caloric equivalent of each gas (see Table 10) in relation to the oxidation of specific food substrates could be determined.

It was observed that during short observational periods, the errors in computation of heat production and calories using indirect measurements of VO_2 and VCO_2 were less than the errors in computation when using direct calorimetry measurements. ¹⁷⁰ Direct calorimetry and respiration chambers in relation to metabolic test-

Table 9. RQ Substrate Interpretation: Interpretation of Substrate Utilization Derived from the Respiratory Quotient

| Substrate Utilized | Respiratory Quotient |
|--------------------------------------|----------------------|
| Ethanol | 0.67 |
| Ketones | 0.67 |
| Fat oxidation | 0.71 |
| Protein oxidation | 0.80-0.82 |
| Mixed substrate oxidation | 0.85-0.90 |
| Carbohydrate oxidation | 1.0 |
| Lipogenisis | 1.0-1.3 |
| Used with permission. See references | s 173-175. |

ing to this day primarily remain as a research tool in animals.

Indirect calorimetry

Indirect calorimetry is the most accurate method for determining RMR and REE in various states of health and disease and is considered to be the gold standard for measuring energy expenditure in critically ill patients. ^{179,180} Indirect calorimetry relies on the determination of VO₂ and VCO₂ using precise measurements from a metabolic analyzer of the inspired and expired fractions of oxygen and carbon dioxide where:

$$VO_2$$
 (mL/min) = (Vi x FiO₂) - (Ve x FeO₂) (1) and

$$VCO_2$$
 (mL/min) = (Ve x FeCO₂) - (Vi x FiCO₂) (2)

The abbreviated Weir equation uses the measured VO₂ and VCO₂ to determine REE where:

REE =
$$(3.9 \times VO_2) + (1.1 \times VCO_2) \times 1.44$$
 (3)

The respiratory quotient, the ratio of VCO_2 to VO_2 , can then be calculated where:

$$RQ = VCO_2/VO_2 \tag{4}$$

Since the normal RQ = 0.85, the volume of CO_2 produced is lower than the volume of O_2 consumed. Therefore, small differences in the inhaled versus exhaled volumes occur. In order to accurately calculate VO_2 and VCO_2 , the gas concentration measurements of a metabolic analyzer need to be within \pm 0.01%. In regards to VO_2 measurements, elevated FiO_2 introduces error as the oxygen concentration approaches 1.0. As a result, the accuracy of IC diminishes as FiO_2 increases. Additionally, any error in gas concentration analysis or delivery is amplified at a higher FiO_2 . Due to this technical

| Table 1 | 0. Ca | loric | Equival | lence |
|---------|-------|-------|---------|-------|
|---------|-------|-------|---------|-------|

| Substrate | Respiratory Quotient | Oxygen Caloric Equivalent (kcal/L) | Carbon Dioxide Caloric Equivalent (kcal/L) |
|--------------|-------------------------|--|--|
| Carbohydrate | 1.0 | 5.05 | 5.05 |
| Mixed | 0.90 | 4.83 | 5.52 |
| Protein | 0.80 | 4.46 | 5.57 |
| Fat | 0.71 | 4.74 | 6.67 |

limitation, IC is not recommended or considered to be accurate at $FiO_2 > 0.60$.^{7,173,181}

Respiratory quotient was once thought to be useful as a means to determine nutritional substrate utilization. However, the accuracy of this assumption has never been substantiated. The large stores of CO₂ in the body can be mobilized with ventilation and, thus, would reflect an increase in CO2 excretion but not necessarily production. An increase in VCO₂ measured as a result of this mechanism would have an erroneous effect on the measured RQ. See Figure 11.173

Therefore, the use of the RQ measurement is of limited clinical value. Measured values of RO between the physiologic ranges of 0.67-1.3 should be used as a means of quality control and a way to verify test validity. Values of RQ outside of this range obtained during IC testing invalidate the results due to technical measurement errors and should be repeated.¹⁷³

Indirect calorimetry is performed using a standalone metabolic cart by hood, face mask, and mouthpiece, or by connection to a ventilator. See Figure 12.

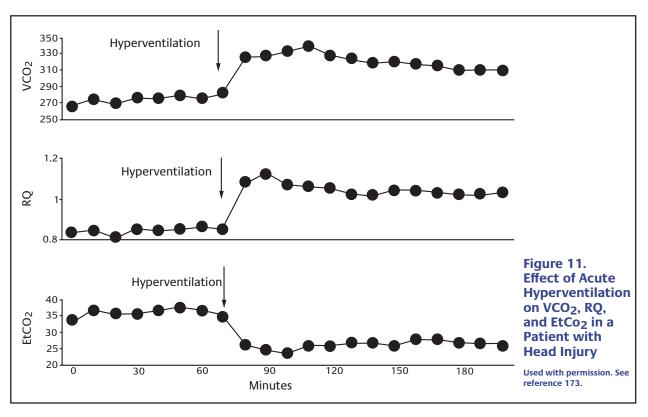
Open circuit systems sample inspired gas concentrations, measured expired gas concentrations, and expired minute volume collected back into the analyzer to determine VCO2, VO2, and RQ. Indirect calorimetry has also been integrated into several ventilators. 182,183 See Table 11. Handheld calorimeters are also available. 184-186 See Figure 13.

Newer open-circuit breath-by-breath designs use a system where inspired and expired gases and volumes are measured at the airway, which simplify the measurement procedure. See Figure 14.

Accuracy of indirect calorimetry measurements are dependent on the technical aspects of test performance and patient care related variables.

Technical considerations when performing IC measurements during mechanical ventilation include:99,187

- Warm-up time of 30 minutes for the indirect calorimeter
- Errors in calibration of flow, oxygen, and carbon dioxide sensors
- Presence of leaks (ventilator circuit, artificial airway, broncho-pleural fistulas)
- $FiO_2 > 60\%$
- Fluctuation of $FiO_2 > \pm 0.01\%$
- Changes in the ventilator setting within 1 to 2 hours of testing
- Acute hyperventilation or hypoventilation (changes body CO2 stores)
- Moisture in the sampling system
- Bias flow through the ventilator may affect accuracy of indirect calorimeter



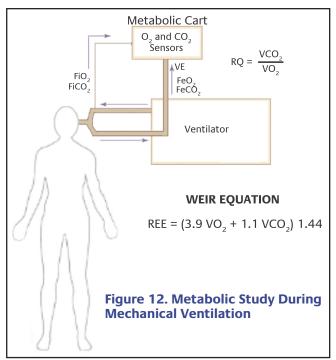






Figure 14. CCM Express indirect calorimeter can be used on intubated and non-intubated patients.

Courtesy of Medical Graphics Corp., St Paul, MN

Figure 13. MedGem handheld indirect calorimeter measures oxygen consumption to calculate resting energy expenditure using the caloric equivalent of oxygen.



Courtesy of Microlife USA Inc., Clearwater, FL

- · Attachment of indirect calorimeter may affect ventilator function
- · Use of inhaled nitric oxide
- Presence of anesthetic gases

Recommendations for improving accuracy of IC include:187

- Patient should be hemodynamically stable.
- Patient should be in a comfortable resting position for 30 minutes before the study.
- · Avoid instability caused by disconnection from high levels of positive end-expiratory pressure (PEEP).
- Avoid voluntary activity for 30 minutes. ^{175,188,190}
- · Avoid intermittent feedings or meals taken four hours before study. 175,190
- · Nutrient infusion should remain stable for at least 12 hours before and during the study. 175,190
- · Measurements are made in a quiet, neutralthermal environment. 175,188-190
- · Limited voluntary skeletal muscle activity during the study. 175,188-190
- Use of steady state data (coefficient of variation ≤ 10%). 175,188-190
- · No general anesthesia within six to eight hours before the study. 175
- Analgesics or sedatives for pain or agitation given at least 30 minutes prior to study. 175,188-191
- Delay study for three to four hours after hemodialysis. 175,188,192
- · Delay study for one hour after painful procedures.175
- Delay study for unstable body temperature.
- · Routine care or activities avoided during the study. 175,189

Table 11. Advantages and Disadvantages of Ventilators with Integrated Indirect Calorimetry. See Figure 15

Advantages

- Enables real-time monitoring of metabolic stress during critical illness
- Allows frequent repeatable measurements for closer monitoring of caloric balance
- Disconnection from ventilator when on high PEEP levels may be avoided
- Reduces interference with ventilator function from attachment of an external device
- Reduces infection risks from moving metabolic cart between patients
- Fick cardiac output estimates are more accessible

