Extreme Pediatric Obesity: Weighing the Health Dangers

Pediatric healthcare workers and researchers have long embraced the term overweight to describe children with excess adiposity, and they have reserved the arguably more stigmatizing term obesity for the adult form of this disease. In adults, necessity has driven the use of even more explicit terminology to describe those in the upper echelons of weight. Data from the Behavioral Risk Factor Surveillance System indicate that extremely obese (body mass index [BMI] ≥40 kg/m^2) adults quadrupled in number, from 0.5% to 2%, between the years 1986 and 2000. In fact, estimates from 2003 to 2004 show that 7% of women and 3% of men are extremely obese. This adult epidemic appears to be foreshadowing a similar problem in children. Obesity prevalence has doubled in children and tripled in adolescents over the last three decades. Clinicians and parents have increasingly appreciated that our children are also affected by extreme obesity, but no systematic attempts have been made to describe the epidemiology or clinical relevance of extreme obesity in pediatric age groups until now.

In this issue of The Journal, Freedman et al critically analyzed two large population-based datasets and determined that a staggering 4% of children and adolescents in the US now have a BMI for age ≥99th percentile, and nearly all of these children (94%) have excess adiposity. By the total number of children and adolescents in the US in 2005, more than 2 million children may therefore be affected by this extreme of obesity. More importantly, these authors also quantify cardiovascular and metabolic risk factors (including dyslipidemia, hypertension, and hyperinsulinemia) in the highest percentiles of BMI, using data from the Bogalusa Heart Study. Although numerous prior studies have focused on health risks associated with pediatric overweight (>85th percentile) and obesity (>95th percentile), this is the first attempt to explicitly examine the health burdens of those at the very highest extremes of pediatric obesity. Their cross-sectional look at more than 6000 children in the Bogalusa Heart Study demonstrates that of children with BMI ≥95th percentile, 39% had at least two risk factors, although among those with a BMI ≥99th percentile, the proportion with these risk factors dramatically increased to 59%. Interestingly, this likelihood of harboring “silent” cardiovascular risk factors was independent of age, with those <12 and >12 years of age being similarly affected by these comorbidities of obesity.

Indeed, based on this information, it appears safe to assume that the 99th percentile not only describes a large population that is extremely obese, but it also describes true health risk because it reliably identifies children with a high likelihood of having associated cardiovascular and metabolic abnormalities. In this regard, the 99th percentile for children and adolescents seems to describe the childhood equivalent of clinically significant, extreme obesity.

Use of the 99th percentile as a definition for extreme pediatric obesity also has two other important attributes: persistence into adulthood and synchrony with adult definitions. Freedman et al have shown in a longitudinal analysis that all of the children in the 99th percentile remained obese into adulthood with an average adult BMI of 43 kg/m^2. Therefore, the 99th percentile serves as a useful predictor of future adult morbidity. Obesity is an independent risk factor for development of type II diabetes mellitus, and elevated childhood BMI has repeatedly been associated with increased risks of cardiovascular disease, left ventricular hypertrophy, and mortality in early adulthood. Also, the 99th percentile makes more intuitive “sense” as a definition for pediatric extreme obesity because this 99th percentile cut-point for older adolescents approximates BMI values between 35 and 40 kg/m^2.

If we accept the 99th percentile as an evidence-based criterion for extreme pediatric obesity with clinical significance, what are the implications? Although prevention is an integral component of pediatric weight management,
once an individual has developed obesity, effective treatment approaches become paramount. Unfortunately, therapeutic options for obese children and adolescents are limited by availability, treatment effectiveness, and insurance coverage. The first line of treatment for pediatric obesity remains behavioral modification to decrease caloric intake and increase physical activity. However, weight loss in obese and overweight children and adolescents achieved by behavioral therapy is time intensive, generally produces modest effects, and has only rarely been shown to be sustainable in the real world. Likewise, pharmacotherapy to either decrease appetite or promote malabsorption of fat has only been moderately effective, with frequent weight regain once the medications are stopped voluntarily or due to undesirable side effects. Significantly, these types of interventions may be even less effective for the most extremely obese children and adolescents.

