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Surgical approach to adolescent obesity

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Pediatric obesity has reached near epidemic proportions in the United States. Parallel to this, adult bariatric surgery has been proven to be safe and effective. It is only natural to consider bariatric surgical interventions for severely obese adolescents. Currently, there is very little written specifically about the appropriateness of bariatric surgical procedures for adolescents with severe obesity, and even less about the program constituents for delivery of surgical options for weight loss in children. This article focuses primarily on a discussion of (1) the indications for bariatric surgical intervention in adolescence, (2) the team approach to delivery of bariatric services in this age group, (3) principles of perioperative and postoperative patient management, (4) the surgical procedures available for weight loss, and (5) the results of bariatric surgical intervention in adolescence.

Phylogenetically, people are endowed with the biochemical machinery and cellular mechanisms to protect themselves during times of nutritional deprivation. These protective mechanisms, honed over time, take the form of efficient and highly regulated metabolic processes, whereby excess energy intake is stored, predominantly as fat, for use during times when nutrients are not abundant. Until

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now, this biologic plan has served people well. Currently, however, societies have developed and refined the food production industry such that in most industrialized countries, no supply shortage exists. In fact, the same technologic revolution that has all but eliminated food shortage also has resulted in the mechanical automation of society to a greater extent than ever existed before. So for now, energy intake is not limited, less physical activity is necessary for daily living, and over the past several decades, a worldwide epidemic of obesity has developed [1].

It also has been learned that the basic physiology of weight regulation may be hampering attempts to combat the growing obesity problem. There is increasing evidence for the existence of a central barostat controlling the body's innate response to weight loss—to activate anabolic pathways and deactivate catabolic pathways. Data suggest that the barostat is designed to oppose the maintenance of a body weight that is different than the usual body weight, analogous to the way a thermostat controls room temperature [2–4]. This homeostatic mechanism is inherently more vigorous at defending against weight loss than preventing weight gain [5]. In this context, the ever-increasing obesity problem may represent an expected biologic outcome given people's genetic make-up and the environment. What was entirely unexpected by the world health care community, however, was the rapidity with which the population has fattened over the last several decades, and the rate at which children have begun to manifest health consequences of the obesity epidemic.

Currently, cross-sectional national survey data show that nearly 16% of adolescents in the United States are obese, an increase of 39% over the last 20 years [6]. Perhaps even more alarming are longitudinal data that suggest that the prevalence of adolescent obesity may be as high as 39% in some regions of the United States [7]. As the epidemic of childhood and adolescent obesity progresses unchecked, physicians and families increasingly are considering more extreme measures to treat severe obesity and the obesity-related comorbid conditions seen in adolescence. Comprehensive behavioral treatments for obesity in childhood (8 to 12 years of age) have been found to be superior to nutrition education alone in achieving short-term weight loss [8] and long-term maintenance of treatment efficacy [9] for less extreme levels of obesity. The literature concerning behavioral treatment of adolescents is considerably less developed or remarkable [10], and adult behavioral treatment outcomes have been unimpressive and characterized by weight regain [11]. The evidence clearly suggests that behavioral weight management may have longer-lasting effects in children compared with adolescents or adults and may be less effective for severe obesity [12].

Weight loss after bariatric surgery results in improvement, if not resolution, of most obesity-related comorbidities in adults [13–15]. Preliminary results suggest that this is also true for adolescents [16–19]. There are several factors, however, that must be considered when contemplating bariatric surgery for severe obesity in adolescents [20]. In addition, knowledge gaps exist about long-term effectiveness and potential adverse consequences of bariatric procedures. These potentially adverse effects, which are related chiefly to decreased absorption of

macro- and micronutrients, suggest that substantial deliberation is required when considering surgical weight management options in younger age groups.

Defining obesity

Dramatic changes in adiposity, height, and weight occur during the first 20 years of life. Accurate assessment of body fat content requires underwater weighing, dual energy x-ray absorptiometry (DEXA), or bioelectric impedance. At extremes of weight, the more convenient DEXA and bioelectric impedance methods can be of limited accuracy. Conversely, measurement of body mass index (BMI, calculated as weight [kg]/height [cm]/height [cm] × 10,000 and expressed in kg/m^2]) is a clinically useful estimate of adiposity when adjusted for age and sex and expressed as a standardized percentile within the reference population [21–24]. Data collected through the National Health Evaluation Survey in the 1960s and in subsequent decades through the National Health and Nutrition Evaluation Surveys (NHANES) predate the rapid increase in obesity within the US population and were used to construct the 2000 reference charts for BMI [25,26]. The 85th and 95th percentiles of BMI for age from these reference charts have been used to define overweight and obesity [27]. The 95th percentile of BMI for age also corresponds to a BMI in young adults (18 year olds) of 30 kg/m^2, which is the widely accepted definition of obesity for adults.

Although the 95th percentile definition of obesity is helpful when considering treatment options for most children, this definition has little practical use when contemplating a level of obesity that might warrant surgical therapy. For the very severe level of obesity that prompts consideration of obesity surgery in adults (eg, BMI of 40 kg/m^2), there are no reliable population-based data by which accurate percentile boundaries can be calculated for those younger than 18 years of age. This is because youth with BMI values in the range of greater than or equal to 40 kg/m^2 are represented very poorly in NHANES [28]. Hence, the maximum BMI that can be plotted on standard growth charts is 37 kg/m^2. In the absence of an epidemiologically accurate method to define very severe obesity for each age during adolescence, a decision to arbitrarily use BMI $\geq$ 40 kg/m^2 to consider surgical intervention [18] can be considered a conservative threshold. It is important to recognize in this context that some muscular individuals with BMI values in this range may not be severely obese, and, therefore, considerable caution must be used when considering indications for operation.

