A.S.P.E.N. Clinical Guidelines: Nutrition Support of Hospitalized Pediatric Patients With Obesity
Cheryl Jesuit, Cristin Dillon, Charlene Compher, American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) Board of Directors and Carine M. Lenders
JPEN J Parenter Enteral Nutr 2010 34: 13
DOI: 10.1177/0148607109354088

The online version of this article can be found at:
http://pen.sagepub.com/content/34/1/13

Published by:
SAGE
http://www.sagepublications.com

On behalf of:
American Society for Parenteral & Enteral Nutrition

Additional services and information for Journal of Parenteral and Enteral Nutrition can be found at:
Open Access: Immediate free access via SAGE Choice
Email Alerts: http://pen.sagepub.com/cgi/alerts
Subscriptions: http://pen.sagepub.com/subscriptions
Reprints: http://www.sagepub.com/journalsReprints.nav
Permissions: http://www.sagepub.com/journalsPermissions.nav

>> Version of Record - Jan 6, 2010
What is This?
pediatric obesity has reached epidemic proportions in the United States, 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. The origin of pediatric obesity is multifactorial and leads to numerous complications affecting inflammatory processes as well as nutrient metabolism. As a result, current estimations of nutrition status and requirements among obese patients remain unclear. Recognizing that body mass index (BMI) may predict obesity-related complications even in adulthood, the Institute of Medicine (IOM) and, more recently, the American Academy of Pediatrics (AAP) recommend that the term obesity be used in children aged 2–20 years (BMI ≥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, 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. 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. 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...
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 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

### Table 1. Grading of Guidelines and Levels of Evidence

<table>
<thead>
<tr>
<th>Grading of Guidelines</th>
<th>Levels of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>I. Large randomized trials with clear-cut results; low risk of false-positive (alpha) and/or false-negative (beta) error</td>
</tr>
<tr>
<td>B</td>
<td>II. Small, randomized trials with uncertain results; moderate to high risk of false-positive (alpha) and/or false-negative (beta) error</td>
</tr>
<tr>
<td>C</td>
<td>III. Nonrandomized cohort with contemporaneous controls</td>
</tr>
<tr>
<td>D</td>
<td>IV. Nonrandomized cohort with historical controls</td>
</tr>
<tr>
<td>E</td>
<td>V. Case series, uncontrolled studies, and expert opinion</td>
</tr>
</tbody>
</table>


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

<table>
<thead>
<tr>
<th>Guideline Recommendation</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Body mass index is the preferred practical method to screen children for obesity.</td>
<td>D</td>
</tr>
<tr>
<td>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).</td>
<td>E</td>
</tr>
<tr>
<td>3. When possible, energy requirements of obese hospitalized children should be assessed using indirect calorimetry rather than predictive equations.</td>
<td>D</td>
</tr>
<tr>
<td>4. There is no adequate evidence to assess the clinical outcomes of hypertocaloric 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.</td>
<td>E</td>
</tr>
</tbody>
</table>
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 BMI for sex and age charts in childhood predicts 95th percentile on BMI for sex and age charts in childhood predicts adult BMI, obesity, adiposity, and mortality.²⁹-³⁷ (Table 3); however, more tracking (longitudinal) data are needed, especially on clinical risks associated with obesity.¹⁰,³⁸ 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 years¹⁰,³⁹-⁴⁰ 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 ≥95th 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.⁵⁷,⁷,¹⁰,⁴⁶,⁴⁷ 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

