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Childhood Obesity for Pediatric Gastroenterologists

Jeannie S. Huang^{*}, Sarah E. Barlow[†], Ruben E. Quiros-Tejiera[‡], Ann Scheimann[§], Joseph Skelton^{||}, David Suskind[¶], Patrika Tsai[#], Victor Uko^{}, Joshua P. Warolin^{††}, and Stavra A. Xanthakos^{‡‡} for the NASPGHAN Obesity Task Force**

^{*}University of California, San Diego and Rady Children's Hospital, San Diego, CA

[†]Department of Pediatrics, Division of Pediatric Gastroenterology, Hepatology, and Nutrition, Baylor College of Medicine, Houston, TX

[‡]University of Nebraska and Children's Hospital & Medical Center, Omaha, NE

[§]Department of Pediatric Gastroenterology, Johns Hopkins School of Medicine, Baltimore, MD

^{||}Department of Pediatrics, Wake Forest School of Medicine, Winston-Salem, NC

[¶]Department of Pediatrics, University of Washington, Seattle, WA

[#]Department of Pediatric Gastroenterology, University of California, San Francisco, CA

^{**}Cleveland Clinic, Cleveland, OH

^{††}Department of Pediatrics, Vanderbilt School of Medicine, Nashville, TN

^{‡‡}Division of Pediatric Gastroenterology, Hepatology, and Nutrition, Cincinnati Children's Hospital Medical Center, Cincinnati, OH

Abstract

Obesity in childhood is one of the major health issues in pediatric health care today. As expected, the prevalence of obesity-related comorbidities has risen in parallel with that of obesity. Consultation regarding these concomitant diseases and subsequent management by subspecialists, including pediatric gastroenterologists, is now common and has resulted in obesity being recognized as a chronic disease requiring coordination of care. Although medications and even surgery may provide effective, though often temporary, treatments for obesity and its comorbidities, behavioral interventions addressing healthy dietary and physical activity habits remain a mainstay in the obesity treatment paradigm. Therefore, the issue of weight management must be addressed by both general practitioner and subspecialist alike. In this report, we review select aspects of pediatric obesity and obesity-related management issues because it relates in particular to the field of pediatric gastroenterology and hepatology.

Keywords

bariatric surgery; diet; micronutrient deficiency; motivational interviewing; nonalcoholic fatty liver disease; obesity; sleep

Qualifying Statements

Further controlled clinical studies may be needed to clarify aspects of this consensus statement. This consensus statement may be revised as necessary to account for changes in technology, new data, or other aspects of clinical practice. This consensus statement is intended to be an educational device to provide information that may assist pediatric gastroenterologists in providing care to pediatric patients who are overweight or obese. This consensus statement is not a rule and should not be construed as establishing a legal standard of care or as encouraging, advocating, requiring, or discouraging any particular treatment. Clinical decisions in any particular case involve a complex analysis of the patient's condition and available courses of action. Therefore, clinical considerations may lead a pediatric gastroenterologist to take a course of action that varies from these guidelines.

Introduction

Obesity in childhood is one of the major health issues in pediatric health care today. As expected, the prevalence of obesity-related comorbidities has risen in parallel with that of obesity. Consultation regarding these concomitant diseases and subsequent management by subspecialists, including pediatric gastroenterologists, is now common and has resulted in obesity being recognized as a chronic disease requiring coordination of care. Although medications and even surgery may provide effective, often temporary, treatments for obesity and its comorbidities, behavioral interventions addressing healthy dietary and physical activity habits remain a mainstay in the obesity treatment paradigm. Therefore, both general practitioner and subspecialist alike must address the issue of weight management. In this report, we review select aspects of pediatric obesity and obesity-related management issues as they relate in particular to the field of pediatric gastroenterology and hepatology.

Epidemiology

The prevalence of childhood obesity in the United States is 17% (1), which is about 3 times higher than rates in the 1960s and 1970s. Nationally representative height and weight measures, obtained before the rise in obesity prevalence, were used to establish norms of body mass index (BMI) for boys and girls ages 2 to 20 years, and BMI 95th percentile from those data is the cut point that presently defines obesity. Using that definition, prevalence rose from about 5% before 1980 to 16.9% overall in 2007–2008 (1). Prevalence of overweight, defined as BMI 85th to 94.9th percentile, also increased from about 10% to 14.8%. Severe obesity is also increasing, although the exact definition is debated. Proposals have included the adult definition of morbid obesity (BMI of 40 kg/m²) in adolescents, BMI of 99th percentile, or a recent proposal of BMI 120% of the obesity cutpoint (95th percentile BMI) (2,3). Each approach has some limitations, but all indicate that 2% to 4% of children meet the criterion, and the related risk of health problems is high in this subgroup. The stabilization of obesity prevalence rates between 1999 and 2008 (1) (among all of the groups except 6 to 19 year old boys with BMI >97th percentile) provides some hope that increased attention to this problem is slowing progression and may eventually lead to gradual reduction. Although obesity affects all of the pediatric populations, regardless of age, race, or sex, prevalence disparities are evident. In particular, Hispanic and African American youth have substantially higher rates of obesity (around 25%) than non-Hispanic white children. Age-related differences are also evident; the obesity prevalence in 2007–2008 among children 6 to 19 years of age was 18.7% in contrast to the prevalence of 10.4% in children 2 to 5 years of age (1). In contrast to adults, poverty is not consistently associated with higher obesity risk in children (4,5).