Rationale for surgical treatment of adolescent obesity

Adolescent obesity is a multi-faceted disease with numerous recognized antecedents [29–43] and serious medical consequences [44–48]. The increased risk for adult mortality as a late consequence of adolescent obesity also has been previously documented [47,49]. In addition, adolescent obesity has formidable

Box 1. Selected consequences and comorbidities of adolescent obesity

Psychosocial
- Poor self-esteem [53,54]
- Depression [45]
- Eating disorders [55]
- Discrimination and prejudice [56]
- Quality of life [56,57]
- Sexual abuse [58]

Neurological
- Pseudotumor cerebri [59–61]

Pulmonary
- Sleep apnea [62–69]
- Asthma and exercise tolerance [70]

Cardiovascular
- Dyslipidemia [71,72]
- Hypertension [73–76]
- Coagulopathy [77]
- Chronic inflammation [78]
- Endothelial dysfunction [79]

Gastrointestinal
- Gallstones [80]
- Nonalcoholic fatty liver disease [81–84]

Renal
- Glomerulosclerosis [85]

Endocrine
- Type 2 diabetes mellitus [86–89]
- Diabetic precursors/insulin resistance [88,90]
- Precocious puberty [52,91]
- Polycystic ovary syndrome [92]
- Hypogonadism (boys) [93]

Musculoskeletal
- Slipped capital femoral epiphysis [94]
- Blount's disease [45,95]
- Forearm fractures [96]
- Flat feet [97]

Excess health care costs [52] Pediatric obesity reviews [44,48,98–102].

social and economic adverse consequences [50–52]. A detailed discussion of the myriad physical, mental, and socioeconomic consequences of adolescent obesity is beyond the scope of this text, but can be found in the citations included in Box 1.

The most compelling reasons for performing bariatric procedures in adolescence are to reduce or resolve obesity-related comorbidities, prevent others from developing, and reduce the risk of early mortality that has been documented in the obese [49]. Arguably, some of the most severe consequences of obesity seen in adolescents include type 2 diabetes mellitus, obstructive sleep apnea syndrome (OSAS), and pseudotumor cerebri. These diseases have significant, deleterious effects on the lives and well-being of young people. Because these conditions are ameliorated or eliminated in adults after bariatric surgery (diabetes [103–106], OSAS [107,108], pseudotumor cerebri [109,110]), perhaps they should be viewed as special indications for surgery in adolescents also.

Patient selection criteria

Indications for bariatric surgery in adolescence should be considered in the context of the natural history of severe obesity in young people and the outcomes of bariatric surgery documented in adults. All-cause mortality for adults begins to rise considerably above a BMI of 35 kg/m^2 [111]. National Institutes of Health (NIH) guidelines suggest that it is reasonable to consider weight loss surgery for individuals as young as 18 years old with a BMI of at least 35 kg/m^2 in the presence of severe obesity-related comorbidities or at a BMI of at least 40 kg/m^2 even in the absence of defined comorbidities [112]. There is no empiric evidence on which to base guidelines for adolescent bariatric surgery. More restrictive guidelines for operation in adolescents are justified, because the long-term risks and durability of the surgery in this age group are unknown [113]. Also, there are virtually no data regarding the optimal time to perform obesity surgery to produce the optimum results and the least complications . For example, there is justified concern that there may be unforeseen consequences for bone mineralization, and that compliance with dietary and exercise recommendations may be poorer in adolescents than in adults [114]. These factors may lead to increased metabolic complications after surgery (Towbin, et al, unpublished data, 2004). Conversely, there is also theoretic concern that for some conditions, such as type 2 diabetes, nonalcoholic fatty liver disease, atherosclerotic coronary vascular disease, and myocardial hypertrophy, a window of opportunity may exist in extremely obese youth to intervene and prevent progression of potentially irreversible anatomic changes.

There have been some individuals with BMI of at least 40 kg/m^2 who have benefited from behavioral treatment of obesity, and many adolescents with BMI of at least 40 kg/m^2 do not have serious comorbid conditions that might prompt surgical consideration. That stated, for adolescents with very severe obesity (BMI of at least 40 kg/m^2) who have been unsuccessful with conventional weight

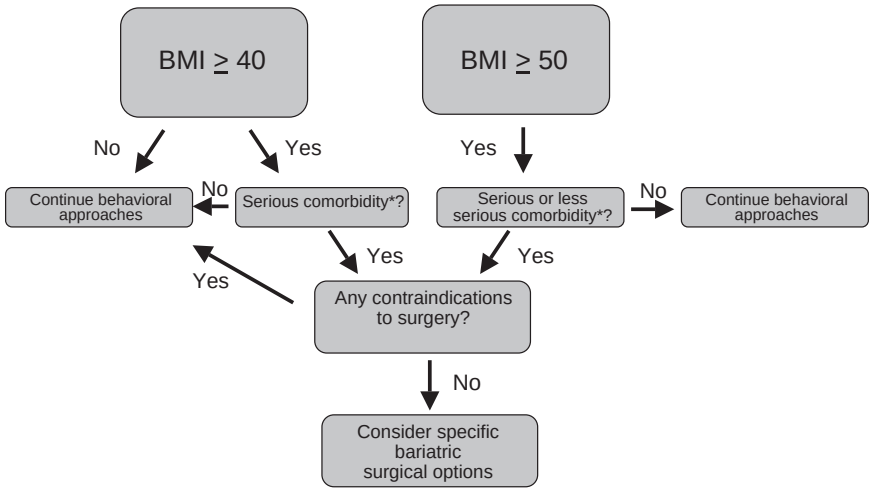

Fig. 1. Proposed algorithm for severely obese adolescents who largely have completed linear growth and in whom previous organized attempts at weight management have been unsuccessful. Severe and less severe comorbidities defined in Box 2.

Box 2. Obesity-related conditions that may be improved with bariatric surgery

Serious comorbidities

Type 2 diabetes mellitus
Obstructive sleep apnea
Pseudotumor cerebri

Less serious comorbidities

Hypertension
Dyslipidemias
Nonalcoholic steatohepatitis
Venous stasis disease
Significant impairment in activities of daily living
Intertriginous soft tissue infections
Stress urinary incontinence
Gastroesophageal reflux disease
Weight-related arthropathies which impair physical activity
Obesity-related psychosocial distress

management efforts, the presence of a serious comorbid condition should prompt consideration for bariatric surgery (Fig. 1, Box 2). Bariatric surgery also should be considered an option for adolescents with a BMI of at least 50 kg/m^2 with less serious obesity-related comorbid conditions, if the conditions can be corrected predictably with surgical weight loss, and if the competing risks of persistence or progression of the comorbidity are thought to be greater than the risks of bariatric surgery. Adopting a BMI-based algorithm as a general guideline for considering bariatric surgery is undertaken with the understanding that a morbidly obese adolescent with an advanced, severe, and incontrovertibly weight-related comorbidity also should be considered for weight loss surgery without strictest regard to level of BMI. Finally, there are some conditions or factors that should represent absolute contraindications for bariatric surgery in adolescence. These include:

- Presence of a medically correctable cause of obesity
- Patient or family is unable or unwilling to participate in long-term follow-up
- Absence of decisional capacity on the part of the patient
- Existence of a medical, psychiatric, or cognitive condition that may impair the ability of patient to assent to surgery or adhere to postoperative dietary and medication regimen
- Existence of substance abuse in preceding year
- Current lactation, pregnancy, or plans for pregnancy in upcoming 2 years

One inherent difficulty in the decision-making regarding eligibility for bariatric surgery is the inability to precisely define the concept of failure of conventional weight management attempts. Most conventional weight management programs are not designed for adolescents. In addition, because the number of pediatric weight management programs is limited, this approach may be unavailable to most individuals in need of services. It also has been suggested that patients must fail physician-supervised attempts at weight management before surgical consideration. This definition of failure may be unrealistic also, because physicians often lack the expertise and opportunity to provide meaningful weight loss interventions for adolescents with established severe obesity. Therefore, the decision to proceed with more intensive therapies like surgery must be made on an individual basis, with active involvement of the primary care provider and family. There must be agreement that all reasonable and available efforts at nonsurgical weight management have been pursued aggressively for an adequate period of time and have been unsuccessful.

Bariatric programs for adolescents

Adolescents who are severely obese can present unique challenges to providers of bariatric surgical care. Thus, adolescents who are considering bariatric surgery should be referred to centers with a multi-disciplinary bariatric team

capable of providing a comprehensive initial evaluation, modern surgical intervention, and postoperative medical, psychological, and surgical surveillance tailored for the younger age group. Guidelines have been established by the NIH [115], the American Society for Bariatric Surgery (www.asbs.org), and the American College of Surgeons [116], suggesting that teams providing bariatric care should include specialists with expertise in obesity evaluation and management, psychology, nutrition, physical activity, and bariatric surgery. Depending on the individual needs of the adolescent patient, additional pediatric subspecialty expertise in adolescent medicine, endocrinology, pulmonology, gastroenterology, cardiology, and orthopedics should be readily available. Medical evaluation must focus on screening for the rare existence of endogenous obesity that may be amenable to medical treatment and characterization of apparent and potentially occult comorbid conditions. At Cincinnati Children's Hospital, the patient review process is similar to that used in multi-disciplinary oncology and transplant programs [18]. This review by a panel of experts from different disciplines results in a treatment plan for individual patients, including consideration for operative intervention.

Factors influencing timing of surgery

The timing for surgical treatment for adolescents is controversial and should consider the health needs of the patient. There are certain physiological factors that need to be considered in planning an essentially elective operation. Physiologic maturation is generally complete by sexual maturation (Tanner) stage 3 or 4 [117]. Skeletal maturation (adult stature) is normally attained by the age of 13 to 14 in girls [118] and 15 to 16 in boys [119]. Overweight children generally experience accelerated onset of puberty. As a result, they are likely to be taller and have advanced bone age compared with age matched nonoverweight children. If there is uncertainty about whether adult stature has been attained, skeletal maturation (bone age) can be assessed objectively with a radiograph of the hand and wrist [120]. If an individual has attained at least 95% of adult stature [121] by this examination, there should be little concern that a bariatric procedure would impair completion of linear growth significantly.

The negative impact of severe obesity on psychological health [122], and on weight-related quality of life [123,124] has been documented. Others have begun to characterize psychosocial outcomes of bariatric surgery for adults [125]. Similar information specific for severely obese adolescents, however, is quite limited. Further, given that bariatric surgery is a relatively new intervention for adolescents, information describing the psychosocial outcomes of this intervention is nonexistent. Future clinical research will be needed to determine predictors of surgical success and to help identify the unique psychosocial needs of severely obese adolescents.

A psychologist with pediatric expertise is an important member of the adolescent bariatric team. The authors' recommendations are for candidates for bar-

iatric surgery to undergo a comprehensive psychological evaluation. Goals of this evaluation include:

- Determining the level of emotional maturity and cognitive development, primarily to judge the extent to which the adolescent is capable of participating in the decision to proceed with the intervention
- Identifying past/present psychiatric, emotional, behavioral, or eating disorders
- Defining potential supports and barriers to regimen adherence and family readiness for surgery and the required lifestyle changes (particularly if at least one parent is obese)
- Assessing reasoning styles and problem solving ability
- Assessing whether there are reasonable outcome expectations
- Assessing family unit stability and identifying psychological stressors or conflicts within the family
- Determining whether the adolescent is motivated autonomously to consider bariatric surgery or whether any element of coercion is present
- Assessing weight-related quality-of-life status

Although this evaluation may help determine candidacy for surgery, its primary purpose is to aid in the development of treatment plan that matches the needs and meets the predictable challenges of each adolescent and his or her family.

Surgical options and preoperative education

The NIH Bariatric Consensus Development Conference in 1991 laid the groundwork for a dramatic increase in adult bariatric surgery volume that has been realized primarily over the past 5 years. This conference concluded that (in 1991) roux-en Y gastric bypass (RYGBP) and vertical banded gastroplasty were reasonable surgical options for morbidly obese adults but that insufficient data existed to make recommendations for patients younger than 18 years of age. Outcome data remain very limited for the adolescent age group, and no bariatric operation has been studied in a controlled fashion in adolescents. Of the many procedures that have been advocated for weight loss, the operations that have been used most can be classified as either purely restrictive or restrictive and malabsorptive.

There are reasons to postulate that adolescents may be served better by purely restrictive options such as the adjustable gastric band, as illustrated in Fig. 2 [19]. Although nutritional deficiencies are not as likely with purely restrictive operations as with malabsorptive options, restrictive operations still impair overall nutrient intake significantly, which also can lead to impaired intake of important vitamins and minerals if not adequately supplemented. Nonetheless, with surgical procedures that do not transect the gastrointestinal (GI) tract, there is less operative risk and a reportedly lower mortality risk compared with RYGBP [126]. The adjustable gastric band is not approved by the US Food and Drug

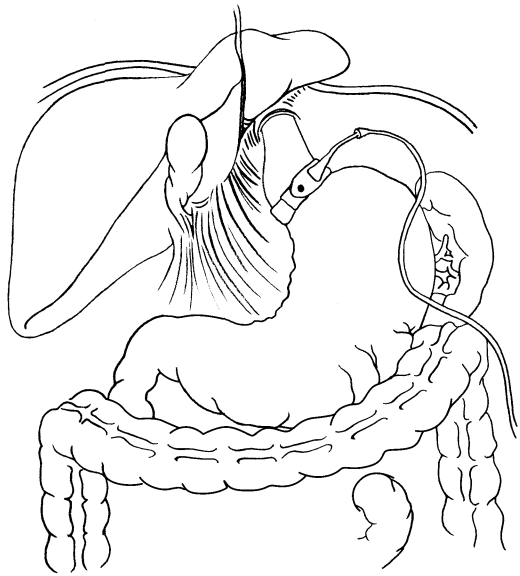

Fig. 2. Laparoscopically placed adjustable gastric band.