<table>
<thead>
<tr>
<th>Study</th>
<th>Population</th>
<th>Intervention</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Freedman29</td>
<td>n = 2,392 Age 5–14 y in 1973–1974, 17 y follow-up</td>
<td>Tracking childhood overweight to adult obesity by race</td>
<td>Tracking differs by race, with 65% (white girls) to 84% (black girls) of “overweight” children becoming obese adults</td>
</tr>
<tr>
<td>Level III</td>
<td>Louisiana (USA)</td>
<td>Longitudinal models for repeated measure data</td>
<td>Childhood BMI strongly predicts adult adiposity</td>
</tr>
<tr>
<td>Level III</td>
<td>Louisiana (USA)</td>
<td>Spearman correlation, simple linear regression</td>
<td></td>
</tr>
<tr>
<td>Guo31</td>
<td>In 1929 (n = 347), age 2–25 y; age 35–45 y</td>
<td>Child vs adult BMI, adiposity, adiposity by TSF</td>
<td></td>
</tr>
<tr>
<td>2000</td>
<td>White race only Ohio, Indiana, Kentucky (USA)</td>
<td>Predict adult overweight/obesity from childhood/adolescent BMI cutoffs</td>
<td>Half of children and adolescents with BMI</td>
</tr>
<tr>
<td>Guo32</td>
<td>In 1929 (n = 338), age 2–25 y; age 35–45 y</td>
<td>ROC, logistic regression</td>
<td>With BMI ≥95th %tile, children 62%–98% more likely to be overweight at age 35 y</td>
</tr>
<tr>
<td>Level III</td>
<td>Hydrostatic weight data (n = 85)</td>
<td>BMI patterns during childhood, puberty, postpuberty vs overweight and body fatness at age 35–45 y</td>
<td>Change in childhood BMI related to adult overweight and adiposity, especially in females</td>
</tr>
<tr>
<td></td>
<td>White race only Ohio, Indiana, Kentucky (USA)</td>
<td>Pediatric BMI gain/y at BMI rebound, puberty, postpuberty</td>
<td>Early BMI rebound associated with maximum BMI velocity and BMI as adult</td>
</tr>
<tr>
<td></td>
<td></td>
<td>General linear models</td>
<td>Maximum BMI velocity strong predictor of total and percent body fat</td>
</tr>
<tr>
<td>Casey33</td>
<td>At birth in 1930s (n = 296), age 18 y (n = 134), at age 50 y (n = 91)</td>
<td>Tracking BMI with early and late adolescence defined as 2 y before or 2 y after peak height velocity</td>
<td>Starting in late adolescence, BMI predictive of adult obesity, especially for males</td>
</tr>
<tr>
<td>Level III</td>
<td>White race only Massachusetts (USA)</td>
<td>Age categories: childhood, early and late adolescence, ages 18, 30, 40, and 50 y</td>
<td>With Foulkes-Davis tracking index, subjects at age 18 y unlikely to change BMI category</td>
</tr>
<tr>
<td>Bjørge34</td>
<td>In 1963–1974 (n = 226,678), age 14–19 y, 34.9 y follow-up</td>
<td>Risk of death according to categories of adolescent BMI %tiles: &lt;3; 3–4; 5–9; 10–24; 25–74; 75–84; 85–94; ≥95th or &lt;25; 25–74; 75–84, and &gt;85th, adult BMI categories &lt;18.5; 18.5–22.49; 22.5–24.99; 25–27.49; 25–29.99; ≥30 kg/m²</td>
<td>Adolescent BMI &gt;75th %tile predicts increased mortality in middle age</td>
</tr>
<tr>
<td>Level III</td>
<td>Race not specified Norway</td>
<td>Multivariate Cox proportional hazards models, spline analysis</td>
<td></td>
</tr>
</tbody>
</table>

(continued)
<table>
<thead>
<tr>
<th>Study</th>
<th>Population</th>
<th>Intervention</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Engeland\textsuperscript{35} 2003 Level III</td>
<td>In 1963–1975 (n = 128,121), age 10–19 y; follow-up \textgreater=10 y (up to 29 y) Race not specified Norway</td>
<td>Risk for death as adult associated with adolescent “obesity” using adolescent BMI %tiles: \textless25; 25–74; 75–85; \textgreater=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</td>
<td>Adolescent BMI \textgreater=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</td>
</tr>
<tr>
<td>Gunnell\textsuperscript{36} 1998 Level III</td>
<td>In 1937–1939 (n = 2,990), age 2–14 y, Follow-up to 1995, to age 57 y Race not specified England</td>
<td>BMI in adolescents and adults vs mortality from all causes, from CVD BMI z scores at BMI \textless25; 25–49; 50–74; \geq75 kg/m\textsuperscript{2} Hazard ratios with reference = BMI 25th–49th %tile; BMI &gt;90th %tile vs &gt;90th %tile; Cox proportional hazards model</td>
<td>Nonlinear association BMI vs overall mortality Reference BMI with lowest mortality Greater mortality risk in older children, those with BMI \textgreater90th %tile Age and gender differences in all-cause and CVD mortality</td>
</tr>
<tr>
<td>Nieto\textsuperscript{37} 1992 Level III</td>
<td>In 1933–1945 (n = 13,146), age 5–18 y, follow-up to 1985 Race not specified Maryland (USA)</td>
<td>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</td>
<td>Higher mortality with higher relative weight, at both prepubertal and postpubertal age</td>
</tr>
</tbody>
</table>

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.