Etiology

Physiology

Obesity is the result of a complex interplay between the environment and the body's predisposition to obesity based on genetics and epigenetic programming. Although the explanation that excess energy intake or decreased energy expenditure leads to weight gain is attractive in its simplicity, research during the last decade shows that appetite regulation and energy homeostasis rely on a large number of hormones, many of which are secreted by the gastrointestinal tract. Ghrelin is presently the only known orexigenic or appetite-stimulating gut hormone and is secreted primarily by the oxyntic glands of the stomach. It appears to be involved in meal initiation as levels rise shortly before mealtimes (6). Other gut hormones are anorectic or decrease appetite and food intake, including peptide tyrosine tyrosine (PYY), pancreatic polypeptide, oxyntomodulin, amylin, glucagon, glucagon-like peptide-1 (GLP-1), and GLP-2 (7). For example, PYY acts as a satiety signal, and levels rise within 15 minutes after food intake, reducing food intake (8). The secretion profiles of many of these hormones change with bariatric surgery, and these changes are thought to contribute to the improvement of type 2 diabetes mellitus even before significant weight loss occurs (9).

Increased understanding of the role of gastrointestinal hormones in the signaling of hunger, satiety, and energy homeostasis has led to progress in available therapies. In particular, appetite-related gut hormones have become important targets for the development of pharmacologic treatments for obesity and its comorbidities. GLP-1 agonists such as exenatide and liraglutide are available for treatment of type 2 diabetes mellitus, with initial studies demonstrating improvement in HbA1c and limited weight loss; however, concerns regarding potential associations with pancreatitis and cancer may limit their use. Other emerging medications include ghrelin antagonists, PYY and oxyntomodulin analogues, and combination therapies with GLP-1 and glucagon coagonists (10).

Physiologic and Genetic Risk Factors for Obesity

Endocrine disorders, hypothalamic defects (congenital or acquired), medication effects, perinatal environment, and genetic disorders should be considered during evaluation of children with obesity with evaluation based upon detailed history and clinical examination. Development of obesity early in infancy raises concern for mutations of the leptin signaling pathway (extremely rare but potentially treatable by leptin replacement) or melanocortin-4 receptor abnormalities (<5% of children with early-onset obesity) (11,12). Metabolic programming also appears to contribute to obesity. The perinatal environment including the intrauterine hormonal milieu, maternal nutrition status, and postnatal diet may cause epigenetic changes that can increase the risk for obesity. The prevalence of obesity is lower in children born to women after bariatric surgery compared with siblings born to these same women before bariatric surgery (13). Such data suggest that altering the perinatal environment is a potential avenue for preventing obesity. A short duration of obesity is suggestive of an endocrine or central cause of obesity. History of early hypotonia, feeding difficulties with hypogonadism during infancy followed by the onset of rapid weight gain in early childhood without acceleration in growth velocity (due to growth hormone deficiency) raise clinical suspicion for the presence of Prader-Willi-Labhart syndrome, the most common genetic syndrome associated with obesity. Commonly associated features among the obesity-related genetic syndromes include cognitive impairment, relative short stature (due to endocrine abnormalities), digital abnormalities (polydactyly/syndactyly), visual impairment, deafness, and altered onset of pubertal development (14).

Genetic factors also contribute to the development of obesity in childhood independent of clinical disorders and syndromes. Detailed twin, adoption, and family studies have demonstrated that genetic variance between individuals contributes significantly to differences in BMI among adults (15). A variety of mutations in genes associated with appetite regulation (leptin, pro-opiomelanocortin), brain-derived neurotrophic factor, FTO (fat mass– and obesity-associated gene), taste (*TAS2R38*—bitter taste sensitivity/preference for sweet), metabolism (*PNPLA3*), and adipocyte development have been discovered among populations with severe obesity and related comorbidities (16,17).

Although genetic factors may predispose an individual to develop obesity, environmental factors interact with genes to determine development of the obesity phenotype. Ongoing research demonstrates modification of environmental effects by genetic components on the risk for obesity (18). Further large-scale and prospective research needs to be performed to better delineate and describe complex gene-environment interactions.

Environment

It has been generally accepted that environmental factors contribute to the development of obesity via the provision or lack of opportunities for ample physical activity and access to healthy food options, and cultural and social effects; however, the contribution of the built environment to the development of obesity has only recently been evaluated. Built environment characteristics that influence weight-related diet and physical activity behaviors particularly among the disadvantaged include supermarket access or food sources, places to exercise, and safety (19,20). Culture influences perception of risk associated with obesity and behaviors associated with obesity development (ie, feeding practices and engagement in physical activity) (21). There is also intriguing evidence that environmental influences on obesity development may extend to social environments as well, with recent data suggesting spread of obesity through social ties (22).

Clinical Aspects

Comorbid Conditions

Obesity is a health condition that affects nearly every organ system, including the gastrointestinal, musculoskeletal, endocrine, reproductive, cardiovascular, and pulmonary systems. In particular, obese children and adolescents are at increased risk for a large number of medical disorders including tibia vara (Blount disease), slipped capital femoral epiphysis, asthma, sleep-disordered breathing, pseudotumor cerebri, hypertension, type 2 diabetes mellitus, hyperlipidemia, hyperandrogenemia, and polycystic ovarian syndrome (23–25). Obesity also predisposes to psychosocial dysfunction, reduced quality of life, and social isolation (26,27). In regard to gastrointestinal disease, nonalcoholic fatty liver disease (NAFLD), nonalcoholic steatohepatitis (NASH), cirrhosis, and cholelithiasis (23) are related to obesity. These associated disease conditions result from both direct anthropometric changes as well as metabolic derangements related to increased fat mass and insulin resistance (25). Subspecialists should be familiar with screening procedures for common nongastrointestinal obesity-related comorbidities to best expedite care (Table 1) (28–30).