Administration (FDA) for use in adolescents, but it may be an option in the near future. In adults, use of the adjustable gastric band has resulted in amelioration of most serious comorbidities of obesity [103,104,123,127–131]. The adjustable gastric band has not resulted in the rapid and dramatic weight loss seen with the combined restrictive and malabsorptive or purely malabsorptive options discussed subsequently. Long-term (15 to 20 years) outcomes of adjustable gastric banding are lacking.

Like vertical banded gastroplasty, RYGBP consists of a restrictive stomach pouch but adds a restrictive gastrojejunal anastomosis and a malabsorptive component (the roux limb bypass of stomach and duodenum) (Fig. 3). If sweets are consumed, this operation can result in an additional negative reinforcement in some patients known as dumping syndrome, which results in diarrhea, flushing, and anxiety. The operation has been used successfully in adults for over 40 years and is the most commonly performed procedure for obesity in the United States [132,133]. RYGBP can be performed with standard open laparotomy or minimally invasively with laparoscopy (Fig. 4). Most adults lose 65% to 80% of excess weight initially, and 50% excess weight loss over 14 years has been shown for at least one cohort [134]. There are postoperative nutritional concerns with this procedure that will be addressed subsequently.

Finally, the partial biliopancreatic bypass with duodenal switch is a primarily malabsorptive procedure that results in excellent weight loss (75% of excess weight) for adults with the highest classes of obesity (generally BMI greater than 60 kg/m^2), but at the expense of higher risks of operative complications and postoperative malnutrition. For this reason, this operation is not performed

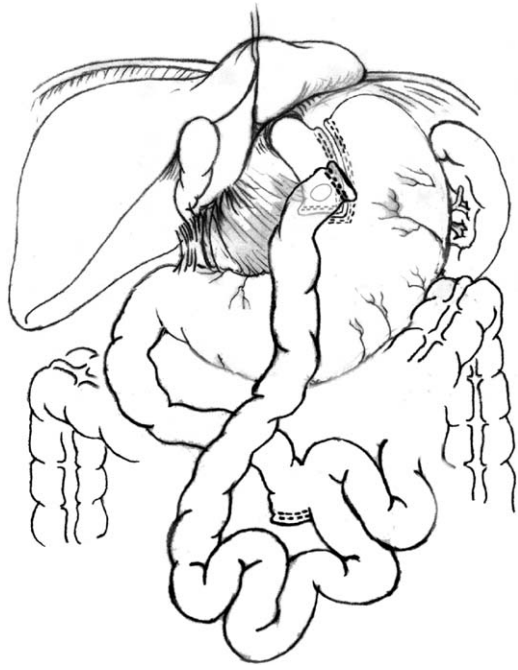

Fig. 3. Laparoscopic roux en Y gastric bypass.

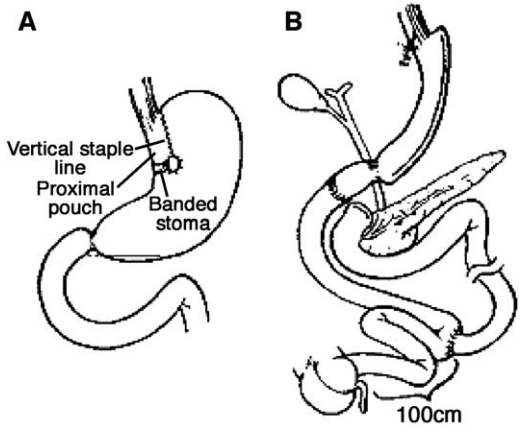

Fig. 4. Bariatric operations used less frequently today. (*A*) Vertical banded gastroplasty. (*B*) Biliopancreatic diversion with duodenal switch.

commonly or recommended for most adolescents. A broader discussion of these procedures is beyond the scope of this article, but excellent reviews can be found elsewhere [14,135–137].

In summary, the primary goals of bariatric intervention in adolescence are weight loss efficacy and long-term safety. For these reasons, the RYGBP or the adjustable gastric band may be considered appropriate choices for adolescents. Currently, there is insufficient information available to determine more specifically which option is most beneficial. Carefully designed trials will be needed to evaluate the relative efficacy of these interventions in this age group.

Preoperative education

Intensive patient and family preoperative education are required before surgery. It is important that this process is organized and not rushed, because there is a great deal of information for patients and parents to comprehend about the intervention and postoperative requirements. This education necessarily includes detailed information about the surgical procedure recommended, nursing care, dietary strategies, physical activity, and behavioral approaches to support adherence to the postoperative regimen. Also, many patients benefit from discussion with others who have undergone surgery, and often more questions arise as a result. In the week before operation, a final outpatient visit for anesthesiology consultation, final informed permission (consent) discussion, and final review of the postoperative regimen is scheduled. At the conclusion of this visit at Cincinnati Children's Hospital Medical Center, the patient takes a written test that is scored and reviewed with the patient as further documentation of his or her level of understanding of the procedure and its known and potential consequences. Bariatric surgery should not be performed if there is any concern that the principles of informed consent have not been satisfied completely.

Preoperative studies that that the authors routinely perform include a chemistry and liver profile, lipid profile, cell blood count, hemoglobin A1C, fasting blood glucose, thyroid stimulating hormone, and pregnancy test for females. Because unrecognized sleep disorders are relatively prevalent in the severely obese, a complete sleep history including history of snoring, irregular breathing, and increased daytime somnolence is sought. A history suggestive of sleep apnea should prompt formal polysomnography. Patients who have not been diagnosed with diabetes undergo a 2-hour glucose tolerance test. Both of these tests may be required when patients are being evaluated for eligibility for surgery, or may be required separately for third party coverage of the intervention.