Disadvantages

- Limited use to a single patient attached to the ventilator
- Cost of installation on several ventilators may be prohibitive
- Difficult or unable to use on patients without an artificial airway (hood, face mask, and mouth piece studies)

Indications for Indirect Calorimetry

Indirect calorimetry measurements are indicated when the use of predictive equations are inaccurate because of the patient's clinical condition, when patients fail to respond to nutrition support based on predictive equations, and when serial adjustments to the nutritional support plan are necessary as caloric requirements change during the stress response phases of critical illness. Use of this methodology and use of IC improves nutritional care and reduces complications associated with over- or underfeeding. 187

The conditions where caloric requirements estimated by predictive equations may be inadequate include: 175,182,187,189,193

- Acute respiratory distress syndrome
- Chronic respiratory disease
- Large or multiple open wounds, burns
- Multiple trauma or neurologic trauma
- Multisystem organ failure
- Systemic inflammatory response syndrome, sepsis
- Postoperative organ transplantation
- Use of sedation and paralytic agents
- Altered body composition:
 - Limb amputation
 - Peripheral edema
 - BMI <18
 - BMI >30
 - Ascites

Use and Interpretation of Indirect Calorimetry Measurements

Resting energy expenditure from indirect calorimetry is recognized as an accurate, objective, patientspecific reference standard for determining energy expenditure. Current recommendations are to decrease the reliance on predictive equations in critically ill patients.

REE measurements should be the targeted goal for nutritional calories in ICU patients without the use of correction factors for activity and metabolic stress. REE measures total caloric needs of the patient but does not distinguish protein from non-protein calories needed. Current practice recommendations are to provide adequate protein calories, as high as 1.5-2.5 g protein/kg/day, balanced with carbohydrates and fats to meet >50-65% of goal calories predicted or measured by REE. 10,193

Several preliminary studies show that preventing a cumulative nutrition deficit and a degree of tight calorie balance reduces mortality and may impact the consequences of overfeeding and underfeeding, such as increased ventilator days, infection rates, and length of stay. 179,192,194-196



Several studies have shown that a cumulative negative energy balance >10,000 kcal determined by IC measurements resulted in worse clinical outcomes. In a small multi-center study comparing nutritional support guided by predictive equations (control group) to measured energy requirements by IC (study group), the proportion of patients with a positive energy balance was higher in the study group and there was a significant difference in ventilator and ICU days when patients had a positive versus a negative energy balance. See Table 12 192,196

Another study using IC reported a lower incidence of organ failure and mortality of 26% when the calorie deficit was < 10,000 kcal versus a mortality of 75% when the calorie deficit was > 10,000 kcal.¹⁹⁷

In a third study, there was a correlation with calorie deficit determined by IC and the development of pressure ulcers in nursing home patients. This correlation was stronger in patients where the negative energy balance exceeded 10,000 kcal. ^{188,198}

This data suggests that nutritional support guided by sequential monitoring and use of IC to maintain a positive energy balance may provide important clinical benefits.

Indirect Calorimetry Calculated Using Other Methods

Modifications to the Weir equation can be used to calculate REE. By substituting a calculated factor for either VO_2 or VCO_2 adjusted for a normal RQ, the REE based on VO_2 (REE- VO_2) or VCO_2 (REE- VO_2) can be calculated where:

REE-O₂ =
$$(3.9 \times VO_2) + (1.1 \times [VO_2 \times .85]) \times 1.44$$
 (5) or

REE-CO₂ =
$$(3.9 \times [VCO_2/.85]) + (1.1 \times VCO_2) \times 1.44$$
 (6)

When the actual RQ is equal to 0.85, both the REE-O $_2$ and REE-CO $_2$ equations will return a REE value equiva-

lent to the value calculated by the standard Weir method. The CCM Express® metabolic analyzer (Medical Graphics Corporation, St. Paul, MN) uses Equation 2 to calculate REE-CO₂ with an accuracy of approximately +/- 10% compared to the REE. ^{189,199}

The REE measured by the CCM Express using the standard Weir equation was retrospectively compared to the calculated REE-CO $_2$ in 67 adult medical and surgical ICU patients. The correlation coefficient r=0.99 and the coefficient of determination $r^2=0.98$, with bias and precision between measurements of -15 \pm 126 kcal/day. When comparing the differences between REE to REE-CO $_2$ to the measured RQ, there was a distinct pattern of agreement, whereby as RQ approached 0.70 the percent error (mean bias / mean REE for the range of RQ) became more positive, and as RQ approached 1.0 the percent error became more negative. More importantly, when RQ was within the normal range of 0.80 to 0.90, the average error was approximately \pm 5%. See Figure 16. 200

The ability to perform IC measurements using just a determination of VCO2 has several important implications. VCO2 determinations are technically easier to perform compared to VO2 and VCO2, and measurement capabilities are increasingly more available on standalone monitors and ventilators. This makes IC estimates of REE more accessible where metabolic analyzers are not available. Additionally, FiO2 does not affect the accuracy of the REE-CO2 calculation. Therefore, settings of FiO₂ > 0.60 may no longer be a limitation of measuring REE within a known range of acceptable error of \pm 5–10%. This means that even the most severe critically ill patients on mechanical ventilation receiving 100% oxygen can have REE estimates performed to manage their complex nutritional needs using the REE-CO₂.

Both the REE-O₂ and REE-CO₂ equations can be further simplified. By solving either equation with any

Table 12. Impact of Energy Balance on Clinical Outcome

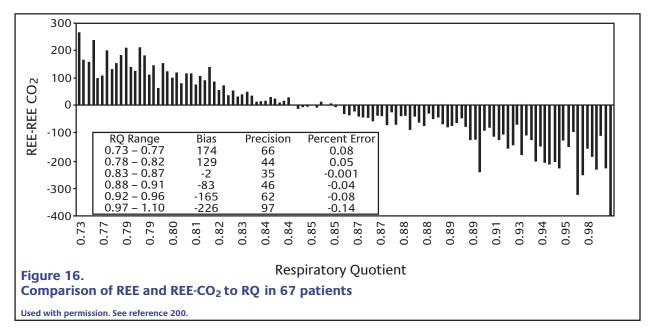
Impact of energy balance on clinical outcome

| impact of chicigy balance on chillea | | | | |
|--------------------------------------|----------|----------|-------------|-------------|
| (*p < 0.005, ‡p < 0.05) | Controls | Study | Vent Days | ICU Days |
| Positive Energy Balance (PEB) | 24 (69%) | 27 (84%) | 10.6 ± 1.1 | 15.9 ± 1.6 |
| (n = 51) | | | | |
| Negative Energy Balance (NEB) | 11 (31%) | 5 (16%) | 19.9 ± 3.9* | 24.6 ± 4.0‡ |
| (n = 16) | | | | |

Note: Negative energy balance was defined by > 10,000 kcal deficit.

Control patients received nutrition support per standard estimations of energy and protein (blinded to MREE and UUN). Study patients received nutrition support per daily MREE and UUN.

Used with permission. See references 192 and 196.



combination of VCO2 and VO2 that equals an RQ of 0.85, and dividing the calculated REE by the measured VCO₂ or VO2, a single factor can be derived for calculating REE-O₂ and REE-CO₂, whereby:

$$REE-CO_2 = 8.19 \times VCO_2 \tag{7}$$

$$REE-O_2 = 6.96 \times VO_2$$
 (8)

For example, when $VCO_2 = 221$ mL/min and $VO_2 =$ 260 mL/min, RQ = 221/260 = 0.85, the Weir equation returns a calculate REE of 1810 kcal/day whereby:

$$REE = (3.9 \times 260) + (1.1 \times 221) \times 1.44 = 1810 \text{ kcal/day}$$

REE-CO₂ can be calculated as follows:

REE-CO₂ = $(3.9 \times (221/0.85)) + (1.1 \times 221) \times 1.44 =$ 1810 kcal/day,

REE-CO₂ Factor = 1810/221 = 8.19, and

 $REE-CO_2 = 8.19 \times 221 = 1810 \text{ kcal/day}.$

Similarly, REE-O₂ becomes:

REE-O₂ = $(3.9 \times 260) + (1.1 \times [260 \times .85]) \times 1.44 =$ 1810 kcal/day,

REE- O_2 Factor = 1810/260 = 6.96, and $REE-O_2 = 6.96 \times 260 = 1810 \text{ kcal/day}.$

The MedGem® (Microlife USA Inc., Clearwater, FL) handheld calorimeter uses measurements of VO2 and

a calculation similar to Equation 8 above. The accuracy of this handheld device shares the same technical limitations and inaccuracies as traditional metabolic testing using IC and can only be performed on spontaneously breathing patients using a mouth piece and nose clips.