Functional gastrointestinal complaints also are common in the setting of childhood obesity, including constipation, gastro-esophageal reflux disease, irritable bowel syndrome, encopresis, and functional abdominal pain (31). Poor dietary habits in obese children and adolescents appear to be related to constipation and encopresis, whereas abnormal lower esophageal sphincter relaxation and elevated intraabdominal pressure, a consequence of excess subcutaneous and visceral fat, contribute to increased gastroesophageal reflux disease symptoms.

NAFLD/NASH in Children

NAFLD is the most frequent liver disease in children. In a prevalence study of pediatric autopsy cases done during a 10-year period, fatty liver was noted in 5% of normal-weight children, 16% of overweight children, and 38% of obese children with 23% of children with fatty liver showing evidence of NASH (32). Recent data also suggest that risk for NAFLD may also be related to severity of obesity as well as markers of metabolic derangement and inflammation (33). Unlike adults, NAFLD is more common in boys than in girls at a ratio of almost 2:1 (34). In both adults and children, ethnicity appears to affect risk for NAFLD, with the greatest prevalence of NAFLD documented in those of Hispanic background and the lowest prevalence among African Americans when adjusted for BMI (32).

NAFLD is usually asymptomatic and thus screening is required for detection. Unfortunately, presently no screening guidelines have been established outside of recognition of those at risk by weight categorization (overweight or obese: BMI $\geq 85\%$ for age and sex). The diagnosis of NAFLD has relied on the noninvasive detection of markers of liver injury (ie, elevation in liver enzymes) or fibrosis, and/or fatty infiltration on ultrasound or magnetic resonance imaging; however, such diagnostic methods are suboptimal in sensitivity and specificity for NAFLD. Although abdominal ultrasound may demonstrate findings consistent with fatty infiltrate, it does not exclude other causes of liver disease, has limited accuracy in assessing hepatic fat contents, and demonstrates little correlation with fibrosis. As a result, the need to exclude other causes of liver disease including hepatitis B, hepatitis C, autoimmune hepatitis, α_1 -antitrypsin deficiency, and, in older children, Wilson disease is paramount in the evaluation of an overweight or obese child with elevated transaminases.

NASH is the progressive form of NAFLD associated with progression to cirrhosis (35). Ideally, monitoring for development of NASH should be performed; however, noninvasive measures have not proven sensitive in regards to detection of the NASH. Thus, liver biopsy remains the criterion standard for staging and grading of NAFLD. NASH demonstrates a different histologic pattern in children compared with adults (36). Although the classic adult pattern consists of zone 3 involvement with macro-vesicular steatosis, ballooning, and perisinusoidal fibrosis, histologic findings of NASH in children more commonly involve zone 1 with periportal steatosis and portal tract expansion but absence of ballooning degeneration; both adult and pediatric histology patterns can be seen in children (37).

Whether and when to pursue a liver biopsy to establish the diagnosis of NASH in children remains controversial. Proponents suggest that establishing the presence of NASH or documenting significant fibrosis may guide the decision whether to initiate medical and surgical therapies along with lifestyle changes for weight management. Such therapies would include initiation of high-dose vitamin E (38), which has been shown to improve the degree of hepatocellular ballooning degeneration among children with biopsy-proven NASH (38), and/or potentially bariatric surgery (see Bariatric Surgery) (39) in select adolescents. Others, however, maintain that the risk-benefit ratio of liver biopsy compared with present empirical treatment options is still too high to support universal performance of liver biopsy. Presently, the only established treatment for NAFLD/NASH is lifestyle changes for weight loss and management.

Micronutrient Deficiencies

Emerging evidence suggests that multiple micronutrient deficiencies are more prevalent in overweight and obese children and adults as compared with normal-weight counterparts (40). Obesity is now recognized as a risk factor for a number of nutritional and micronutrient deficiencies, including fat-soluble vitamins, minerals, and antioxidants.

Consumption of energy-dense, yet nutrient-poor foods likely contributes to the development of such nutritional deficiencies. In particular, increased consumption of sugar-sweetened beverages is linked with calcium and vitamin D deficiency (presumably via reduced milk intake) (40). In addition, multiple studies have shown an inverse relation between BMI and serum vitamin D (25-OH) levels, possibly related to reduced activity level, decreased sun exposure, and increased storage in adipose tissue (41). Serum levels of fat-soluble antioxidants, including β -carotene (vitamin A) and α -tocopherol (vitamin E), are also significantly lower in obese children, potentially leading to increased low-density lipoprotein oxidation (42). The relation of dietary intake of fat to inadequate nutrient intake is less clear. Two pediatric studies have suggested that higher fat diets carry an increased risk of suboptimal intake of vitamins A, D, and folate (43,44). Conversely, lower fat intake may also be associated with lower intake of calcium, magnesium, phosphorous, vitamin E, vitamin B₁₂, thiamine, niacin, riboflavin, and zinc (45). In the setting of bariatric surgery, micronutrient deficiencies are common and include that of iron, ferritin, thiamine, copper, and vitamin B₁₂ (45,46).