Complications of bariatric surgery

Complications can be categorized as intraoperative, early postoperative, and late postoperative (Box 3). Intraoperative complications during performance of

Box 3. Complications of laparoscopic roux-en Y gastric bypass surgery [126]

Early

Gastrointestinal leakage, 0% to 4.4%
Bleeding, 0.6% to 3.4%
Wound infection, 0% to 8.7%
Pulmonary complication, 0% to 5.8%
Deep vein thrombosis/pulmonary embolus, 0% to 3.3%

Late

Stomal stenosis, 1.6% to 22%
Bowel obstruction, 0.6% to 20%
Incisional hernia, 0% to 1.8%
Internal hernia, 0% to 3.3%
Marginal ulcer, 0% to 10%
Gallstone formation, 0% to 2%
Iron deficiency, 0% to 10%
Mortality, 0% to 3.3%

RYGBP or adjustable gastric band surgery primarily involve unintentional perforation of bowel (esophagus, stomach, small intestine, or colon) which may or may not be recognized at the time of the injury. Additional intraoperative complications include hemorrhage, particularly from the abdominal wall, mesentery, liver, spleen, or retroperitoneal structures; and anesthetic complications related to management of a difficult airway. If the procedure is being performed laparoscopically, any one of these complications might require conversion to an open laparotomy to manage the problem. Postoperatively, potential early complications include respiratory insufficiency, pneumonia, deep vein thrombosis with or without pulmonary embolus, wound infection, GI hemorrhage [138], and gastrojejunal anastomotic obstruction caused by edema. In addition, acute dilation of the gastric remnant with rupture can occur because of ileus or obstruction of the jejunojejunal anastomosis. Leakage of intestinal contents from staple lines (either the gastric pouch or on the side of the remnant stomach) or from either gastrojejunal or jejunojejunal anastomosis can occur, leading to peritonitis [139]. In adults, a mortality risk of approximately 0.5% has been reported. Late complications including dehydration, specific nutritional deficiencies, gastrojejunal stomal stenosis, marginal ulceration beyond the gastrojejunal anastomosis, dumping syndrome, and internal hernias also have been recognized.

The primary care provider may be the first to detect symptoms of late complications after adolescent gastric bypass, and early plans for appropriate

studies or interventions can be made. Anastomotic stenosis can lead to nutritional deficiencies (such as beriberi or essential fatty acid deficiency) and severe dehydration [139]. Recurrent emesis may or may not be present, as the patient may learn to adapt his or her diet to avoid items that will not pass through the anastomosis. The dehydration may not be appreciated easily on physical examination. Highly concentrated urine (specific gravity of at least 1.030), hypokalemia, prerenal azotemia, and fractional excretion of sodium less than 0.5% can be present. Upper GI contrast studies may show esophageal dilation or reflux from the pouch, or impaction of nonemptying solids within the pouch. Upper endoscopy is the most sensitive test for stomal stenosis, and endoscopically directed balloon dilation of the stenosis is usually successful.

Upper GI bleeding or pain can occur because of marginal ulceration of the jejunal mucosa just distal to the anastomosis [140]. This problem is treated with acid suppression and elimination of the possible precipitating medications, such as nonsteroidal anti-inflammatory agents. It is important to remember that acid production in the gastric remnant (and risk of duodenal ulcer) still exists after RYGBP. If upper GI bleeding is suspected and not found by upper endoscopy, a duodenal source should be suspected, and testing for *Helicobacter pylori* performed to determine if the patient might benefit from antibiotic therapy. Regardless, empiric acid reduction therapy is indicated if gastroduodenal ulceration is suspected.

Sudden-onset ability to eat large amounts of food or persistent marginal ulceration despite medical therapy should prompt consideration of gastric staple line breakdown and gastrogastric fistula formation. An upper GI contrast study should demonstrate this problem, and surgical revision is the treatment.

Primary care providers should be aware of the signs of internal herniation after gastric bypass. Crampy, intermittent, periumbilical, or upper abdominal pain usually associated with nausea and retching can result from intestinal obstruction from internal hernias. The three common anatomic positions where hernias can occur are through the defect in the transverse mesocolon (if a retrocolic roux limb was used), between the roux limb mesentery and the mesocolon, and through a defect in the mesentery where the jejunojejunostomy was created. These hernias can be notoriously difficult to diagnose, even with GI contrast radiography and CT. Because the consequences of internal hernias are intestinal incarceration, strangulation, and necrosis, early referral for surgical evaluation is mandatory. Exploratory laparoscopy or laparotomy can make the diagnosis definitively and correct the defect.

Nutritional deficiencies including vitamin B_{12}, calcium, and iron have been described following bariatric surgery in adults [141–149]. Vitamin B_{12} absorption is impaired because of the absence of gastric pouch acidity, which is necessary for release of vitamin B_{12} bound to food. Replacement therapy by the parenteral (intramuscular), sublingual, or intranasal routes have been used. Calcium and iron are absorbed maximally in the duodenum that is bypassed completely after RYGBP. This fact can have important implications for bone health and erythropoiesis. For example, normally bone density increases as a function of prog-

ressive bone mineralization up to the middle of the third decade of life. Proper bone mineralization is related directly to adequate intake of vitamin D and calcium, and the requirement for each of these micronutrients is higher in adolescence than any other time in life. Thus, for the gastric bypass patient, in whom milk often is not tolerated well, and neither vitamin D nor calcium absorption is normal, there is considerable concern for the possibility of negatively impacting bone mineral accretion, with a resulting increased risk of osteoporosis or brittle fracture long-term [150]. It is essential to ensure that vitamin D and calcium are supplemented adequately. Objectively monitoring bone mineral density postoperatively may be beneficial. Similar to the approach in other chronic conditions [151], behavioral strategies may improve compliance with postoperative micronutrient intake that should influence overall health positively after adolescent bariatric surgery.

Female adolescent bariatric patients are soon to be in the childbearing years of their lives. Folate is important for prevention of fetal malformations including neural tube defects and perinatal complications such as low birth weight, prematurity, and placental abruption and infarction [152]. Deficiency of this B vitamin also can occur after RYGBP. Thus, patients should take folate during the periconceptional period and through the first trimester to avoid embryopathy. Primary care providers caring for these individuals must stress the importance of daily micronutrient supplement intake and should consider monitoring of serum vitamin levels when uncertainty about compliance exists.

Hair loss (telogen effluvium) has been reported in up to one third of patients following surgical weight loss, and numerous possible causes have been cited including protein insufficiency, inadequate intake of iron or zinc, or simply as a result of the sudden, severe emotional and physiologic stress of the surgical intervention and recovery process [153]. Normally, 10% to 15% of scalp hair follicles are resting at any one time. In some patients, the surgical stress causes more follicles (up to 80%) to enter the resting phase (telogen) that may last for 2 to 3 months. Maximizing protein intake and supplementing iron and zinc intake (200 mg zinc sulfate three times daily [154]) has been recommended for this emotionally disturbing consequence of bariatric surgery, although management approaches have not been studied rigorously. If hair loss occurs as a consequence of the stress of the operation, there is little more that can be done to reverse the process, and in fact, the loss of hair actually signals the regrowth of replacement hair within the resting follicles.