Bariatric surgery at present is the only intervention that has been shown to provide sustained weight loss with resolution of associated comorbidities in extremely obese adults and adolescents. Current criteria for bariatric surgery in adolescents are a conservative adaptation of National Institutes of Health (NIH) consensus guidelines for adults. Pediatric patients with extreme obesity should be considered for bariatric interventions by a multidisciplinary team with pediatric, dietary, psychological, and surgical expertise. General guidelines suggest that surgical intervention not be considered for youth unless a person has reached a BMI of at least 40 kg/m² and has developed severe medical comorbidity (type II diabetes, obstructive sleep apnea, pseudotumor cerebri) or has reached a BMI of 50 kg/m² with either a less severe medical comorbidity or significant psychosocial or functional impairments. Of note, these guidelines have been challenged as too conservative if rigidly applied.

In the face of mounting evidence of medical and psychosocial risks of extreme obesity in youth, and the increasing success and acceptance of surgical therapies for pediatric obesity, Freedman et al provide an evidence base to begin to define a disease process that is a major health threat for children. The 99th percentile of BMI for age coupled with the presence of a comorbidity or significant risk factor may represent a clinically justifiable starting point for use of intensive therapies or surgical weight loss interventions.

Irrespective of what guidelines are used as indications for bariatric surgery in youth, it will be important to prospectively examine in greater detail the surgical outcomes for morbidly obese adolescents to better characterize benefits and safety. In this regard, the NIH–funded Teen Longitudinal Assessment of Bariatric Surgery (Teen–LABS) study is a multicenter, prospective observational study being launched in early 2007. This study is built on the design template of the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)–sponsored adult LABS study and has an overarching goal of comparing outcomes between adolescents and adults undergoing surgery, to begin to understand if unique benefits (or risks) exist when surgery is applied before the deleterious effects of long-term obesity are manifest.

In summary, Freedman et al have provided valuable evidence that use of the >99th percentile of BMI for age may be a medically rational threshold at which to define clinically severe, extreme pediatric obesity. Use of an evidence-based definition may improve our efforts to define the causes and consequences of this most extreme form of obesity, as well as to identify optimum treatment strategies and improve access to treatments for this subpopulation of youth at considerable medical and psychosocial risk.

**REFERENCES**


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The Vascular Contribution to Necrotizing Enterocolitis

Feeding, gastrointestinal bacterial colonization, gut motility, cytokines, vascular factors, and gut blood flow all have been implicated in the etiology and pathogenesis of necrotizing enterocolitis (NEC). Further, neonatal complications such as respiratory distress syndrome, respiratory disease, apnea, infection, hypotension, asphyxia, and patent ductus arteriosus increase the risk for development of NEC. Infants with NEC generally present with feeding intolerance, abdominal distension, bilious gastric residuals or vomiting, or bloody stools. Nonspecific presenting signs and symptoms include apnea, bradycardia, desaturation episodes, cyanosis, lethargy, temperature instability, poor peripheral perfusion, hypoglycemia, jaundice, and shock. Diagnosis of NEC is confirmed by the presence of the classic finding of pneumatosis intestinalis on abdominal x-ray. Associated laboratory abnormalities can include metabolic acidosis, leukopenia, thrombocytopenia, anemia, and clotting abnormalities of disseminated intravascular coagulation. Treatment consists of supportive medical care and surgical care.

Vascular flow to the gastrointestinal tract plays a significant role in gut development, integrity, and function. Blood flow is balanced between needs of the bowel and needs for blood flow to other organs. Control of blood flow, specifically the balance between vasoconstriction and vasodilatation plays a key role in maintaining gut integrity. Nowicki et al should be commented for their report, published in this issue of the Journal, in delineating the role of endothelial nitric oxide synthase (eNOS) in NEC. This is the first study to demonstrate reduced eNOS function in human tissue samples from patients with NEC.

Nowicki et al show that eNOS activity and NO production are decreased in the arterioles of infants with NEC. The lack of function of a vasodilatory regulator certainly adds to the description of lack of appropriate flow to the bowel in the etiology and pathogenesis of NEC. Increased endothelin results in significant vasoconstriction that is not able to be reversed in the arterioles of the small intestine with NEC. Nowicki et al previously provided evidence that intestinal tissue from patients with NEC had higher concentrations of endothelin. The balance between vasoconstriction and vasodilation is critical in assuring bowel homeostasis.

Perhaps modulation of eNOS function may be one modality to target increasing blood flow in ischemic bowel. Possibly, infants who develop NEC have genetic susceptibility factors that render them vulnerable to NEC,