The caloric equivalence of oxygen and carbon dioxide can also be used to indirectly calculate REE. (See Table 10.) When the RQ = 0.90, the CO_2 or O_2 caloric equivalent factors equal 5.52 and 4.83 kcal/L respectively, where:

REE-CO₂ Equivalent =
$$5.52 \times VCO_2 \times 1.44$$
 (9)
REE-O₂ Equivalent = $4.83 \times VO_2 \times 1.44$ (10)

See Table 10, Caloric Equivalence. 176-178 This technique has been compared to the Harris Benedict calculation and the Weir equation. The HBE significantly underestimated REE, but there was no significant difference between the Weir and REE-O2 or CO2 equivalent calculations.¹⁷⁶ Due to the technical complexity of measuring VO2, the REE-CO2 equivalent equation is simpler and should be the preferred method, especially when the FiO₂ is >0.60 or when small air leaks that would otherwise invalidate the VO2 calculation are present.177

The Weir equation is a superior method for IC measurements, especially when RQ is at or close to the physiologic extremes of 0.67 and 1.3. However, due to the cost and availability of metabolic analyzers or ventilators with an integrated function, and the technical

Determining Nutritional Requirements

problems associated with measuring VO_2 , REE measurement based on VCO_2 is an attractive alternative. The accuracy of equations 6, 7, and 9 above compared to the Weir equation are within clinically acceptable limits needed for monitoring nutritional interventions. Since VCO_2 monitoring is becoming more readily available, can be performed on any FiO₂, and is less costly than

traditional metabolic testing, its use should be considered for incorporation into a standard nutrition assessment and treatment process. Additional validation studies and outcome measurements are needed to determine the true impact of these alternative methods of indirect calorimetry.

Clinical Practice Recommendations for Nutritional Support

Several clinical practice guidelines from different organizations for the various aspects of nutritional assessment and treatment have been developed. The Society of Critical Care Medicine and the American Society of Parenteral and Enteral Nutrition (SCCM/ASPEN), the European Society for Clinical Nutrition and Metabolism (ESPEN), the Academy of Nutrition and Dietetics (AND), and the Canadian Clinical Practice Guideline for Nutritional Support (CCPG) have developed best practice recommendations based on the interpretation of available evidence, consensus agreement, and expert opinion. The present concentration on evidence-based practice dictates that guidelines be supported by the available literature. The problem with multiple guidelines from different professional societies is that they often contradict one another. See Table 13. 10,201-206

Varying degrees of agreement, disagreement, and controversy over the strength of the evidence can be confusing to the clinician. The review and interpretation of practice recommendations, knowledge of the current available literature, clinical judgment, the specific patient population, and the needs of the individual patient should drive the translation of recommendations into clinical practice.

The following is a summary of some of the "best practice" recommendations from the various organizations:

Nutritional support should be initiated early within the first 24–48 hours in critically ill patients.

Primary goals of nutritional support and care are to:

- Preserve and maintain lean muscle mass
- Provide continuous assessment, reassessment, and modification to optimize outcome
- Monitor the patient for tolerance and complications, such as refeeding syndrome
- Prevent protein energy malnutrition by giving higher protein content while providing adequate total calories
- Monitor nutrition goals and target achievement rate of > 50% within the first week
- Prevent accumulation of a caloric deficit.

Indirect calorimetry should be used when available or when predictive equations are known to be inaccurate

Current EN practice recommendations are to:

- Preferentially feed via the enteral route
- Initiate EN within 24-48 hours
- Reduce interruptions of EN for nursing care and procedures to prevent underfeeding
- HOB elevation to reduce aspiration risk
- Accept GRV up to 500 mL before reducing or stopping EN in the absence of clear signs of intolerance
- Use of motility agents to improve tolerance and reduce GRV
- Promote post-pyloric feeding tube placement when feasible.

Current PN practice recommendations are to:

- Only use PN when enteral route not feasible
- Use PN based on the patient's nutritional risk classification for malnutrition
- Initiate PN early if Nutritional Risk Class III or IV
- Delay PN up to seven days if the patient is in Nutritional Risk Class I or II
- Convert to EN as soon as tolerated to reduce the risks associated with PN.

The following is a summary of some of the "best practice" recommendations from the various organizations:

Use of trophic or "trickle feeding" and permissive underfeeding may be beneficial.

Use of pharmaconutrients and immunonutrition:

- Omega-3 fatty acids (fish oils) may be beneficial in ARDS patients
- Increase omega-3 fatty acids to omega-6 fatty acid ratios
- Use of arginine, glutamine, nucleotides, antioxidants, and probiotics may be beneficial
- Avoid using arginine in patients with severe sepsis.

| Topics | ASPEN/SCCM | AND | Canadian | ESPEN |
|---|---|--|---|--|
| Route of Nutrition | EN | EN | EN | EN |
| Use and Timing of EN | 24-48 hours following admission to ICU | 24-48 hours following admission to ICU | 24-48 hours following admission to ICU | <24 hours |
| Use of Indirect Calorimetry | IC or predictive equations | IC is the standard for determining RMR | Insufficient data | Use predictive equations if IC is not available |
| Dose of Nutrition and Achieving Target | Provide >50-65% of goal calories over the first week of hospitalization; BMI >30 22-25 kcals/kg ideal body weight | At least 60-70% of total estimated energy requirements | No specific recommendation | No specific recommendation |
| Protein Target Per Day | BMI ≤ 30 1.2 - 2.0 g/kg actual body weight; BMI 30-40 ≥ 2.0 g/kg ideal body weight/day; BMI ≥ 40 ≥ 2.5 gm/kg ideal body weight/day | No specific recommendation | Insufficient data, No specific recommendation | EN/PN 1.2 - 2.0 g/kg Ideal or actual body weight depending patient condition |
| EN: Arginine | Recommended for use with surgical ICU patients, but caution with medical ICU patients and those with severe sepsis | Not recommended for routine use | Should not be used | Use in elective upper GI surgical patients; trauma, mild sepsis; avoid with severe sepsis |
| EN: Fish Oil (omega-3 fatty acids) | Recommended for ARDS | No specific recommendation | Should be considered for ARDS | Recommended for ARDS |
| EN: Glutamine | Consider in burn, trauma and mixed ICU patients | No specific recommendation | Consider in burn/ trauma, caution with shock and MOF | Consider in burn and trauma patients |
| EN: High Fat, Low CHO | Not recommended | No specific recommendation | Insufficient data | No specific recommendation |
| EN: Gastric Residual Volume (GRV) | Holding EN for GRVs <500 mL in the absence of other signs of intolerance should be avoided. | Hold EN if GRV >250 ml on two more occasions | GRVs >250 ml consider post pyloric feeding tube | No specific recommendation |
| EN: Motility Agents | Use when clinically feasible | If history of gastroparesis or high GRVs | Recommended with EN intolerance | Recommended with EN intolerance |
| EN: Small Bowel Feeding | Gastric or small bowel feeding is acceptable | Gastric is acceptable; consider small bowel for supine position or heavy sedation | Routine use of small bowel feeding tubes | No significant difference in jejunal versus gastric |

| EN: Body Position | HOB should be elevated | HOB >45 degrees | HOB >45 degrees | No specific recommendation |
|--|--|-----------------------------|---|--|
| EN: Prebiotics/Probiotics/ Synbiotics | No specific recommendation; may consider in transplant, major abdominal surgery, and severe trauma | No specific recommendation | Consider probiotics | No specific recommendation |
| EN: Continuous vs Bolus | For high-risk patients or those shown to be intolerant | No specific recommendation | Insufficient data | No specific recommendation |
| EN with PN | If unable to meet energy needs after 7-10 days via EN | No specific recommendation | Not recommended | Not recommended; consider if unable to be fed sufficiently |
| Parenteral Nutrition | After 7-10 days in nourished patient, as soon as possible in malnourished patient | No specific recommendation | Not recommended | Consider if unable to feed by EN; do not exceed nutrition requirements |
| PN: Lipids | Avoid omega-6 soy-based lipid in the first week | No specific recommendation | Reduce omega-6 load; insufficient data on type | Integral part of PN for energy; varying lipid emulsions available in Europe |
| PN: Glutamine | Consider | No specific recommendation | Strongly recommended; avoid in shock and MOF | Strongly recommended |
| PN: Intensive Insulin Therapy | Protocol should be in place; 110-150 mg/dL | 80-110 mg/dL; <140 mg/dL | 120-160 mg/dL | 80-110 mg/dL |

Summary

Nutritional support is important in the care of patients with acute and chronic illness. The prevention of malnutrition and the maintenance of appropriate nutritional care brings with it the potential for reducing morbidity and mortality, shortening the duration of mechanical ventilation and the length of hospital stay, and lowering health care costs while improving functional quality of life. Appropriate nutritional management is best achieved by using a comprehensively designed nutritional care process supported by the best available evidence. This process should include an interdisciplinary team approach and organizational standards of care with policies and procedures that ensure implementation, continuous assessment, and monitoring of the nutrition care plan.