Patient management considerations after surgery

Postoperative follow-up after bariatric surgery in adolescence is intensive: weekly for 1 month, then monthly until 6 months, then quarterly until first 12 months, then biannually until 24 months, and yearly thereafter. RYGBP essentially results in surgically enforced very low-calorie, low-carbohydrate dietary intake, thus requiring attention to an adequate daily protein intake (0.5 to 1.0 g/kg

per day based on actual weight) to reduce lean mass loss during rapid weight loss. Initially, a protein-supplemented, liquid-to-edentulous diet is prescribed to avoid intraluminal obstruction as the gastrojejunostomy heals. Dietary advancement after the first month is a slow process of introducing new items of gradually increasing complexity, toward the goal of a well-balanced, small portion (approximately 1 cup per meal) diet.

Because of an increased risk of gallstone formation and peptic ulcer development after gastric bypass, ursodiol and ranitidine are prescribed. Nonsteroidal anti-inflammatory medications that are nonselective cyclooxygenase inhibitors (eg, ibuprofen and indomethacin) should be avoided lifelong to reduce the risk of iatrogenic intestinal ulceration and bleeding. Chewable multivitamins and a calcium supplement are prescribed; menstruating females also should take an iron supplement. Strong consideration also should be given to additional supplementation of B-complex vitamins (particularly thiamine) to further reduce the risk of beriberi, which has been documented in adults [141,155] and adolescents after gastric bypass surgery [156]. Finally, there are five basic rules that are stressed with each postoperative patient encounter. These are:

- Eat lean protein first with every meal, toward goal of 0.5 to 1 g/kg per day.
- Drink 64 to 96 oz of sugar-free liquids daily.
- No snacking between meals.
- Exercise 30 to 60 minutes per day.
- Always remember to take vitamins and minerals.

Serum chemistries, complete blood count, urine specific gravity, prothrombin time (evidence of vitamin K adequacy), and representative B complex vitamin levels (eg., B1, folate) are obtained postoperatively. The rationale for this practice is that poor compliance with postoperative supplements, which has been documented in adolescents after gastric bypass [114], has adverse effects on fat and water soluble vitamin levels after bypass [149]. Body composition is assessed with bioelectrical impedance or DEXA (for patients weighing less than 300 pounds) at regular intervals during rapid weight loss (3 and 6 months) and then again once weight essentially has stabilized (12 months). DEXA not only allows for the measurement of rate and relative amounts of fat and lean body mass loss but also provides a quantitative assessment of bone mineral density and content. This information can be used to modify dietary plans intended to preserve lean body mass during the period of rapid weight loss.

Outcomes of adolescent bariatric surgery

Bariatric surgery in adolescence has not been evaluated or compared with nonoperative approaches in a prospective manner. The limited experience accrued in several small series, however, suggests that gastric bypass [16–18,

114,157–160] and adjustable gastric banding [19] can be performed safely and effectively in adolescents.

Whereas most of the literature has reported outcomes of the open RYGBP, laparoscopic RYGBP has been used successfully in adolescents with few complications [18,20,158]. Stanford et al reported an excess weight loss over 80% in three of four patients who were followed at least 20 months following laparoscopic RYGBP surgery [158].

Excess weight loss following gastric bypass has been satisfactory in most series. Strauss reported 9 of 10 patients who lost over 59% of their initial excess weight [17]. Similarly, Sugerman reported a 56% excess weight loss in 20 adolescents (ages 12 to 17 years old) who were surveyed over 10 years after gastric bypass surgery [16].

Despite consideration of the RYGBP as the gold standard for the achievement of sustained weight loss [134], adjustable gastric banding may be associated with fewer risks. In the United States, FDA approval of a specific adjustable gastric band device (Lap Band, Inamed Health, Santa Barbara, California) in June of 2001 was confined to the adult population; additional approval for use in adolescents will be required. The appeal of adjustable gastric banding for adolescents lies in the reversibility, low incidence of morbidity and mortality, and potential avoidance of severe nutritional risks associated with malabsorptive procedures. Dolan et al reported on 17 adolescents (ages 12 to 19 years old) who underwent laparoscopic adjustable gastric banding for obesity (mean preoperative BMI of 44.7 kg/m^2) with an average of 2 years follow-up. These patients achieved 59.3% excess weight loss and most (76.5%) lost at least 50% of their excess weight (postoperative BMI of 30.2 kg/m^2) [19]. Many patients in this study experienced marked improvement of obesity-related comorbidities. Long-term follow-up is necessary to determine whether the elimination of comorbidities and maintenance of weight loss are sustained and whether complications related to the gastric banding approach are acceptable.

There has been no reported procedure-related mortality in adolescents undergoing RYGBP or adjustable gastric banding. Early complications of RYGBP in adolescents have included pulmonary embolism, wound infection, stomal stenoses, and marginal ulcers. Late complications have included small bowel obstructions; incisional hernias; symptomatic cholelithiasis; protein, calorie, vitamin, and micronutrient deficiencies; and late weight regain (10% to 15% incidence) [16,157]. The predictability of such complications parallels adult series, necessitating lifelong follow-up.

In 2001, the first children's hospital-based comprehensive weight management program including bariatric surgery was developed at Cincinnati Children's Hospital Medical Center. As of Spring of 2004, 32 adolescents have undergone RYGBP. The average age was 17 for girls and 16 for boys, while the mean BMI was 56. The youngest patient was a 14-year-old girl with type 2 diabetes mellitus. All patients had comorbidities of obesity. Twelve of these patients are now greater than 6 months following operation. This cohort had an average preoperative BMI of 57 (BMI range 44 to 85), and after 6 months

patients had lost on average 31% of preoperative BMI (range 22% to 43%). Anecdotally, resolution of comorbidities after surgical weight loss has been gratifying for patients; a detailed follow-up study of this cohort has not been completed.

