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A.S.P.E.N. Clinical Guidelines: Nutrition Support of Hospitalized Pediatric Patients With Obesity

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Pediatric obesity has reached epidemic proportions in the United States,¹ and there are reports of greater discharge diagnosis of obesity-related complications such as diabetes, sleep apnea, and gallbladder disease and longer length of stay.² The origin of pediatric obesity is multifactorial and leads to numerous complications^{3,4} affecting inflammatory processes⁵ as well as nutrient metabolism.⁶⁻⁹ As a result, current estimations of nutrition status¹⁰⁻¹² and requirements among obese patients remain unclear.¹³⁻¹⁵ Recognizing that body mass index (BMI) may predict obesity-related complications even in adulthood, the Institute of Medicine (IOM)¹⁶ and, more recently, the American Academy of Pediatrics (AAP)⁴ recommend that the term *obesity* be used in children aged 2–20 years (BMI \geq 95th percentile). Once obesity has been identified, the role of nutrition support is to prevent complications associated with the provision of enteral or parenteral feedings. Undernutrition may result in energy and protein deprivation,^{17,18} whereas overzealous nutrition support may result in hypophosphatemia, typically observed in refeeding syndrome, and hyperglycemia; all of these complications may affect morbidity and mortality risk.¹⁹ Thus, neither undernutrition nor

overnutrition can be recommended during hospitalization of the obese child.

Methods

The American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) consists of healthcare professionals representing the disciplines of medicine, nursing, pharmacy, dietetics, and nutrition science. The mission of A.S.P.E.N. is to improve patient care by advancing the science and practice of nutrition support therapy. A.S.P.E.N. vigorously works to support quality patient care, education, and research in the fields of nutrition and metabolic support in all healthcare settings. These clinical guidelines were developed under the guidance of the A.S.P.E.N. Board of Directors. Promotion of safe and effective patient care by nutrition support practitioners is a critical role of the A.S.P.E.N. organization. The A.S.P.E.N. Board of Directors has published clinical guidelines since 1986.²⁰⁻²² Starting in 2007, A.S.P.E.N. has revised these clinical guidelines on an ongoing basis by reviewing about 20% of the chapters each year in order to keep them as current as possible.

These A.S.P.E.N. clinical guidelines are general. They are based upon general conclusions of health professionals who, in developing such guidelines, have balanced potential benefits to be derived from a particular mode of medical therapy against certain risks inherent with such therapy. However, the professional judgment of the attending health professional is the primary component of quality medical care. Because guidelines cannot account for every variation in circumstances, the practitioner must always

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exercise professional judgment in their application. These clinical guidelines are intended to supplement, but not replace, professional training and judgment.

These clinical guidelines were created in accordance with the IOM recommendations as “systematically developed statements to assist practitioner and patient decisions about appropriate healthcare for specific clinical circumstances.”²³ These clinical guidelines are for use by healthcare professionals who provide nutrition support services and offer clinical advice for managing adult and pediatric patients in inpatient and outpatient (ambulatory, home, and specialized care) settings. The utility of the clinical guidelines is attested to by the frequent citation of this document in peer-reviewed publications and its frequent use by A.S.P.E.N. members and other healthcare professionals in clinical practice, academia, research, and industry. The guidelines inform professional clinical activities, serve as educational tools, and influence institutional practices and resource allocation.²⁴

These clinical guidelines are formatted to promote the ability of the end user of the document to understand the strength of the literature used to grade each recommendation. Each guideline recommendation is presented as a clinically applicable definitive statement of care and should help the reader make the best patient care decision. The best available literature was obtained and carefully reviewed. Chapter authors completed a thorough literature review using Medline, the Cochrane Central Register of Controlled Trials, the Cochrane Database of Systematic Reviews, and other appropriate reference sources. The results of the literature search and review formed the basis of an evidence-based approach to the clinical guidelines. Chapter editors work with the authors to ensure compliance with the authors' directives regarding content and format. The initial draft is reviewed internally to ensure consistency with the other A.S.P.E.N. Guidelines and Standards and reviewed externally (either by experts in the field within our organization or outside of our organization) for appropriateness of content. Finally, the draft is reviewed and approved by the A.S.P.E.N. Board of Directors.