Sleep

Children with fewer hours of sleep are more likely to be overweight or obese by BMI criteria. This association between sleep duration and obesity has been well established in cross-sectional studies on a broad range of ages and also in many different countries (47). Generally, less sleep increases odds of obesity by about 1.5. In addition, a dose response was demonstrated in a large Japanese study, with higher risk associated with fewer hours of sleep (48). Results from longitudinal cohort studies have been mixed; although several have shown inverse relationships between sleep duration and overweight and obesity (49–51), a large study from Australia showed no relationship between sleep duration for 0 to 5 years and BMI status at age 7 years (52).

Although the cross-sectional studies do control for factors known to be associated with obesity, such as parental BMI, the relation between sleep and obesity is not necessarily causal. Another caveat is potential reporter bias; in most studies, parent report rather than direct measurement establishes sleep duration. Although sleep duration is modifiable and therefore ripe for intervention, little research has been done either to establish the cause and effect relation or to demonstrate benefits of modification. In one recent intervention study, primary care clinicians provided counseling to parents of infants and assessed sleep at age 6 years (53). The study showed no effect on BMI at age 6 years, but the intervention was low intensity and far removed from BMI assessment (53).

Despite uncertainty about sleep's role in obesity, promotion of adequate sleep is recommended because of cognitive and health benefits and low risk of harm. The Centers for Disease Control and Prevention endorse the recommendations from the National Sleep Foundation for hours of sleep at different ages (Table 2).

Weight Bias and Bullying

Discrimination against overweight people is as common as discrimination based on sexual orientation, ethnicity, physical disability, and religion (54). Despite the common prevalence of overweight and obesity, weight-based stigmatization remains a relatively acceptable societal norm, perhaps due to a lack of knowledge about the resulting harmful effects on these individuals. There is nevertheless evidence suggesting adult obese patients have an increased prevalence of psychiatric morbidity, most commonly depression, related to weight bias and particularly as a result of being bullied in school and psychiatric morbidity carried over from childhood (55).

Weight-based peer victimization is defined as unsolicited bullying and teasing as a result of weight status and can be either overt or relational. Youth who are obese are more likely to be bullied, regardless of demographics or social and academic standing (56). Among school-age children, the odds of being bullied are 26% greater among the overweight as compared with their normal weight peers, and 85% greater in obese children (57). In addition, overweight and obese adolescents up to 15 years of age have much greater odds of being victims of bullying and aggression as well as withdrawn friendships, rumors and lies, name calling, teasing, hitting, and kicking than normal-weight adolescents (58). At 15 to 16 years of age, however, when compared to normal-weight classmates, boys and girls with increased BMI were more likely to be the perpetrators of bullying (58). These results indicate that overweight and obese school-aged children are more likely to be not only the victims but also the perpetrators of bullying behaviors as compared with their normal-weight peers. The unfortunate outcomes of peer victimization based on weight status are low self-esteem, body dissatisfaction, social isolation, marginalization, poor psychosocial adjustment, depression, eating disorders, suicidal ideation, suicide attempts, and poor academic performance.

There are evidence-based bullying interventions and prevention programs, which have been implemented in the school setting and via teacher training (59,60). In addition, there are many resource books for parents. Although it has not been recommended to regularly screen for bullying, it is important for physicians to be aware of the problem in overweight and obese children and to know that although overweight and obese children can be the victims of bullying, they can also be the perpetrators of bullying behaviors (58). Physicians should never minimize or ignore a parent or child's report of bullying and teasing, and be sympathetic and supportive to their concerns. Furthermore, physicians should be cognizant of their own feelings and biases toward families with overweight or obese children (61). Indeed, stigmatization of obese individuals may counteract and be detrimental to efforts to motivate or encourage adoption of healthier behaviors and weight.

Gastrointestinal Procedural Issues

Childhood obesity is associated with a number of gastrointestinal complaints and conditions. During initial evaluation and subsequent monitoring of these conditions, obese children may undergo a variety of gastrointestinal procedures including, but not limited to endoscopy, liver biopsy, cholecystectomy, as well as bariatric surgical procedures. In preparation for these procedures, screening for obesity-related conditions associated with perioperative complications such as asthma, hypertension, sleep apnea, and diabetes mellitus should be performed. Obesity itself also affects perioperative risk and increases the Physical Status classification of the American Society of Anesthesiologist (ASA-PS) (62) with higher ASA-PS classification correlating with mortality (63).

Perioperative adverse events, such as arterial oxygen desaturation, difficult mask ventilation, airway obstruction, bronchospasm, and resulting morbidity occur more frequently in obese as compared with nonobese children undergoing surgical procedures (64). Required specifications for the obese patient have been published (65). Both overdosing and subtherapeutic dosing have been described in obese patients (66), which may result from differences in volume of distribution and drug metabolism (Table 3) (67–70). To reduce the likelihood of adverse events, appropriate dosing scalars such as lean body weight should be used for obese patients.

It is important to have appropriately sized instruments, such as high-capacity scales, large blood pressure cuffs (large adult and thigh sized), power-assisted transport gurneys, and extra-wide surgical tables, to accommodate the needs of the obese patient (71). Similarly, modifications may have to be made to diagnostic imaging techniques (eg, ultrasound, conventional radiology) to account for the size and habitus of obese patients. Additional

considerations that require attention include the ingress and egress of patients from clinical areas, appropriately sized seating for both patients and adult family members, who may also be significantly obese, as well as accommodations for obese patients and families in nonclinical areas such as waiting rooms, lobbies, and restrooms. Required specifications for the obese patient have been published by the American Institute of Architects (72).