Summary

The obesity epidemic has resulted in the premature onset of traditionally adult diseases in children and adolescents. Bariatric surgery can be used to achieve significant weight loss and resolution of major and minor comorbidities of obesity. It is not known, however, whether weight loss or comorbidity resolution after adolescent bariatric surgery is sustainable over the lifetime, given the powerful biologic mechanisms that are thought to play an important role in development of severe obesity in these patients. There are also concerns about whether bariatric procedures may have unanticipated adverse nutritional consequences when applied in adolescence. Principles of adolescent medicine and evidence from adult bariatric surgical studies can be used to rationally guide the application of bariatric procedures to a group of young patients wither serious medical and psychological comorbidities of severe obesity. Given the immediacy of some of the medical and psychosocial complications, impaired quality of life, and the added health care costs of adolescent obesity, adolescent bariatric surgery programs should be developed to meet these needs. Adolescent bariatric surgery programs should have expertise that enables them to assess and meet the unique medical, cognitive, physiological, and psychosocial needs of the adolescent. Indications for operation during adolescence should be conservative until appropriately designed clinical studies have demonstrated the safety and efficacy of these techniques applied to adolescents.

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References

[1] Friedman JM. A war on obesity, not the obese. Science 2003;299(5608):856–8.
[2] Rosenbaum M, Leibel RL, Hirsch J. Obesity. N Engl J Med 1997;337(6):396–407.
[3] Verdery RB, Walford RL. Changes in plasma lipids and lipoproteins in humans during a 2-year period of dietary restriction in Biosphere 2. Arch Intern Med 1998;158(8):900–6.
[4] Leibel RL, Rosenbaum M, Hirsch J. Changes in energy expenditure resulting from altered body weight. N Engl J Med 1995;332(10):621–8.
[5] Schwartz MW, Woods SC, Seeley RJ, Barsh GS, Baskin DG, Leibel RL. Is the energy homeostasis system inherently biased toward weight gain? Diabetes 2003;52(2):232–8.

[6] Troiano RP, Flegal KM. Overweight children and adolescents: description, epidemiology, and demographics. Pediatrics 1998;101:497–504.
[7] Kimm SY, Barton BA, Obarzanek E, McMahon RP, Kronsberg SS, Waclawiw MA, et al. Obesity development during adolescence in a biracial cohort: the NHLBI Growth and Health study. Pediatrics 2002;110(5):e54.
[8] Epstein LH, Wing RR, Steranchak L, Dickson B, Michelson J. Comparison of family-based behavior modification and nutrition education for childhood obesity. J Pediatr Psychol 1980; 5(1):25–36.
[9] Epstein LH, Valoski A, Wing RR, McCurley J. Ten-year follow-up of behavioral, family-based treatment for obese children. JAMA 1990;264(19):2519–23.
[10] Jelalian E, Saelens BE. Empirically supported treatments in pediatric psychology: pediatric obesity. J Pediatr Psychol 1999;24(3):223–48.
[11] Kramer FM, Jeffery RW, Forster JL, Snell MK. Long-term follow-up of behavioral treatment for obesity: patterns of weight regain among men and women. Int J Obes 1989;13(2):123–36.
[12] Yanovski JA. Intensive therapies for pediatric obesity. Pediatr Clin North Am 2001; 48(4):1041–53.
[13] Livingston EH. Obesity and its surgical management. Am J Surg 2002;184(2):103–13.
[14] Brolin RE. Bariatric surgery and long-term control of morbid obesity. JAMA 2002; 288(22):2793–6.
[15] Wittgrove AC, Clark GW. Laparoscopic gastric bypass, Roux-en-Y- 500 patients: technique and results, with 3–60 month follow-up. Obes Surg 2000;10(3):233–9.
[16] Sugerman HJ, Sugerman EL, DeMaria EJ, Kellum JM, Kennedy C, Mowery Y, et al. Bariatric surgery for severely obese adolescents. J Gastrointest Surg 2003;7(1):102–8.
[17] Strauss RS, Bradley LJ, Brolin RE. Gastric bypass surgery in adolescents with morbid obesity. J Pediatr 2001;138(4):499–504.
[18] Inge TH, Garcia VF, Daniels SR, Kirk S, Langford L, Roehrig H, et al. A Multidisciplinary Approach to the Adolescent Bariatric Surgical Patient. J Pediatr Surg 2004;39(4):442–7.
[19] Dolan K, Creighton L, Hopkins G, Fielding G. Laparoscopic gastric banding in morbidly obese adolescents. Obes Surg 2003;13(1):101–4.
[20] Garcia VF, Langford L, Inge TH. Application of laparoscopy for bariatric surgery in adolescents. Curr Opin Pediatr 2003;15(3):248–55.
[21] Himes JH, Dietz WH. Guidelines for overweight in adolescent preventive services: recommendations from an expert committee. The Expert Committee on Clinical Guidelines for Overweight in Adolescent Preventive Services. Am J Clin Nutr 1994;59(2):307–16.
[22] Daniels SR, Khoury PR, Morrison JA. The utility of body mass index as a measure of body fatness in children and adolescents: differences by race and gender. Pediatrics 1997; 99(6):804–7.
[23] Pietrobelli A, Faith MS, Allison DB, Gallagher D, Chiumello G, Heymsfield SB. Body mass index as a measure of adiposity among children and adolescents: a validation study. J Pediatr 1998;132(2):204–10.
[24] Dietz WH, Robinson TN. Use of the body mass index (BMI) as a measure of overweight in children and adolescents. J Pediatr 1998;132(2):191–3.
[25] Strauss RS, Pollack HA. Epidemic increase in childhood overweight, 1986–1998. JAMA 2001;286(22):2845–8.
[26] Ogden CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and trends in overweight among US children and adolescents, 1999–2000. JAMA 2002;288(14):1728–32.
[27] Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. BMJ 2000;320(7244):1240–3.
[28] Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, Mei Z, et al. 2000 CDC growth charts for the United States: methods and development. Vital Health Stat 11 2002; 246:1–190.
[29] Wahlqvist ML. Chronic disease prevention: a life-cycle approach which takes account of the environmental impact and opportunities of food, nutrition and public health policies—the