The system used to categorize the level of evidence for each study or article used in the rationale of the guideline statement and to grade the guideline recommendation is outlined in Table 1.²⁵

The grade of a guideline is based on the levels of evidence of the studies used to support the guideline. A randomized controlled trial (RCT), especially one that is double-blind in design, is considered to be the strongest level of evidence to support decisions regarding a therapeutic intervention in clinical medicine.²⁶ A systematic review (SR) is a specialized type of literature review that analyzes the results of several RCTs. A high-quality SR usually begins with a clinical question and a protocol that

Table 1. Grading of Guidelines and Levels of Evidence

Grading of Guidelines	
A	Supported by at least two level I investigations
B	Supported by one level I investigation
C	Supported by at least one level II investigation
D	Supported by at least one level III investigation
E	Supported by level IV or V evidence
Levels of Evidence	
I	Large randomized trials with clear-cut results; low risk of false-positive (alpha) and/or false-negative (beta) error
II	Small, randomized trials with uncertain results; moderate to high risk of false-positive (alpha) and/or false-negative (beta) error
III	Nonrandomized cohort with contemporaneous controls
IV	Nonrandomized cohort with historical controls
V	Case series, uncontrolled studies, and expert opinion

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Table 2. Nutrition Support Guideline Recommendations of Hospitalized Pediatric Patients With Obesity

Guideline Recommendation	Grade
1. Body mass index is the preferred practical method to screen children for obesity.	D
2. Pediatric obese inpatients may be at increased nutrition risk. We recommend testing for potential laboratory abnormalities for safety reasons (eg, fasting blood sample, including lipid profile, glucose, phosphorus, and complete blood count).	E
3. When possible, energy requirements of obese hospitalized children should be assessed using indirect calorimetry rather than predictive equations.	D
4. There is no adequate evidence to assess the clinical outcomes of hypocaloric or hypercaloric feeding during hospitalization of obese children. Therefore, the goals for the provision of energy to the pediatric obese inpatient should be similar to their nonobese counterparts.	E

addresses the methods to answer this question. These methods usually state how the literature is identified and assessed for quality, what data are extracted, how they are analyzed, and whether there were any deviations from the protocol during the course of the study. In most instances, meta-analysis (MA), a mathematical tool to combine data from several sources, is used to analyze the data. However, not all SRs use MA. SR is considered among the most important level of evidence in the field of evidence-based

medicine. A level of I, the highest level, will be given to large RCTs where results are clear and the risk of alpha and beta error is low (well-powered). A level of II will be given to RCTs that include a relatively low number of patients or are at moderate to high risk for alpha and beta error (under-powered). Meta-analyses can be used to combine the results of studies to further clarify the overall outcome of these studies but will not be considered in the grading of the guideline. A level of III is given to cohort studies with contemporaneous controls and to validation studies, whereas cohort studies with historic controls will receive a level of IV. Case series, uncontrolled studies, and articles based on expert opinion alone will receive a level of V.

Practice Guidelines and Rationales

Table 2 provides the entire set of guideline recommendations for nutrition support of hospitalized pediatric patients with obesity.

Practice Guidelines

1. BMI is the preferred practical method to screen children for obesity. (Grade: D)

Rationale. Although BMI (kg/m²) does not directly measure body fat, it has been recognized as a useful predictor of adiposity and medical complications of obesity. BMI is a measure of relative weight rather than adiposity.²⁷ Tracking studies from childhood to adulthood provide the best available evidence to support the validity of BMI as a screening criterion for obesity in children and adolescents.²⁸ There is increasing evidence that ≥ 95 th percentile on BMI for sex and age charts in childhood predicts adult BMI, obesity, adiposity, and mortality²⁹⁻³⁷ (Table 3); however, more tracking (longitudinal) data are needed, especially on clinical risks associated with obesity.^{10,28} Although BMI is an adequate screening method for older children and at a group level, its strength as an indicator of adiposity decreases at younger ages (<13 years) and may vary by ethnicity and race.^{10,38} There is no current valid measure for children younger than 2 years^{10,39-40} or for severe obesity at any age.^{10,38,41-43}