\textsuperscript{a}Foulkes-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

<table>
<thead>
<tr>
<th>Study</th>
<th>Population</th>
<th>Intervention</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lazzeri&lt;sup&gt;50&lt;/sup&gt;</td>
<td>Children with BMI &gt;99th %tile Age 7–18 y (n = 574), age 12–18 y (n = 53)</td>
<td>Develop and cross-validate new equations for severely obese children and adolescents using indirect calorimetry BMI &gt;99th %tile by Italian growth charts, 1997, body fat by BIA</td>
<td>First equation based on age, gender, weight, and height; second equation based on age, gender, FM, and FFM Both predict REE with mean difference &lt;2%</td>
</tr>
<tr>
<td>Schmelzle&lt;sup&gt;51&lt;/sup&gt;</td>
<td>Children with BMI &gt;95th %tile In 1999–2000 (n = 82), age 4–15 y</td>
<td>Compare measured and calculated REE using 14 published equations DXA scan for body composition</td>
<td>Published equations for obese children yield scattered data LBM improves accuracy of predicted REE</td>
</tr>
<tr>
<td>Derumeaux-Burel&lt;sup&gt;11&lt;/sup&gt;</td>
<td>Children with BMI z score ≥2 (n = 471 derivation), (n = 211 validation), age 3–18 y</td>
<td>Establish new equations using indirect calorimetry, compare with HB, Schofield, WHO, Tverskaya equations; FM from BIA</td>
<td>FFM explained &gt;75% of REE in both genders All predictive equations miscalculated REE</td>
</tr>
<tr>
<td>McDuffie&lt;sup&gt;14&lt;/sup&gt;</td>
<td>Children with BMI &gt;95 %tile (n = 502), age 6–11 y</td>
<td>Compare measured REE with FAO/WHO/UNU, Schofield, Molnar, Maffeis, Tverskaya equations, by race; DXA for body composition</td>
<td>After adjusting for race, gender, and overweight status, no equation accurately predicted REE Authors propose new equation</td>
</tr>
<tr>
<td>Tverskaya&lt;sup&gt;52&lt;/sup&gt;</td>
<td>Children with BMI &gt;28 kg/m&lt;sup&gt;2&lt;/sup&gt; (n = 110), age 3–18 y in 1992–1996</td>
<td>Compare measured BMR to equations, create new equations</td>
<td>Former equations do not predict BMR accurately New equation predicts within 4% of measured BMR</td>
</tr>
<tr>
<td>Kaplan&lt;sup&gt;15&lt;/sup&gt;</td>
<td>Children with 76% as FTT, 19% obesity in 1988, (n = 102), age 2–10 y in 1990–1994</td>
<td>Measured vs FAO/WHO/UNU, HB, Schofield equations; paired t test</td>
<td>Predictive equations closely predict REE in only 40% of subjects</td>
</tr>
<tr>
<td>Molnár&lt;sup&gt;53&lt;/sup&gt;</td>
<td>Children ≥120% expected weight for height (n = 371), age 10–16 y</td>
<td>Measured vs calculated REE by FAO/WHO/UNU, Robertson and Reid, Fleisch, Mayo equations</td>
<td>Equations overestimate REE LBM may explain up to 80% of REE</td>
</tr>
<tr>
<td>Maffeis&lt;sup&gt;14&lt;/sup&gt;</td>
<td>25% of children ≥120% expected weight for height (n = 130), age 6–10 y</td>
<td>Measured vs calculated REE by FAO/WHO/UNU, Robertson and Reid, Fleisch, Talbot, and Mayo equations</td>
<td>FFM is best predictor of REE Most equations overestimate REE</td>
</tr>
</tbody>
</table>

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.
Acknowledgments

We thank Kathleen Gura, PharmD, for her guidance at the initiation of these guidelines in 2007. We are very thankful for the contribution of Howard Bauchner, MD, in the review of the manuscript, with support from his 5 K24 HD042489-5.

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

Mark R. Corkins, MD; Tom Jaksic, MD, PhD; Elizabeth M. Lyman, RN, MSN; Ainsley M. Malone, RD, MS; Stephen A. McClave, MD; Jay M. Mirtallo, RPh, BSNSP; Lawrence A. Robinson, PharmD; Kelly A. Tappenden, RD, PhD; Charles Van Way III, MD; Vincent W. Vanek, MD; and John R. Wesley, MD.

References