Varying degrees of agreement, disagreement, and controversy from various organizations regarding nutritional support include the role and timing of enteral versus parenteral route, positioning of feeding tubes, thresholds for GRV, and use of predictive equations and indirect calorimetry. The use of immunonutrition and dietary supplements continue to evolve as practice changes develop when new evidence becomes available. All members of the integrated health care team should maintain awareness of the importance and continued evolution of best practices for nutritional assessment and treatment. Optimizing nutritional support and care of the critically ill and patients with acute and chronic respiratory disorders will contribute to improved outcomes and reduced health care costs.

References

- 1. Coats KG, Morgan SL, Bartolucci AA, Weinsier RL. Hospital-associated malnutrition: a reevaluation 12 years later. J Am Diet Assoc 1993; 93(1):27-33.
- 2. Grover A, Khashu M, Mukherjee A, Kairamkonda V. Iatrogenic malnutrition in neonatal intensive care units: urgent need to modify practice. JPEN J Parenter Enteral Nutr 2008; 32(2):140-144.
- 3. Barker LA, Gout BS, Crowe TC. Hospital malnutrition: prevalence, identification and impact on patients and the healthcare system. Int J Environ Res Public Health 2011; 8(2):514-527.
- 4. Mueller CM, Merritt RJ, McClave S. The A. S. P. E. N. Adult Nutrition Support Core Curriculum, 2nd edition. American Society for Parenteral and Enteral Nutrition; 2012.
- 5. Wall BT, van Loon LJ. Nutritional strategies to attenuate muscle disuse atrophy. Nutr Rev 2013; 71(4):195-208.
- 6. Taylor B, Renfro A, Mehringer L. The role of the dietitian in the intensive care unit. Curr Opin Clin Nutr Metab Care 2005; 8(2):211-216. 7. American Association for Respiratory Care. AARC clinical practice guideline. Metabolic measurement using indirect calorimetry during mechanical ventilation - 2004 revision & update. Respir Care 2004;
- 8. Macht M, Wimbish T, Clark BJ, et al. Postextubation dysphagia is persistent and associated with poor outcomes in survivors of critical illness. Crit Care 2011; 15(5):R231.
- 9. Macht M, King CJ, Wimbish T, et al. Post-extubation dysphagia is associated with longer hospitalization in survivors of critical illness with neurologic impairment. Crit Care 2013; 17(3):R119.
- 10. McClave SA, Martindale RG, Vanek VW, et al. Guidelines for the Provision and Assessment of Nutrition Support Therapy in the Adult Critically Ill Patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.). JPEN J Parenter Enteral Nutr 2009; 33(3):277-316.
- 11. White JV, Guenter P, Jensen G, et al. Consensus statement: Academy of Nutrition and Dietetics and American Society for Parenteral and Enteral Nutrition: characteristics recommended for the identification and documentation of adult malnutrition (undernutrition). JPEN J Parenter Enteral Nutr 2012; 36(3):275-283.
- 12. Hiesmayr M. Nutrition risk assessment in the ICU. Curr Opin Clin Nutr Metab Care 2012; 15(2):174-180.
- 13. Barr J, Hecht M, Flavin KE, et al. Outcomes in critically ill patients before and after the implementation of an evidence-based nutritional management protocol. Chest 2004; 125(4):1446-1457.
- 14. Ochoa JB, Magnuson B, Swintowsky M, et al. Long-term reduction in the cost of nutritional intervention achieved by a nutrition support service. Nutr Clin Pract 2000; 15(4):174-180.
- 15. Cahill NE, Dhaliwal R, Day AG, et al. Nutrition therapy in the critical care setting: what is "best achievable" practice? An international multicenter observational study. Crit Care Med 2010; 38(2):395-401.
- 16. Jensen GL, Hsiao PY, Wheeler D. Adult nutrition assessment tutorial. JPEN J Parenter Enteral Nutr 2012; 36(3):267-274.
- 17. Hiller L, Lowery JC, Davis JA, et al. Nutritional status classification in the Department of Veterans Affairs. J Am Diet Assoc 2001;
- 18. Russell MK, Andrews MR, Brewer CK, et al. Standards for specialized nutrition support: adult hospitalized patients. Nutr Clin Pract 2002; 17(6):384-391.
- 19. International dietetic and nutrition terminology (IDNT) reference manual, 2nd ed. Chicago IL: American Dietetic Association; 2009.

- 20. Maqbool A, Olsen IE, Stallings VA. Clinical assessment of nutritional status. In: Duggan C, Watkins J, Walker WA. Nutrition in pediatrics, 4th ed. Hamilton, Ontario, Canada: BC Decker Inc; 2008.
- 21. World Health Organization. Report of a WHO consultation on obesity. Obesity: preventing and managing the global epidemic. World Health Organization: Geneva; 1998.
- 22. Lee RD, Neiman DC. Nutritional assessment, $\mathbf{4}^{\text{th}}$ ed. Boston: Mc-Graw-Hill; 2007.
- 23. Soler-Cataluna JJ, Sanchez-Sanchez L, Martinez-Garcia MA, et al. Mid-arm muscle area is a better predictor of mortality than body mass index in COPD. Chest 2005; 128(4):2108-2115.
- 24. Janssen I, Katzmarzyk PT, Ross R. Waist circumference and not body mass index explains obesity-related health risk. Am J Clin Nutr 2004; 79(3):379-384.
- 25. Snijder MB, van Dam RM, Visser M, Seidell JC. What aspects of body fat are particularly hazardous and how do we measure them? Int J Epidemiol 2006; 35(1):83-92.
- 26. Ness-Abramof R, Apovian CM. Waist circumference measurement in clinical practice. Nutr Clin Pract 2008; 23(4):397-404.
- 27. Forse RA, Shizgal HM. Serum albumin and nutritional status. JPEN J Parenter Enteral Nutr 1980; 4(5):450-454.
- 28. Jensen GL. Inflammation as the key interface of the medical and nutrition universes: a provocative examination of the future of clinical nutrition and medicine. JPEN J Parenter Enteral Nutr 2006; 30(5):453-463.
- 29. Don BR, Kaysen G. Serum albumin: relationship to inflammation and nutrition. Semin Dial 2004; 17(6):432-437.
- 30. Mahan LK, Escott-Stump S, editors. Krauses's food, nutrition and diet therapy, 11th ed. Philadelphia: Saunders; 2004.
- 31. Law DK, Dudrick SJ, Abdou NI. Immunocompetence of patients with protein-calorie malnutrition. The effects of nutritional repletion. Ann Intern Med 1973; 79(4):545-550.
- 32. Deodhar SD. C-reactive protein: the best laboratory indicator available for monitoring disease activity. Cleve Clin J Med 1989; 56(2):126-130.
- 33. Ettinger WH Jr, Harris T, Verdery RB, et al. Evidence for inflammation as a cause of hypocholesterolemia in older people. J Am Geriatr Soc 1995; 43(3):264-266.
- 34. Linscheid P, Seboek D, Schaer DJ, et al. Expression and secretion of procalcitonin and calcitonin gene-related peptide by adherent monocytes and by macrophage-activated adipocytes. Crit Care Med 2004; 32(8):1715-1721.
- 35. McDermin JM, Jaye A, Schim van der Loeff MF, et al. Elevated iron status strongly predicts mortality in West African adults with HIV infection. J Acquir Immune Defic Syndr 2007; 46(4):498-507.
- 36. Ochs-Balcom HM, Grant BJ, Muti P, et al. Antioxidants, oxidative stress, and pulmonary function in individuals diagnosed with asthma or COPD. Eur J Clin Nutr 2006; 60(8):991-999.
- 37. Jensen GL, Mirtallo J, Compher C, Dhaliwal R, et al. Adult starvation and disease-related malnutrition: a proposal for etiology-based diagnosis in the clinical practice setting from the International Consensus Guideline Committee. JPEN J Parenter Enteral Nutr 2010; 34(2):156-159.
- 38. Krawinkel MB. Interaction of nutrition and infections globally: an overview. Ann Nutr Metab 2012; 61(Suppl 1):39-45.
- 39. Hoffer LJ, Bistrian BR. Why critically ill patients are protein deprived. J Parenter Enteral Nutr 2013; 37(3):300-309.