Treatment

The foundation of obesity treatment is behavioral change to improve energy balance through improved diet and physical activity. Other therapies, including medication and surgery, have all been evaluated in the context of ongoing support to change health behaviors. The United States Preventive Services Task Force reviewed the evidence and concluded that comprehensive, moderate- to high-intensity programs were effective for treatment of childhood obesity. Comprehensive programs provided counseling on healthy diet, physical activity, and techniques for behavior change; moderate- to high-intensity programs engaged participants in more than 25 hours of contact. Weight loss in such programs is modest, especially in contrast to the dramatic change sought by patients, parents, and providers, but can be sustained (73). Ideally, pediatric gastroenterologists should refer obese patients, with their parents, to such programs when initial lifestyle modification counseling has not been successful, although lack of program availability and lack of insurance coverage for the programs are barriers.

Dietary Therapy

There are a number of dietary therapies touted for weight loss and weight management. A recent comparison trial of the various macronutrient weight-loss diets among adults concluded that reduced-energy diets regardless of macronutrient composition result in clinically meaningful weight loss, although actual differences in macronutrient compositions between study diets were modest (74). Although data exist in general for adults in regards to the efficacy of varied nutritional regimens, data are relatively sparse for children and adolescents. Available randomized controlled trial (RCT) data examining diets exclusively among youth are few and have been limited by high attrition rates and relatively short-term follow-up periods. The pediatric portion of the Diogenes (diet, obesity, and genes) dietary study performed in 8 European countries examined the effect of a 26-week dietary intervention on weight changes among families with at least 1 overweight parent/adult and 1 healthy child. Children were randomized (along with their overweight parent) to specific ad libitum diets (5 low-fat [25%–30% fat by energy]) diets were available: low protein (LP) and low glycemic index (LGI); LP and high glycemic index (HGI); high protein (HP) and LGI; HP and HGI; and control). Of the 800 children enrolled and randomized, 465 (58%) completed all of the assessments (baseline and 4 and 26 weeks). Although neither GI nor protein dietary content had an effect on body composition separately, the LP and HGI diet increased body fat (1.5%), whereas the HP and LGI diet reduced the percentage of overweight or obese children (46.2% at baseline to 39.6% at 26 weeks) during the study period (75). The high dropout rate limits the validity of these findings. Another study evaluated meal replacements (MR) as a method of weight loss in a RCT of 1300- to 1500-calorie diets delivered via MR $\times$ 12 months versus MR $\times$ 4 months + conventional food diet (CFD) $\times$ 8 months versus CFD $\times$ 12 months among 120 obese adolescents. Among the 75 adolescents completing the trial, participants receiving MR had greater weight loss at month 4 (6.3% reduction in BMI vs 3.8% BMI reduction, MR vs CFD, $P = 0.01$) compared with CFD, but no differences in weight outcomes were seen between groups at 12 months. In fact, all of the groups increased their BMI in months 5 to 12 of the study. Thus, although MR improved short-term weight loss compared with CFD, continued use did not promote continued weight loss in the long term (76). In general, long-term diets must be palatable

and adaptable to the typical lifestyle to maintain patient compliance with prescribed regimens.

Dietary therapy may also be considered for acute management of severe obesity, in which the goal of therapy is to quickly reduce weight and associated morbidities. In particular, there are data to suggest that extremely low energy diets such as the protein-sparing modified fast are well tolerated with noteworthy metabolic, cardiovascular risk, and obstructive sleep apnea improvements (77,78). Among children, the protein-sparing modified fast (600–800 kcal/day with 2 g/kg protein up to 100 g/day) has demonstrated promising results on weight and serum lipids in the short term (79,80); however, such diets require close medical management and monitoring, and the benefits of such restrictive diets in the long term in both children and adults have yet to be demonstrated.

Motivational Interviewing

Today, weight loss through decreasing energy intake and increased physical activity remains the cornerstone to treat not only obesity but also its associated comorbidities. Because behavioral modification remains the key to success, physicians, both generalist and subspecialist alike, must effectively counsel patients to adopt and maintain healthy weight-related behaviors. In particular, weight reduction and maintenance behaviors recommended by the 2007 American Academy of Pediatrics' Recommendations for Treatment of Child and Adolescent Overweight and Obesity include avoidance of sugar-sweetened beverages, reduced portion size, intake of 5 to 9 fruit and vegetable servings per day, 1 hour of moderate to vigorous physical activity daily, daily breakfast, maximum daily screen-time exposure of 2 hours, and eating at home (vs at fast food restaurants) (81).

Present guidelines advocate the use of patient-centered communication as a means to motivate families to change behaviors (81). Motivational interviewing (MI) is a client-centered approach used to enhance an individual's intrinsic motivation for behavior change by exploring ambivalence and taking steps to resolve it (82). MI has been successfully applied to other health behaviors, including alcoholism (83), diabetes mellitus (84), and HIV (85). A critical component of MI is an empathetic care provider who understands that patient ambivalence is normal and responds to such ambivalence nonjudgmentally. Without undue confrontation, care providers can use MI to collaboratively guide patients toward health-related goals by allowing them to express their own motivations for change (82). Reflective listening techniques and open-ended questions are important tools during the MI process that focus communication toward change, rather than maintenance of present behaviors.