2. Pediatric obese inpatients may be at increased nutrition risk. Testing for potential laboratory abnormalities is recommended for safety reasons (eg, fasting blood sample, including lipid profile, glucose, phosphorus, and complete blood count). (Grade: E)

Rationale. Although the prevalence of pediatric obesity (based on BMI ≥ 95 th percentile) is elevated, studies of obesity prevalence and nutrition support outcomes among

obese compared with nonobese children in the hospital setting have not been evaluated. Nevertheless, we believe that hospitalized pediatric patients should undergo nutrition screening to identify those who require formal nutrition assessment with development of a nutrition care plan. Obese children are at increased risk for anemia,⁴⁴⁻⁴⁵ low fat-soluble vitamins levels (such as vitamin D),⁸ low vitamin B status,⁹ hyperlipidemia, insulin resistance, and hyperglycemia.^{6,7,10,46,47} The presence of the metabolic syndrome in children is not well defined and may not predict obesity in adulthood.⁴⁸ There is some evidence from adult studies that tight control of hyperglycemia may affect morbidity and mortality, and there are anecdotal reports of hypophosphatemia following glucose provision in long-term fasting.⁴⁹

3. When possible, energy requirements of obese hospitalized children should be assessed using indirect calorimetry rather than predictive equations. (Grade: D)

Rationale. Resting energy expenditure (REE) varies with obesity status but is best explained by differences in lean body mass. The percentage of lean body mass for each additional kilogram of weight above ideal weight is highly variable. Therefore, the calculation of excess weight to estimate ideal body weight is imprecise. As there is no practical and valid tool to evaluate lean body mass in order to estimate ideal weight in hospitalized patients, assessment of REE using indirect calorimetry is an alternative to the imprecision of equations (Table 4).^{13-15,50-54}

4. There is not adequate evidence to assess the clinical outcomes of hypocaloric or hypercaloric feeding during hospitalization of obese children. Therefore, the goals for the provision of energy to pediatric obese inpatients should be similar to the goals for their nonobese counterparts until more evidence is available. (Grade: E)

Rationale. Although hypocaloric solutions are used in the outpatient setting, there is no evidence that these solutions should be initiated during hospitalizations. There are anecdotal reports of use of hypocaloric solutions in patients who are hospitalized for major obesity-related complications such as heart failure, pseudotumor cerebri, and sleep apnea. Finally, note that the use of old guidelines may result in overfeeding (recommended dietary allowances may overestimate needs by up to 20%, depending on the age group) and further complications.⁵⁵