- 40. Raza N, Benotti PN, Still CD. Nutrition in critically ill obese patients. Crit Care Clin 2010; 26(4):671-678.
- 41. Krawinkel MB. Interaction of nutrition and infections globally: an overview. Ann Nutr Metab 2012; 61(Suppl 1):39-45.
- 42. Kanneganti TD, Dixit VD. Immunological complications of obesity. Nat Immunol 2012; 13(8):707-712.
- 43. Adams KF, Schatzkin A, Harris TB, et al. Overweight, obesity, and mortality in a large prospective cohort of persons 50-71 years old. N Engl J Med 2006; 355(8):763-778.
- 44. Whitlock G, Lewington S, Sherliker P, et al. Body-mass index and cause-specific mortality in 900,000 adults: collaborative analyses of 57 prospective studies. Lancet 2009; 373(9669):1083-1096.
- 45. Stienstra R, Tack CJ, Kanneganti TD, et al. The inflammasome puts obesity in the danger zone. Cell Metab 2012; 15(1):10-18.
- 46. Sin DD, Sutherland ER. Obesity and the lung: 4. Obesity and asthma. Thorax 2008; 63(11):1018-1023.
- 47. Stenholm S, Harris TB, Rantanen T, et al. Sarcopenic obesity: definition, cause and consequences. Curr Opin Clin Nutr Metab Care 2008: 11(6):693-700.
- 48. Kapur VK. Obstructive sleep apnea: diagnosis, epidemiology, and economics. Respir Care 2010; 55(9):1155-1167.
- 49. Mokhlesi B. Obesity hypoventilation syndrome: a state-of-the-art review. Respir Care 2010; 55(10):1347-1362.
- 50. Grundy SM, Cleeman JI, Daniels SR, et al. Diagnosis and management of the metabolic syndrome: an American Heart
- Association/National Heart, Lung, and Blood Institute Scientific Statement. Circulation 2005; 112(17):2735-2752.
- 51. Drager LF, Togeiro SM, Polotsky VY, Lorenzi-Filho G. Obstructive sleep apnea: a cardiometabolic risk in obesity and the metabolic syndrome. J Am Coll Cardiol 2013; 62(7):569-576.
- 52. Lin WY, Yao CA, Wang HC, Huang KC. Impaired lung function is associated with obesity and metabolic syndrome in adults. Obesity (Silver Spring) 2006; 14(9):1654-1661.
- 53. Grundy SM, Cleeman JI, Daniels SR, et al. Diagnosis and management of the metabolic syndrome: an American Heart
- Association/National Heart, Lung, and Blood Institute Scientific Statement. Circulation 2005; 112(17):2735-2752.
- 54. Mannarino MR, Di Filippo F, Pirro M. Obstructive sleep apnea syndrome. Eur J Intern Med 2012; 23(7):586-593.
- 55. Coughlin SR, Mawdsley L, Mugarza JA, et al. Obstructive sleep apnoea is independently associated with an increased prevalence of metabolic syndrome. Eur Heart J 2004; 25(9):735-741.
- 56. Lam JC, Mak JC, Ip MS. Obesity, obstructive sleep apnoea and metabolic syndrome. Respirology 2012; 17(2):223-236.
- 57. Mehanna HM, Moledina J, Travis J. Refeeding syndrome: what it is, and how to prevent it and treat it. BMJ 2008; 336(7659):1495-1498. 58. Kraft MD, Btaiche IF, Sacks GS. Review of the refeeding syndrome. Nutr Clin Pract 2005; 20(6):625-633.
- 59. Boateng AA, Sriram K, Meguid MM, Crook M. Refeeding syndrome: treatment considerations based on collective analysis of literature case reports. Nutrition 2010; 26(2):156-167.
- 60. Stanga Z, Brunner A, Leuenberger M, et al. Nutrition in clinical practice-the refeeding syndrome: illustrative cases and guidelines for prevention and treatment. Eur J Clin Nutr 2008; 62(6):687-694.
- 61. Cove ME, Pinsky MR. Early or late parenteral nutrition: ASPEN vs. ESPEN. Crit Care 2011; 15(6):317.
- 62. DeLegge MH. Parenteral nutrition therapy over the next 5-10 years: where are we heading? JPEN J Parenter Enteral Nutr 2012; 36(2 Suppl):56S-61S.
- 63. Cohen MR. Safe practices for compounding of parenteral nutrition. JPEN J Parenter Enteral Nutr 2012; 36(2 Suppl):14S-19S.
- 64. Curtis C, Sacks GS. Compounding parenteral nutrition: reducing the risks. Nutr Clin Pract 2009; 24(4):441-446.

- 65. Kochevar M, Guenter P, Holcombe B, et al. ASPEN statement on parenteral nutrition standardization. JPEN J Parenter Enteral Nutr 2007; 31(5):441-448.
- 66. Bankhead R, Boullata J, Brantley S, et al. Enteral nutrition practice recommendations. JPEN J Parenter Enteral Nutr 2009; 33(2):122-167. 67. Malone A. Enteral formula selection: a review of selected product categories. Prac Gastro 2005; 29(6):44-74.
- 68. Cohen DA, Byham-Gray L, Denmark RM. Impact of two pulmonary enteral formulations on nutritional indices and outcomes. J Hum Nutr Diet 2013; 26(3):286-293.
- 69. Chen Y, Peterson SJ. Enteral nutrition formulas: which formula is right for your adult patient? Nutr Clin Pract 2009; 24(3):344-355.
- 70. Mentec H, Dupont H, Bocchetti M, et al. Upper digestive intolerance during enteral nutrition in critically ill patients: frequency, risk factors, and complications. Crit Care Med 2001; 29(10):1955-1961.
- 71. Mutlu GM, Mutlu EA, Factor P. GI complications in patients receiving mechanical ventilation. Chest 2001; 119(4):1222-1241.
- 72. Reintam A, Parm P, Kitus R, et al. Gastrointestinal symptoms in intensive care patients. Acta Anaesthesiol Scand 2009; 53(3):318-324. 73. Davies AR, Froomes PR, French CJ, et al. Randomized comparison of nasojejunal and nasogastric feeding in critically ill patients. Crit Care Med 2002; 30(3):586-590.
- 74. Marik PE, Zaloga GP. Gastric versus post-pyloric feeding: a systematic review. Crit Care 2003; 7(3):R46-R51.
- 75. Ho KM, Dobb GJ, Webb SA. A comparison of early gastric and post-pyloric feeding in critically ill patients: a meta-analysis. Intensive Care Med 2006; 32(5):639-649.
- 76. Zhang Z, Xu X, Ding J, Ni H. Comparison of postpyloric tube feeding and gastric tube feeding in intensive care unit patients: a metaanalysis. Nutr Clin Pract 2013; 28(3):371-380.
- 77. Parrish CR, McClave SA. Checking gastric residual volumes: a practice in search of science? Pract Gastroenterol 2008; 32:33-47. 78. McClave SA, Lukan JK, Stefater JA, et al. Poor validity of residual volumes as a marker for risk of aspiration in critically ill patients. Crit Care Med 2005; 33(2):324-330.
- 79. Montejo JC, Miñambres E, Bordejé L, et al. Gastric residual volume during enteral nutrition in ICU patients: the REGANE study. Intensive Care Med 2010; 36(8):1386-1393.
- 80. Metheny NA, Schallom L, Oliver DA, Clouse RE. Gastric residual volume and aspiration in critically ill patients receiving gastric feedings. Am J Crit Care 2008; 17(6):512-519.
- 81. Desachy A, Clavel M, Vuagnat A, et al. Initial efficacy and tolerability of early enteral nutrition with immediate or gradual introduction in intubated patients. Intensive Care Med 2008; 34(6):1054-1059. 82. Powell KS, Marcuard SP, Farrior ES, Gallagher ML. Aspirating gastric residuals causes occlusion of small-bore feeding tubes. JPEN J Parenter Enteral Nutr 1993; 17(3):243-246.
- 83. Villet S, Chiolero RL, Bollmann MD, et al. Negative impact of hypocaloric feeding and energy balance on clinical outcome in ICU patients. Clin Nutr 2005; 24(4):502-509.
- 84. Martindale RG, McClave SA, Vanek VW, et al. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient. Crit Care Med 2009; 37(5):1757-1761.
- 85. Taylor SJ, Fettes SB, Jewkes C, Nelson RJ. Prospective, randomized, controlled trial to determine the effect of early enhanced enteral nutrition on clinical outcome in mechanically ventilated patients suffering head injury. Crit Care Med 1999; 27(11):2525-2531.
- 86. Pinilla JC, Samphire J, Arnold C, et al. Comparison of gastrointestinal tolerance to two enteral feeding protocols in critically ill patients: a prospective, randomized controlled trial. JPEN J Parenter Enteral Nutr 2001; 25(2):81-86.
- 87. McClave SA, Lukan JK, Stefater JA, et al. Poor validity of residual volumes as a marker for risk of aspiration in critically ill patients. Crit Care Med 2005; 33(2):324-330.