Despite its promise, MI has been the focus of few pediatric obesity treatment studies. Schwartz et al (86) examined the feasibility of implementation of MI by physicians and dietitians in the primary care setting. In general, results suggested promise for the MI approach with parents reporting positive perceptions of the MI counseling approach with some indications of improvement in behavioral and weight parameters; however, there were methodological study issues including nonrandomization of selection of participating practices and physicians and high dropout rates that limit these preliminary findings (86). Another study examined an MI-based intervention delivered in person and via telephone to families seen at 10 pediatric practices via a cluster RCT (87). Although intervention participants did demonstrate greater decreases in television viewing, fast food and sugar-sweetened intake, and BMI as compared with control counterparts, only television viewing reductions achieved statistical significance. Additional studies emphasizing MI as a primary mode of intervention are forthcoming (88,89) and will provide further comment on whether interventions using this approach improve program adherence and behavioral outcomes in pediatric obesity treatment.

Drug Therapy

Historically, major issues about safety and efficacy of drugs used for weight reduction have led to significant concerns about the role of pharmacological therapy in weight management. To meet criteria for labeling as a “weight loss” drug, an agent must induce 5% weight loss in clinical trials. This criterion is based upon medical reports demonstrating that weight reduction of 5% decreases the risk for diabetes mellitus and cardiovascular disease (90). The only Food and Drug Administration (FDA)–approved weight loss drug for long-term use in adults and children is Orlistat (Xenical). Orlistat causes fat malabsorption by inhibiting pancreatic lipase, but has also been linked to reduction in fat-soluble vitamins and β -carotene as well as gastrointestinal adverse effects. Orlistat's efficacy in reducing weight has been modest; among adolescents taking Orlistat in conjunction with a lifestyle modification intervention, only 26% achieved >5% weight loss (91). For adults, noradrenergic drugs have been approved for weight loss and appetite suppression, including phentermine, phendimetrazine, and diethylpropion; however, these drugs are only approved for short-term use because of their potential for abuse, cardiovascular (sympathomimetic) adverse effects, and lack of data about long-term safety and efficacy. Metformin, widely used in obese children with type 2 diabetes mellitus, is not considered a weight loss drug because the decrease in weight in clinical trials has been relatively small (BMI z score difference between metformin vs placebo = -0.07 among obese insulin-resistant children) (92).

Bariatric Surgery

Weight loss surgery (WLS) has been performed in adolescents sporadically and in small numbers since 1970; however, with the growing prevalence of obesity and related comorbidities in youth, the rate of WLS among adolescents has tripled between 2000 and 2003 (93). This likely reflects not only an increased awareness of severe pediatric obesity and its associated health risks but more important the continued paucity of effective lifestyle and medication interventions for severe pediatric obesity and severe obesity in general. Until recently, adolescent WLS represented <1% of overall bariatric surgery procedures in the United States, with Roux-en-y gastric bypass (RYGB) comprising >90% of cases (93). With increasing data on the safety and efficacy of WLS in teens, the rate of surgery is likely to rise and the types of surgeries offered likely to broaden. For example, the adjustable gastric band (AGB), though not yet FDA approved for patients younger than 18 years, has been studied in industry-sponsored studies in youth as young as 14 years. Although attractive, because of its reduced short-term operative complication risk, AGB has a higher rate of reoperation compared with RYGB in both adults and adolescents (94). Vertical sleeve gastrectomy (VSG) has gained increased interest in the last 5 years as a stand-alone WLS in both adults and adolescents because it shows similar weight loss and comorbidity resolution when compared to RYGB, but long-term data (>5 years) are lacking in both age groups. More aggressive malabsorptive weight loss procedures, such as biliopancreatic diversion with duodenal switch and jejunoileal bypass, are not recommended for adolescents.

WLS results in weight loss in part through dietary restriction (AGB, VSG, RYGB) and malabsorption (RYGB), but also appears to alter secretion of neuroenteric hormones that regulate appetite and energy expenditure beneficially (78). All of the 3 surgical options (AGB, RYGB, VSG) result in clinically significant weight loss, with patients undergoing RYGB and VSG losing on average 50% to 60% of excess weight, which appears to be sustainable in a majority of patients (95,96). AGB results in comparable or slightly lower excess weight loss at a slower, more gradual rate (97). A large prospective controlled study following long-term outcomes among adults who had undergone WLS (AGB, vertical banded gastroplasty, or RYGB) (98) demonstrated a range of 15% to 25% mean weight loss at 15 years postoperative follow-up, although mean weight loss regressed from the maximal

nadir at 1 year; however, of the 265 patients with RYGB enrolled in the present study, <25% (n = 58) were included in the 10-year follow-up measurement and only 4% (n = 10) in the 15-year follow-up.

To date, long-term studies of outcomes of adolescent WLS are limited by retrospective design, small case series, and substantial loss to follow-up at longer time points; however, a recent 2008 systematic review and meta-analysis (94) including only adolescent studies reporting 1 year outcomes on at least 50% of the original cohort demonstrated that AGB resulted in a 95% confidence interval of -13.7% to -10.6% BMI unit decrease at 1 to 3 years postsurgery, whereas RYGB yielded a more substantial BMI unit decrease (95% confidence interval -17.8% to -22.3% BMI units decrease) 1 to 6 years postsurgery. Well-characterized, prospective, longer term cohort data are in the process of being collected by a multicenter consortium sponsored by the National Institutes of Health, but it will take decades to establish whether the positive outcomes seen after adolescent WLS will be sustained over a longer expected postsurgical lifetime period of >60 years.

Along with weight loss, significant postsurgical improvements in key comorbidities, such as diabetes mellitus, obstructive sleep apnea, depression, cardiovascular function, dyslipidemia, and quality of life, have been demonstrated (99). Progressive NASH has been proposed as a major indication for WLS in adolescents; however, there is a lack of controlled prospective data supporting the efficacy of WLS for the treatment of NASH, although a metaanalysis of retrospective data in adults suggests a 70% resolution of NASH with WLS (100).