Table 3. Adult Outcomes of Childhood Obesity

Study	Population	Intervention	Outcome
Tracking studies of BMI and other adiposity measures from childhood to adulthood			
Freedman ²⁹ 2005 Level III	n = 2,392 Age 5–14 y in 1973–1974, 17 y follow-up Louisiana (USA) n = 2,610	Tracking childhood overweight to adult obesity by race Longitudinal models for repeated measure data Child vs adult BMI, adiposity, adiposity by TSF Spearman correlation, simple linear regression	Tracking differs by race, with 65% (white girls) to 84% (black girls) of “overweight” children becoming obese adults Childhood BMI strongly predicts adult adiposity Childhood overweight strongly associated with adult overweight
Freedman ³⁰ 2005 Level III	Age 2–17 y in 1973–1974 and 1992–1994, age 18–37 y in 1982–1996 Louisiana (USA)		
Guo ³¹ 2002 Level III	In 1929 (n = 347) Annual measures ages 3–18 y, age 20 y, age 30–39 y (mean 35 y) White race only Ohio, Indiana, Kentucky (USA)	Predict adult overweight/obesity from childhood/adolescent BMI cutoffs ROC, logistic regression	Half of children and adolescents with BMI ≥ 75 th %tile overweight as adults With BMI ≥ 95 th %tile, children 62%–98% more likely to be overweight at age 35 y
Guo ³² 2000 Level III	In 1929 (n = 338), age 2–25 y; age 35–45 y (n = 159) Hydrostatic weight data (n = 85) White race only Ohio, Indiana, Kentucky (USA)	BMI patterns during childhood, puberty, postpuberty vs overweight and body fatness at age 35–45 y Pediatric BMI gain/y at BMI rebound, puberty, postpuberty General linear models	Change in childhood BMI related to adult overweight and adiposity, especially in females Early BMI rebound associated with maximum BMI velocity and BMI as adult Maximum BMI velocity strong predictor of total and percent body fat
Casey ³³ 1992 Level III	At birth in 1930s (n = 296), age 18 y (n = 134), at age 50 y (n = 91) White race only Massachusetts (USA)	Tracking BMI with early and late adolescence defined as 2 y before or 2 y after peak height velocity Age categories: childhood, early and late adolescence, ages 18, 30, 40, and 50 y Pearson correlation, simple linear regression	Starting in late adolescence, BMI predictive of adult obesity, especially for males With Foulkes-Davis tracking index, ^a subjects at age 18 y unlikely to change BMI category
Tracking studies of BMI and mortality from childhood or adolescence to adulthood			
Björge ³⁴ 2008 Level III	In 1963–1974 (n = 226,678), age 14–19 y, 34.9 y follow-up Race not specified Norway	Risk of death according to categories of adolescent BMI %tiles: <3; 3–4; 5–9; 10–24; 25–74; 75–84; 85–94; ≥ 95 th or <25; 25–74; 75–84, and >85th, adult BMI categories <18.5; 18.5–22.49; 22.5–24.99; 25–27.49; 25–29.99; ≥ 30 kg/m ² Multivariate Cox proportional hazards models, spline analysis	Adolescent BMI >75th %tile predicts increased mortality in middle age

(continued)

Table 3 (continued)

Study	Population	Intervention	Outcome
Engeland ³⁵ 2003 Level III	In 1963–1975 (n = 128,121), age 10–19 y; follow-up ≥10 y (up to 29 y) Race not specified Norway	Risk for death as adult associated with adolescent “obesity” using adolescent BMI %tiles: <25; 25–74; 75–85; ≥95th at ages 14–15 y, 16–17 y, 18–19 y vs adult ages 25–29, 30–34, 35–40, 40–54 y Logistic regression, multivariate Cox proportional hazards model	Adolescent BMI >75th %tile at higher risk for mortality of all causes in adulthood Adjustment for adult BMI reduces excess mortality observed for men, to a lesser extent for women
Gunnell ³⁶ 1998 Level III	In 1937–1939 (n = 2,990), age 2–14 y; Follow-up to 1995, to age 57 y Race not specified England	BMI in adolescents and adults vs mortality from all causes, from CVD BMI z scores at BMI <25; 25–49; 50–74; ≥75 kg/m ² Hazard ratios with reference = BMI 25th–49th %tile; BMI >90th %tile vs >90th %tile; Cox proportional hazards model Hypothesized that body weight and rate of growth during school-age y directly associated with middle-age mortality from all causes Internally defined relative weight USA, National Standards (1979) Quintiles of growth parameters, nested case-control (1:10) Cox proportional hazards model	Nonlinear association BMI vs overall mortality Reference BMI with lowest mortality Greater mortality risk in older children, those with BMI >90th %tile Age and gender differences in all-cause and CVD mortality
Nieto ³⁷ 1992 Level III	In 1933–1945 (n = 13,146), age 5–18 y; follow-up to 1985 Race not specified Maryland (USA)		Higher mortality with higher relative weight, at both prepubertal and postpubertal age

BMI, body mass index; CDC, Centers for Disease Control and Prevention; CVD, cardiovascular disease; %tile, percentile; OR, odds ratio; ROC, receiver operating characteristic; TSF, triceps skinfold thickness. Studies with measured weight and height rather than self-report have been included.

^aFoulkes-Davis tracking index determines probability that mean of the curves of 2 individuals (with repeated measures) selected at random will not cross over time.