- 88. Montejo JC, Miñambres E, Bordejé L, et al. Gastric residual volume during enteral nutrition in ICU patients: the REGANE study. Intensive Care Med 2010; 36(8):1386-1393.
- 89. Poulard F, Dimet J, Martin-Lefevre L, et al. Impact of not measuring residual gastric volume in mechanically ventilated patients receiving early enteral feeding: a prospective before-after study. JPEN J Parenter Enteral Nutr 2010; 34(2):125-130.
- 90. Powell KS, Marcuard SP, Farrior ES, Gallagher ML. Aspirating gastric residuals causes occlusion of small-bore feeding tubes. JPEN J Parenter Enteral Nutr 1993; 17(3):243-246.
- 91. McClure RJ, Newell SJ. Randomized controlled study of clinical outcome following trophic feeding. Arch Dis Child Fetal Neonatal Ed 2000; 82(1):F29-F33.
- 92. Mattei P, Rombeau JL. Review of the pathophysiology and management of postoperative ileus. World J Surg 2006; 30(8):1382-1391. 93. Rice TW, Mogan S, Hays MA, et al. Randomized trial of initial trophic versus full-energy enteral nutrition in mechanically ventilated patients with acute respiratory failure. Crit Care Med 2011;
- 94. Rice TW, Wheeler AP, Thompson BT, et al. Initial trophic vs full enteral feeding in patients with acute lung injury: the EDEN randomized trial. JAMA 2012; 307(8):795-803.
- 95. Hurt RT, Frazier TH, McClave SA, et al. Stress prophylaxis in intensive care unit patients and the role of enteral nutrition. JPEN J Parenter Enteral Nutr 2012; 36(6):721-731.
- 96. Whitney EN, Rolfes SR. Understanding nutrition, 13th ed. Belmont CA: Wadsworth; 2012.
- 97. Shikora SA, Benotti PN. Nutritional support of the mechanically ventilated patient, Respir Care Clin North Am 1997; 3(1):69-90.
- 98. Mechanick JI, Brett EM. Nutrition and the chronically critically ill patient. Curr Opin Clin Nutr Metab Care 2005; 8(1):33-39.
- 99. Egan DF, Kacmarek RM, Stoller JK, Heuer AJ. Egan's fundamentals of respiratory care, 10th ed. St Louis MO: Elsevier/Mosby; 2013.
- 100. Dias CM, Pássaro CP, Cagido VR, et al. Effects of undernutrition on respiratory mechanics and lung parenchyma remodeling. J Appl Physiol 2004; 97(5):1888-1896.
- 101. Baier H, Somani P. Ventilatory drive in normal man during semistarvation. Chest 1984; 85(2):222-225.
- 102. Askanazi J, Weissman C, LaSala PA, et al. Effect of protein intake on ventilatory drive. Anesthesiology 1984; 60(2):106-110.
- 103. Askanazi J, Weissman C, Rosenbaum SH, et al. Nutrition and the respiratory system. Crit Care Med 1982; 10(3):163-172.
- 104. Gorse GJ, Messner RL, Stephens ND. Association of malnutrition with nosocomial infection. Infect Control Hosp Epidemiol 1989; 10(5):194-203.
- 105. Coxson HO, Chan IH, Mayo JR, et al. Early emphysema in patients with anorexia nervosa. Am J Respir Crit Care Med 2004; 170(7):748-752.
- 106. Stein J, Fenigstein H. Pathological anatomy of hunger disease. In: Winick M, editor. Hunger disease: studies by the Jewish physicians in the Warsaw Ghetto. New York: John Wiley & Sons; 1979. p. 207–229.
- 107. Collins PF, Elia M, Stratton RJ. Nutritional support and functional capacity in chronic obstructive pulmonary disease: a systematic review and meta-analysis. Respirology 2013; 18(4):616-629.
- 108. Stratton RJ, Green CJ, Elia M. Disease-related malnutrition: an evidence-based approach to treatment. Wallingford, UK: CABI Publishing: 2003.
- 109. Ezzell L, Jensen GL. Malnutrition in chronic obstructive pulmonary disease. Am J Clin Nutr 2000; 72(6):1415-1416.
- 110. Laaban JP, Kouchakji B, Dore MF, et al. Nutritional status of patients with chronic obstructive pulmonary disease and acute respiratory failure. Chest 1993; 103(5):1362-1368.

- 111. Kelly SM, Rosa A, Field S, et al. Inspiratory muscle strength and body composition in patients receiving total parenteral nutrition therapy. Am Rev Respir Dis 1984; 130(1):33-37.
- 112. Askanazi J, Weissman C, Rosenbaum SH, et al. Nutrition and the respiratory system. Crit Care Med 1982; 10(3):163-172.
- 113. Driver AG, McAlevy MT, Smith JL. Nutritional assessment of patients with chronic obstructive pulmonary disease and acute respiratory failure. Chest 1982; 82(5):568-571.
- 114. Collins PF, Stratton RJ, Elia M. Nutritional support in chronic obstructive pulmonary disease: a systematic review and meta-analysis. Am J Clin Nutr 2012; 95(6):1385-1395.
- 115. Ferreira IM, Brooks D, White J, Goldstein R. Nutritional supplementation for stable chronic obstructive pulmonary disease. Cochrane Database Syst Rev 2012; 12:CD000998.
- 116. Talpers SS, Romberger DJ, Bunce SB, Pingleton SK. Nutritionally associated increased carbon dioxide production. Excess total calories vs high proportion of carbohydrate calories. Chest 1992; 102(2):551-
- 117. Cai B, Zhu Y, Ma Yi, et al. Effect of supplementing a high-fat, lowcarbohydrate enteral formula in COPD patients. Nutrition 2003; 19(3):229-232.
- 118. Vermeeren MA, Wouters EF, Nelissen LH, et al. Acute effects of different nutritional supplements on symptoms and functional capacity in patients with chronic obstructive pulmonary disease. Am J Clin Nutr 2001; 73(2):295-301.
- 119. Itoh M, Tsuji T, Nemoto K, et al. Undernutrition in patients with COPD and its treatment. Nutrients 2013; 5(4):1316-1335.
- 120. DeBellis HF, Fetterman JW Jr. Enteral nutrition in the chronic obstructive pulmonary disease (COPD) patient. J Pharm Pract 2012;
- 121. Malone AM. Specialized enteral formulas in acute and chronic pulmonary disease. Nutr Clin Pract 2009; 24(6):666-674.
- 122. Broekhuizen R, Creutzberg EC, Weling-Scheepers CA, et al. Optimizing oral nutritional drink supplementation in patients with chronic obstructive pulmonary disease. Br J Nutr 2005; 93(6):965-971. 123. Planas M, Alvarez J, García-Peris PA, et al. Nutritional support and quality of life in stable chronic obstructive pulmonary disease (COPD) patients. Clin Nutr 2005; 24(3):433-441.
- 124. Gadek JE, DeMichele SJ, Karlstad MD, et al. Effect of enteral feeding with eicosapentaenoic acid, gamma-linolenic acid, and antioxidants in patients with acute respiratory distress syndrome. Crit Care Med 1999; 27(8):1409-1420.
- 125. Singer P, Theilla M, Fisher H, et al. Benefit of an enteral diet enriched with eicosapentaenoic acid and gamma-linolenic acid in ventilated patients with acute lung injury. Crit Care Med 2006; 34(4):1033-1038.
- 126. Pontes-Arruda A, Aragao AM, Albuquerque JD. Effects of enteral feeding with eicosapentaenoic acid, gamma-linolenic acid, and antioxidants in mechanically ventilated patients with severe sepsis and septic shock. Crit Care Med 2006; 34(9):2325-2333.
- 127. McClave SA, Martindale RG, Vanek VW, et al. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient. JPEN J Parenter Enteral Nutr 2009; 33(3):277-316. 128. Patterson E, Wall R, Fitzgerald GF, et al. Health implications of high dietary omega-6 polyunsaturated fatty acids. J Nutr Metab 2012; 2012:539426
- 129. de Lorgeril M, Salen P. New insights into the health effects of dietary saturated and omega-6 and omega-3 polyunsaturated fatty acids. BMC Med 2012; 10:50.
- 130. Simopoulos AP. The importance of the ratio of omega-6/omega-3 essential fatty acids. Biomed Pharmacother 2002; 56(8):365-379.
- 131. Simopoulos AP. The importance of the omega-6/omega-3 fatty acid ratio in cardiovascular disease and other chronic diseases. Exp Biol Med (Maywood) 2008; 233(6):674-688.