Short-term complications after RYGB and VSG may include leakage at anastomotic sites, wound infections, and gastrojejunal strictures requiring endoscopic dilatation, small bowel obstruction, and development of cholelithiasis. The gastroenterologist may be asked to assist with dilation of strictures and stent placement for leaks. In addition, after RYGB, access to the bypassed stomach and biliopancreatic limb is challenging and may require special endoscopic techniques and equipment (eg, double balloon enteroscopy, endoscopic ultrasound-assisted, or laparoscopic-assisted techniques). Band slippage, gastric obstruction, and esophageal or gastric pouch dilation have been reported after AGB placement; and up to 30% of adult patients with AGB may require major reoperation (101). The incidence of short-term operative complications for adolescents undergoing WLS in academic centers appears to be lower than that seen in adults and no perioperative mortality has been reported (93,102). Long-term complications appear to be primarily nutritional because of deficient intake or absorption of iron, vitamin B₁₂, vitamin D, and other key nutrients; and lifelong vitamin and mineral supplementation is recommended after all types of bariatric surgery. Adherence to supplementation may be particularly poor among adolescents, necessitating close follow-up to monitor serum levels and for symptoms of deficiencies.

Pediatric Obesity Advocacy

Clinicians are called to intervene on behalf of their patients not only in the clinical setting but in the community as well. The 2005 Institute of Medicine report recommends that clinicians, regardless of specialty, serve as role models and provide leadership in their communities for obesity-prevention efforts (103). Successful advocacy for preventing pediatric obesity requires involvement in and beyond the office and includes working with the community and helping to formulate public policy.

Specific interventions include, but are not limited to the following:

1. Routinely discussing obesity prevention recommendations with patients and families (104)

2. Leading by example such as advocating for and providing healthy food options in hospitals
3. Involving the medical/hospital staff in community advocacy programs; community/hospital partnerships, locally and at the state level, have been successfully established across the country and are models for continued efforts (104); physicians can become active at all levels, including speaking at public forums and attending community/school board meetings to offer medical expertise on the issue of pediatric obesity (104)
4. Helping to formulate federal, state, and local policies addressing the problem of pediatric obesity; pediatric gastroenterologists have a unique, and especially qualifying, expertise to serve as spokespersons for pediatric obesity prevention; this includes expertise in nutrition for schools, health education, physical education and activities, food advertising directed to children, and environmental policies such as city green spaces (86)
5. Continuing the efforts of all of the major pediatric societies, such as NASPGHAN, in drafting policy recommendations that deal with pediatric obesity–related issues on the local, state, and federal levels to help effect positive change

Although many pediatric healthcare professionals have answered the call to advocacy with involvement in their local and national communities on topics such as obesity, these unfortunately remain the minority of the available workforce. The paucity of the scientific literature in discussing advocacy as a central issue is evident, with relatively few PubMed articles identified addressing pediatricians with “advocacy” in the title (N = 5) or as a topic (N = 72) of the article (as of June 2011). Nevertheless, public health issues are often most effectively addressed at a population level via interventions involving publicity and effective legislation and policy change (105). Thus, to improve child health and childhood obesity in the present era, physicians and pediatric gastroenterologists must begin to take action, influence, and join community leaders in joint efforts to effect and enact health and environmental policies that permit and encourage healthy behavior choices and lifestyle modification (Table 4).

Conclusions

Obesity is a chronic disease affecting youth that requires collaborative input from both subspecialty and primary care physicians. The role of the pediatric gastroenterologist in the treatment of pediatric obesity is well established given common coexistence of not only functional gastrointestinal disorders but also organic gastrointestinal disease with obesity in childhood. In addition, because of the diverse health effects of obesity, the pediatric gastroenterologist should have a working knowledge of the comorbidities of obesity that affect gastrointestinal as well as other organ systems. Tertiary care interventions often require subspecialty input and gastroenterology providers should thus be familiar with the indications, benefits, and risks of bariatric surgery, as well as the resultant changes in gastrointestinal anatomy and function that may modify indications, risks, and benefits of subsequent endoscopic procedures. In determining treatment plans for the obese child/adolescent, the pediatric gastroenterologist should account for the whole patient (inclusive of family and social situation, and built environment) to achieve the long-term lifestyle modifications necessary to effectively reduce weight and associated comorbidity risk.

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Table 1
Screening procedures for obesity-related comorbidities

Comorbidity	Screening procedures and criteria
Cardiovascular	Clinical and laboratory evaluation
Hypertension	Blood pressure (3 separate readings BP >95% age, height, sex (28))
Hyperlipidemia	Fasting lipids (Total cholesterol >170, LDL-cholesterol >110, triglyceride >100, HDL-cholesterol >45) (29)
Pulmonary	Assess persistent snoring, pauses in breathing, nocturnal enuresis, and daytime somnolence; refer for sleep study if symptoms present; consider otolaryngology referral if symptomatic and tonsils are enlarged
Obstructive sleep apnea	
Obesity hypoventilation syndrome	
Psychiatric	Clinical evaluation
Depression	Clinical symptoms (eg, anorexia, poor sleep, suicidal ideation, anhedonia)
Orthopedic	Symptom and examination screening
Blount disease	Blount = pain at medial aspect of knee (adolescents), 80% unilateral, difference in leg length
SCFE	SCFE = hip/knee pain, decreased internal rotation of hip, may progress to inability to bear weight/decreased range of motion
Endocrinologic	Clinical and laboratory evaluation
	Acanthosis nigricans suggests hyperinsulinemia
	Fasting glucose: diabetes 126 mg/dL, impaired 100–126 mg/dL
Diabetes/insulin resistance	2-h oral glucose tolerance test: diabetes >200 mg/dL, impaired 140–200 mg/dL
	Hemoglobin A1c: diabetes 6.5%, prediabetes 5.7%–6.4% (30)
	Diagnosis of diabetes requires abnormal tests on 2 different days unless there is “unequivocal hyperglycemia”
Polycystic ovarian syndrome	Rotterdam criteria: presence of any 2 of 3 features (a) Oligo/amenorrhea (b) Clinical hyperandrogenism (c) Polycystic ovaries (>10 cysts)—pelvic ultrasound