Table 4. Energy Expenditure in Children with Obesity

Study	Population	Intervention	Outcome
Lazzer ⁵⁰ 2006 Level III	Children with BMI >99th %tile Age 7–18 y (n = 574), age 12–18 y (n = 53) Study period not defined White race Italy	Develop and cross-validate new equations for severely obese children and adolescents using indirect calorimetry BMI >99th %tile by Italian growth charts, 1997, body fat by BIA Multiple linear regression, Bland-Altman Compare measured and calculated REE using 14 published equations DXA scan for body composition Simple linear regression, bootstrap analysis Establish new equations using indirect calorimetry, compare with HB, Schofield, WHO, Tverskaya equations; FM from BIA	First equation based on age, gender, weight, and height; second equation based on age, gender, FM, and FFM Both predict REE with mean difference <2%
Schmelzle ⁵¹ 2004 Level III	Children with BMI >95th %tile In 1999–2000 (n = 82), age 4–15 y Race not defined Germany	Multiple linear regression, Bland-Altman Compare measured and calculated REE using 14 published equations DXA scan for body composition Simple linear regression, bootstrap analysis	Published equations for obese children yield scattered data LBM improves accuracy of predicted REE
Derumeaux-Burel ¹³ 2004 Level III	Children with BMI z score ≥ 2 (n = 471 derivation), (n = 211 validation), age 3–18 y Race not defined France	Establish new equations using indirect calorimetry, compare with HB, Schofield, WHO, Tverskaya equations; FM from BIA ANOVA, regression, Bland-Altman	FFM explained >75% of REE in both genders All predictive equations miscalculated REE
McDuffie ¹⁴ 2004 Level III	Children with BMI >95 %tile (n = 502), age 6–11 y Study period not specified Pennsylvania, Louisiana, Washington, DC (USA)	Compare measured REE with FAO/WHO/UNU, Schofield, Molnar, Maffetis, Tverskaya equations, by race; DXA for body composition	After adjusting for race, gender, and overweight status, no equation accurately predicted REE Authors propose new equation
Tverskaya ⁵² 1998 Level III	Children with BMI >28 kg/m ² (n = 110), age 3–18 y in 1992–1996 New York (USA)	Compare measured BMR to equations, create new equations Multiple regression, Bland-Altman	Former equations do not predict BMR accurately New equation predicts within 4% of measured BMR
Kaplan ¹⁵ 1995 Level III	Children with 76% as FTT, 19% obesity in 1988, (n = 102), age 2–10 y in 1990–1994 Pennsylvania (USA)	Measured vs FAO/WHO/UNU, HB, Schofield equations; paired <i>t</i> test	Predictive equations <i>closely</i> predict REE in only 40% of subjects
Molnár ⁵³ 1995 Level III	Children $\geq 120\%$ expected weight for height (n = 371), age 10–16 y Race not defined Hungary	Measured vs calculated REE by FAO/WHO/UNU, Robertson and Reid, Fleisch, Mayo equations ANOVA, simple linear regression	Equations overestimate REE LBM may explain up to 80% of REE
Maffetis ⁵⁴ 1993 Level III	25% of children $\geq 120\%$ expected weight for height (n = 130), age 6–10 y Study period not defined Race not defined Italy	Measured vs calculated REE by FAO/WHO/UNU, Robertson and Reid, Fleisch, Talbot, and Mayo equations skinfold measurement ANOVA, regression	FFM is best predictor of REE Most equations overestimate REE

ANOVA, analysis of variance; BIA, bioelectrical impedance analysis; BMI, body mass index; BMR, basal metabolic rate; CDC, Centers for Disease Control and Prevention; DXA, dual-energy x-ray absorptiometry; FAO/WHO/UNU, Food and Agriculture Organization/World Health Organization/United Nations University equation; FFM, fat-free mass; FM, fat mass; FTT, failure to thrive; HB, Harris-Benedict equation; LBM, lean body mass; REE, resting energy expenditure; SD, standard deviation; WHO, World Health Organization equation.

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A.S.P.E.N. Board of Directors Providing Final Approval

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