- 132. Bulger EM, Maier RV. Lipid mediators in the pathophysiology of critical illness. Crit Care Med 2000; 28(4 Suppl):N27-N36.
- 133. Rice TW, Wheeler AP, Thompson BT, et al. Enteral omega-3 fatty acid, gamma-linolenic acid, and antioxidant supplementation in acute lung injury. JAMA 2011; 306(14):1574-1581.
- 134. Cook DJ, Heyland DK. Pharmaconutrition in acute lung injury. JAMA 2011; 306(14):1599-1600.
- 135. Long CL, Schaffel N, Geiger JW, et al. Metabolic response to injury and illness: estimation of energy and protein needs from indirect calorimetry and nitrogen balance. J Parenter Enteral Nutr 1979; 3(6):452-456.
- 136. Zaloga GP, Roberts P. Permissive underfeeding. New Horiz 1994; 2(2):257-263.
- 137. Malone AM. Methods of assessing energy expenditure in the intensive care unit. Nutr Clin Pract 2002; 17(1):21-28.
- 138. Dvir D, Cohen J, Singer P. Computerized energy balance and complications in critically ill patients: an observational study. Clin Nutr 2006; 25(1):37-44.
- 139. McClave SA, McClain CJ, Snider HL. Should indirect calorimetry be used as part of nutritional assessment? J Clin Gastroenterol 2001; 33(1):14-19.
- 140. McClave SA. The consequences of overfeeding and underfeeding. J Resp Care Pract 1997; 10:57-58,62-64.
- 141. Doekel RC Jr, Zwillich CW, Scoggin CH, et al. Clinical semi-starvation: depression of hypoxic ventilatory response. N Engl J Med 1976; 295(7):358-361.
- 142. Cuthbertson DP. Second annual Jonathan E. Rhoads Lecture. The metabolic response to injury and its nutritional implications: retrospect and prospect. JPEN J Parenter Enteral Nutr 1979; 3(3):108-129.
- 143. McMahon MM, Nystrom E, Braunschweig C, et al. A.S.P.E.N. clinical guidelines: nutrition support of adult patients with hyperglycemia. JPEN J Parenter Enteral Nutr 2013; 37(1):23-36.
- 144. Jacobi J, Bircher N, Krinsley J, et al. Guidelines for the use of an insulin infusion for the management of hyperglycemia in critically ill patients. Crit Care Med 2012; 40(12):3251-3276.
- 145. Mechanick JI, Scurlock C. Glycemic control and nutritional strategies in the cardiothoracic surgical intensive care unit – 2010: state of the art. Semin Thorac Cardiovasc Surg 2010; 22(3):230-235. 146. Reeds D. Near-normal glycemia for critically ill patients receiving nutrition support: fact or folly. Curr Opin Gastroenterol 2010; 26(2):152-155.
- 147. Dickerson RN, Boschert KJ, Kudsk KA, Brown RO. Hypocaloric enteral tube feeding in critically ill obese patients. Nutrition 2002; 18(3):241-246.
- 148. Elamin EM. Nutritional care of the obese intensive care unit patient. Curr Opin Crit Care 2005; 11(4):300-303.
- 149. Choban PS, Dickerson RN. Morbid obesity and nutrition support: is bigger different? Nutr Clin Pract 2005; 20(4):480-487.
- 150. Malone AM. Permissive underfeeding: its appropriateness in patients with obesity, patients on parenteral nutrition, and non-obese patients receiving enteral nutrition. Curr Gastroenterol Rep 2007; 9(4):317-322.
- 151. Battistella FD, Widergren JT, Anderson JT, et al. A prospective, randomized trial of intravenous fat emulsion administration in trauma victims requiring total parenteral nutrition. J Trauma 1997; 43(1):52-58
- 152. Ahrens CL, Barletta JF, Kanji S, et al. Effect of low-calorie parenteral nutrition on the incidence and severity of hyperglycemia in surgical patients: a randomized, controlled trial. Crit Care Med 2005; 33(11):2507-2512.
- 153. Choban PS, Burge JC, Scales D, Flancbaum L. Hypoenergetic nutrition support in hospitalized obese patients: a simplified method for clinical application. Am J Clin Nutr 1997; 66(3):546-550.

- 154. McCowen KC, Friel C, Sternberg J, et al. Hypocaloric total parenteral nutrition: effectiveness in prevention of hyperglycemia and infectious complications - a randomized clinical trial. Crit Care Med 2000; 28(11):3606-3611.
- 155. Mehta NM, Bechard LJ, Cahill N, et al. Nutritional practices and their relationship to clinical outcomes in critically ill children - an international multicenter cohort study*. Crit Care Med 2012; 40(7):2204-2211.
- 156. American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) Board of Directors. Clinical guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients. JPEN J Parenter Enteral Nutr 2009; 33(3):255-259.
- 157. Mehta NM, Compher C. A.S.P.E.N. Clinical Guidelines: nutrition support of the critically ill child. JPEN J Parenter Enteral Nutr 2009; 33(3):260-276.
- 158. Marik PE, Zaloga GP. Immunonutrition in high-risk surgical patients: a systematic review and analysis of the literature. JPEN J Parenter Enteral Nutr 2010; 34(4):378-386.
- 159. Mizock BA. Immunonutrition and critical illness: an update. Nutrition 2010; 26(7-8):701-707.
- 160. Mlcak RP, Jeschke MG, Barrow RE, Herndon DN. The influence of age and gender on resting energy expenditure in severely burned children. Ann Surg 2006; 244(1):121-130.
- 161. Trumbo P, Schlicker S, Yates AA, Poos M. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. J Am Diet Assoc 2002; 102(11):1621-1630.
- 162. Academy of Nutrition and Dietetics. Nutrition care manual, 2012. Available at: www.nutritioncaremanual.org/welcome.cfm Accessed Sept. 30, 2013
- 163. Frankenfield D, Hise M, Malone A, et al. Prediction of resting metabolic rate in critically ill adult patients: results of a systematic review of the evidence. J Am Diet Assoc 2007; 107(9):1552-1561.
- 164. Frankenfield D, Roth-Yousey L, Compher C. Comparison of predictive equations for resting metabolic rate in healthy nonobese and obese adults: a systematic review. J Am Diet Assoc 2005; 105(5):775-
- 165. Anderegg BA, Worrall C, Barbour E, et al. Comparison of resting energy expenditure prediction methods with measured resting energy expenditure in obese, hospitalized adults. JPEN J Parenter Enteral Nutr 2009; 33(2):168-175.
- 166. Wooley JA. Indirect calorimetry: applications in practice. Respir Care Clin N Am 2006; 12(4):619-633.
- 167. Cerra FB, Benitez MR, Blackburn GL, et al. Applied nutrition in ICU patients: a consensus statement of the American College of Chest Physicians. Chest 1997; 111(3):769-778.
- 168. U.S. Department of Agriculture. Atwater WO, Rosa EB. Description of neo respiration calorimeter and experiments on the conservation of energy in the human body. Off Exp Sta Bull 63, 1899.
- 169. Atwater WO, Benedict FG. A respiration calorimeter with appliances for the direct determination of oxygen. Washington DC: Carnegie Institute of Washington; 1905: Publication 42.
- 170. Harris JA, Benedict FG. A biometric study of human basal metabolism. Proc Natl Acad Sci USA. 1918; 4(12):370-373.
- 171. Benedict FG, Carpenter TM. Metabolism and energy transformations of healthy man during rest. Carnegie Institute of Washington,
- 172. Benedict FG, Tompkins EH. Respiratory exchange, with a description of a respiration apparatus for clinical use. Boston Med Surg J 1916; 174:857-864.
- 173. Branson RD, Johannigman JA. The measurement of energy expenditure. Nutr Clin Pract 2004; 19(6):622-636.
- 174. McClave SA, Lowen CC, Kleber MJ, et al. Clinical use of the respiratory quotient obtained from indirect calorimetry. JPEN J Parenter Enteral Nutr 2003; 27(1):21-26.