HDL = high-density lipoprotein; LDL = low-density lipoprotein; SCFE = slipped capital femoral epiphysis.

Table 2
National Sleep Foundation recommendations for hours of sleep at different ages

Age	Recommended sleep, h
Birth–2 mo	12–18
3–11 mo	14–15
1–3 y	12–14
3–5 y	11–13
5–10 y	10–11
10–17 y	8.5–9.5

Table 3
Volume of distribution and pharmacokinetic half-life of common sedatives used for gastrointestinal procedures according to weight in obese patients

Sedative	Dose calculation	Volume of distribution	Half-life
Chloral hydrate (67)	Based on TBW	Highly protein bound, hence a low volume of distribution	8–11 h
Fentanyl	Based on LBW* In obese patients, the dose should be adjusted to individual needs because of an increased risk for hypoxia postoperatively	Highly lipophilic 4–6 L/kg	Three compartment model: Phase 1: 6 min Phase 2: 60 min Phase 3: 16 h Mean: 2 to 4 h
Ketamine (68)	Based on IBW†	Highly lipophilic 2 L/Kg	2.5 h
Midazolam (69)	Based on TBW‡	Large volume of distribution and highly lipophilic 1–3 L/kg	1–4 h Half-life is increased in obesity and liver cirrhosis
Propofol (70)	Based on TBW‡	Large volume of distribution and highly lipophilic 5–10 L/kg	Three compartment model: Phase 1: 2–3 min Phase 2: 40 min Phase 3: 300–700 min

BMI = body mass index; IBW = ideal body weight; LBW = lean body weight; TBW = total body weight.

* LBW is weight devoid of all adipose tissue and is calculated as follows: male, $1.10 \times \text{TBW} - 0.0128 \times \text{BMI} \times \text{TBW}$; female, $1.07 \times \text{TBW} - 0.0148 \times \text{BMI} \times \text{TBW}$.

† IBW is calculated as follows: male, $49.9 \text{ kg} + 0.89 \times (\text{height in cm} - 152.4) \text{ kg}$; female, $45.4 \text{ kg} + 0.89 \times (\text{height in cm} - 142.4) \text{ kg}$.

‡ This is applicable if used as a single dose. If used as a continuous infusion, the dose should be based on the IBW.

Table 4
Summary and recommendations

Epidemiology	About 1 in 3 children and adolescents is affected by overweight and obesity
	Obesity is particularly prevalent among Mexican American and African American youth
Etiology	Hormonal factors affect appetite and satiety
	Genetics, perinatal metabolic programming, and the environment (social, media, cultural, and built) contribute to the development of obesity
	Obesity comorbidities are common and should be screened for by health care professionals (Table 1)
	Functional GI disorders are also common in obesity, including GERD, altered bowel habits, and abdominal pain
Clinical considerations	NAFLD/NASH is the most common liver disorder in children; detection is limited by suboptimal noninvasive screening methods and diagnosis requires exclusion of other causes of liver disease; staging of disease requires liver biopsy
	Micronutrient deficiencies are common in obesity and may result from unhealthy dietary habits as well as treatment (particularly surgical)
	Reduced sleep duration is associated with increased risk of obesity
	Care professionals should be aware of weight bias, discrimination, and victimization as they treat the obese child
	Clinicians should ensure adequate facilities for and account for increased perioperative risks and altered pharmacokinetics of medications commonly used for sedation in obese patients requiring GI procedures
	Overall, multidisciplinary, behavior-based programs should be used when lifestyle modification counseling has not worked and when available; family involvement is necessary to ensure success
	Lifestyle changes and adoption of healthy physical activity and dietary behaviors remain the mainstay of weight management interventions
Clinical treatment	Motivational interviewing, a client-centered communication approach, offers promise in effecting such behavioral change
	To date, only Orlistat is FDA approved for weight loss in children and adolescents with modest success
	Bariatric surgery results in significant weight loss and metabolic improvements for the majority of patients and remains the most effective therapy for severe obesity in adults and adolescents with significant BMI declines and improvements in comorbidities in the short term; however, widespread use is limited because of concerns related to surgical risks and psychological maturity to fully understand risks and benefits and provide full assent; long-term safety and efficacy is not known for this age group; postoperative micronutrient deficiencies are common and must be treated
Advocacy	Pediatric gastroenterologists should join community leaders in joint efforts to effect and enact health and environmental policies that permit and encourage healthy behavior choice and lifestyle modification
Patient resources	Patient handouts and information: http://www.gastrokids.org

BMI = body mass index; FDA = Food and Drug Administration; GERD = gastroesophageal reflux disease; GI = gastrointestinal; NAFLD = nonalcoholic fatty liver disease; NASH = nonalcoholic steatohepatitis.