- 175. McClave SA, Snider HL. Use of indirect calorimetry in clinical nutrition. Nutr Clin Pract 1992; 7(5):207-221.
- 176. Hess D, Daugherty A, Large E, Agarwal NN. A comparison of four methods of determining caloric requirements of mechanically ventilated trauma patients. Respir Care 1986; 31(12):1197-1203.
- 177. McCamish MA, Dean RE, Ouellette TR. Assessing energy requirements of patients on respirators. JPEN J Parenter Enteral Nutr 1981; 5(6):513-516.
- 178. Wilmore DW. The metabolic management of the critically ill. New York: Plenum Medical Book Company; 1977.
- 179. Lev S, Cohen J, Singer P. Indirect calorimetry measurements in the ventilated critically ill patient: facts and controversies - the heat is on. Crit Care Clin 2010; 26(4):e1-e9.
- 180. Critical Decisions website. Branson RD, Rodriguez J, Tajchman S, Wooley JA. Indirect calorimetry in the ventilated patient. Available at: www.critical-decisions.org/CD_1.pdf Accessed Sept. 30, 2013
- 181. Ultman JS, Bursztein S. Analysis of error in the determination of respiratory gas exchange at varying FIO2. J Appl Physiol Respir Environ Exerc Physiol 1981; 50(1):210-216.
- 182. Brandi LS, Bertolini R, Pieri M, et al. Comparison between cardiac output measured by thermodilution technique and calculated by O2 and modified CO2 Fick methods using a new metabolic monitor. Intensive Care Med 1997; 23(8):908-915.
- 183. Bellani G, Foti G, Spagnolli E, et al. Increase of oxygen consumption during a progressive decrease of ventilatory support is lower in patients failing the trial in comparison with those who succeed. Anesthesiology 2010; 113(2):378-385.
- 184. Glass C, Hipskind P, Cole D, et al. Handheld calorimeter is a valid instrument to quantify resting energy expenditure in hospitalized cirrhotic patients: a prospective study. Nutr Clin Pract 2012;
- 185. Hipskind P, Glass C, Charlton D, et al. Do handheld calorimeters have a role in assessment of nutrition needs in hospitalized patients? A systematic review of literature. Nutr Clin Pract 2011; 26(4):426-433.
- 186. Spears KE, Kim H, Behall KM, Conway JM. Hand-held indirect calorimeter offers advantages compared with prediction equations, in a group of overweight women, to determine resting energy expenditures and estimated total energy expenditures during research screening. J Am Diet Assoc 2009; 109(5):836-845.
- 187. Wooley JA, Sax HC. Indirect calorimetry: applications to practice. Nutr Clin Pract 2003; 18(5):434-439.
- 188. Branson RD. The measurement of energy expenditure: instrumentation, practical considerations and clinical application. Resp Care 1990; 35(7):640-659.
- 189. Porter C, Cohen NH. Indirect calorimetry in critically ill patients: role of the clinical dietitian in interpreting results. J Am Diet Assoc 1996; 96(1):49-57.
- 190. Matarese LE. Indirect calorimetry: technical aspects. J Am Diet Assoc 1997; (10 Suppl 2):S154-S160.
- 191. Swinamer DL, Phang PT, Jones RL, et al. Twenty-four hour energy expenditure in critically ill patients. Crit Care Med 1987; 15(7):637-
- 192. Blumberg A, Keller G. Oxygen consumption during maintenance hemodialysis. Nephron 1979; 23(6):276-281.
- 193. Alberda C, Gramlich L, Jones N, et al. The relationship between nutritional intake and clinical outcomes in critically ill patients: results of an international multicenter observational study. Intensive Care Med 2009; 35(10):1728-1737.

- 194. Strack van Schijndel RJ, Weijs PJ, Koopmans RH, et al. Optimal nutrition during the period of mechanical ventilation decreases mortality in critically ill, long-term acute female patients: a prospective observational cohort study. Crit Care 2009; 13(4):R132.
- 195. Anbar R, Theilla M, Fisher H, et al. Decrease in hospital mortality in tight calorie balance control study: the preliminary results of the TICACOS study. Clin Nutr Suppl 2008; 27:S11.
- 196. Mault J. Energy balance and outcome in critically ill patients: results of a multicenter, prospective randomized trial by the ICU Nutrition Study Group. JPEN J Parenter Enteral 2000; 24(1):S4.
- 197. Bartlett RH, Dechert RE, Mault JR, et al. Measurement of metabolism in multiple organ failure. Surgery 1982; 92(4):771-779.
- 198. Kleber MJ, Lowen CC, McClave SA, et al. Is there a role for indirect calorimetry in maximizing patient outcome from nutritional alimentation in the long-term nursing care setting? Nutr Clin Pract 2000; 15(5):227-233.
- 199. Medgraphics website. CCM Express operators manual. Available at: http://mgcdiagnostics.com/images/uploads/CCMexpress_sellsheet_060065rF_web.pdf

Accessed Oct. 8, 2013

- 200. Siobal MS, Hammoudeh H, Snow M. Accuracy of resting energy expenditure calculated by a modification of the abbreviated Weir equation in mechanically ventilated adult ICU patients (Abstract). Respir Care 2012; 57(10):1721.
- 201. Heyland DK, Dhaliwal R, Drover JW, et al. Canadian clinical practice guidelines for nutrition support in mechanically ventilated, critically ill adult patients. JPEN J Parenter Enteral Nutr 2003; 27(5):355-373.
- 202. Academy of Nutrition and Dietetics website. Critical illness evidence analysis project: American Dietetic Association. Available at: http://andevidencelibrary.com/topic.cfm?cat=1031&auth=1 Accessed Sept. 30, 2013
- 203. Critical Care Nutrition website. Canadian clinical practice guidelines updated in 2013. Available at:
- www.criticalcarenutrition.com/index.php?option=com_content&vie w=category&layout=blog&id=21&Itemid=10 Accessed Sept. 30, 2013 204. Academy of Nutrition and Dietetics website. Critical illness evidence analysis project: American Dietetic Association. Available at: http://andevidencelibrary.com/topic.cfm?cat=1031&auth=1 Accessed Sept. 30, 2013
- 205. Kreymann KG, Berger MM, Deutz NE, et al. ESPEN guidelines on enteral nutrition: intensive care. Clin Nutr 2006; 25(2):210-223. 206. Singer P, Berger MM, Van den Berghe G, et al. ESPEN guidelines on parenteral nutrition: intensive care. Clin Nutr 2009; 28(4):387-400.

Acronyms

| AARC | American Association for | GI | gastrointestinal |
|-----------------|----------------------------------|------------------|-----------------------------------|
| | Respiratory Care | GRV | gastric residual volumes |
| AMA | arm muscle area | HBE | Harris Benedict Equation |
| AND | Academy of Nutrition | HHS | Health and Human Services |
| | and Dietetics | НОВ | head of bed |
| ARDS | acute respiratory distress | IBD | inflammatory bowel disease |
| | syndrome | IC | indirect calorimetry |
| ASPEN | American Society of Parenteral | kcal | kilocalories |
| | and Enteral Nutrition | MAC | mid-upper arm circumference |
| BIA | bioelectrical impedance analysis | MRI | magnetic resonance imaging |
| BMI | body mass index | PEEP | positive end-expiratory pressure |
| BMR | basal metabolic rate | PEG | percutaneous endoscopic |
| CCPG | Canadian Clinical Practice | | gastrostomy |
| | Guidelines for Nutritional | PEM | protein-energy malnutrition |
| | Support | PICC | peripherally inserted central |
| CF | cystic fibrosis | | catheter |
| CNSC | Certified Nutrition Support | PN | parenteral nutrition |
| | Clinician | RDA | recommended dietary allowance |
| CO ₂ | carbon dioxide | REE | resting energy expenditure |
| COPD | chronic obstructive pulmonary | RMR | resting metabolic rate |
| | disease | RQ | respiratory quotient |
| CRP | C-reactive protein | SBS | short bowel syndrome |
| CT | computed tomography | SCCM | Society of Critical Care Medicine |
| DRI | dietary reference intakes | SIRS | systemic inflammatory response |
| DXA | dual-energy X-ray absorptiometry | | syndrome |
| EN | enteral nutrition | TSF | triceps skinfold |
| ESPEN | European Society for Clinical | UUN | urinary urea nitrogen |
| | Nutrition and Metabolism | VCO ₂ | carbon dioxide production |
| FFQ | food frequency questionnaires | VO ₂ | oxygen consumption |
| | | | |

| 1 | | |
|---|--|--|



Copyright © 2013 The American Association for Respiratory Care 9425 N. MacArthur Blvd., Ste. 100, Irving, TX 75063 (972) 243-2272 • Fax (972) 484-2720 • www.aarc.org • info@aarc.org