rationale for an eco-nutritional disease nomenclature. Asia Pac J Clin Nutr 2002;11(Supple 9): S759–62.
[30] Ravelli AC, van Der Meulen JH, Osmond C, Barker DJ, Bleker OP. Obesity at the age of 50 years in men and women exposed to famine prenatally. Am J Clin Nutr 1999;70(5):811–6.
[31] Parsons TJ, Power C, Logan S, Summerbell CD. Childhood predictors of adult obesity: a systematic review. Int J Obes Relat Metab Disord 1999;23(Suppl 8):S1–107.
[32] Sorensen HT, Sabroe S, Rothman KJ, Gillman M, Fischer P, Sorensen TI. Relation between weight and length at birth and body mass index in young adulthood: cohort study. BMJ 1997;315(7116):1137.
[33] Bergmann KE, Bergmann RL, Von Kries R, Bohm O, Richter R, Dudenhausen JW, et al. Early determinants of childhood overweight and adiposity in a birth cohort study: role of breast-feeding. Int J Obes Relat Metab Disord 2003;27(2):162–72.
[34] He Q, Karlberg J. Probability of adult overweight and risk change during the BMI rebound period. Obes Res 2002;10(3):135–40.
[35] Cameron N, Demerath EW. Critical periods in human growth and their relationship to diseases of aging. Am J Phys Anthropol 2002;(Suppl 35):159–84.
[36] Whitaker RC, Pepe MS, Wright JA, Seidel KD, Dietz WH. Early adiposity rebound and the risk of adult obesity. Pediatrics 1998;101(3):E5.
[37] Eriksson JG, Forsen T, Tuomilehto J, Osmond C, Barker DJ. Early adiposity rebound in childhood and risk of type 2 diabetes in adult life. Diabetologia 2003;46(2):190–4.
[38] Rolland-Cachera MF, Deheeger M, Bellisle F, Sempe M, Guilloud-Bataille M, Patois E. Adiposity rebound in children: a simple indicator for predicting obesity. Am J Clin Nutr 1984;39(1):129–35.
[39] Silverman BL, Rizzo TA, Cho NH, Metzger BE. Long-term effects of the intrauterine environment. The Northwestern University Diabetes in Pregnancy Center. Diabetes Care 1998; 21(Suppl 2):B142–9.
[40] Caprio S. Insulin resistance in childhood obesity. J Pediatr Endocrinol Metab 2002;15(Suppl): 1487–92.
[41] Heald FP, Khan MA. Teenage obesity. Pediatr Clin North Am 1973;20(4):807–17.
[42] Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. N Engl J Med 1997;337(13):869–73.
[43] Whitaker RC. Understanding the complex journey to obesity in early adulthood. Ann Intern Med 2002;136(12):923–5.
[44] Must A, Strauss RS. Risks and consequences of childhood and adolescent obesity. Int J Obes Relat Metab Disord 1999;23(Suppl 2):S2–11.
[45] Dietz WH. Health consequences of obesity in youth: childhood predictors of adult disease. Pediatrics 1998;101:518–25.
[46] Dietz WH. Childhood weight affects adult morbidity and mortality. J Nutr 1998;128(Suppl 2): 411S–4S.
[47] Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents. A follow-up of the Harvard Growth study of 1922 to 1935. N Engl J Med 1992;327(19):1350–5.
[48] Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. JAMA 1999;282(16):1523–9.
[49] Fontaine KR, Redden DT, Wang C, Westfall AO, Allison DB. Years of life lost due to obesity. JAMA 2003;289(2):187–93.
[50] Strauss RS. Childhood obesity. Pediatr Clin North Am 2002;49(1):175–201.
[51] Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. N Engl J Med 1993;329(14):1008–12.
[52] Wang G, Dietz WH. Economic burden of obesity in youths aged 6 to 17 years: 1979–1999. Pediatrics 2002;109(5):E81.
[53] French SA, Story M, Perry CL. Self-esteem and obesity in children and adolescents: a literature review. Obes Res 1995;3(5):479–90.
[54] Strauss RS. Childhood obesity and self-esteem. Pediatrics 2000;105(1):e15.

[55] Adami GF, Meneghelli A, Scopinaro N. Night eating and binge eating disorder in obese patients. Int J Eat Disord 1999;25(3):335–8.
[56] Wadden TA, Sarwer DB, Womble LG, Foster GD, McGuckin BG, Schimmel A. Psychosocial aspects of obesity and obesity surgery. Surg Clin North Am 2001;81(5):1001–24.
[57] Schwimmer JB, Burwinkle TM, Varni JW. Health-related quality of life of severely obese children and adolescents. JAMA 2003;289(14):1813–9.
[58] Felitti VJ. Long-term medical consequences of incest, rape, and molestation. South Med J 1991;84(3):328–31.
[59] Balcer LJ, Liu GT, Forman S, Pun K, Volpe NJ, Galetta SL, et al. Idiopathic intracranial hypertension: relation of age and obesity in children. Neurology 1999;52(4):870–2.
[60] Cinciripini GS, Donahue S, Borchert MS. Idiopathic intracranial hypertension in prepubertal pediatric patients: characteristics, treatment, and outcome. Am J Ophthalmol 1999;127(2): 178–82.
[61] Lessell S. Pediatric pseudotumor cerebri (idiopathic intracranial hypertension). Surv Ophthalmol 1992;37(3):155–66.
[62] Amin R, Daniels S. Relationship between obesity and sleep-disordered breathing in children: is it a closed loop? J Pediatr 2002;140(6):641–3.
[63] Amin RS, Kimball TR, Bean JA, Jeffries JL, Willging JP, Cotton RT, et al. Left ventricular hypertrophy and abnormal ventricular geometry in children and adolescents with obstructive sleep apnea. Am J Respir Crit Care Med 2002;165(10):1395–9.
[64] Bower CM, Gungor A. Pediatric obstructive sleep apnea syndrome. Otolaryngol Clin North Am 2000;33(1):49–75.
[65] Redline S, Tishler PV, Schluchter M, Aylor J, Clark K, Graham G. Risk factors for sleep-disordered breathing in children. Associations with obesity, race, and respiratory problems. Am J Respir Crit Care Med 1999;159(5 Pt 1):1527–32.
[66] Marcus CL, Curtis S, Koerner CB, Joffe A, Serwint JR, Loughlin GM. Evaluation of pulmonary function and polysomnography in obese children and adolescents. Pediatr Pulmonol 1996;21(3):176–83.
[67] Marcus CL, Loughlin GM. Obstructive sleep apnea in children. Semin Pediatr Neurol 1996; 3(1):23–8.
[68] Silvestri JM, Weese-Mayer DE, Bass MT, Kenny AS, Hauptman SA, Pearsall SM. Polysomnography in obese children with a history of sleep-associated breathing disorders. Pediatr Pulmonol 1993;16(2):124–9.
[69] Mallory Jr GB, Fiser DH, Jackson R. Sleep-associated breathing disorders in morbidly obese children and adolescents. J Pediatr 1989;115(6):892–7.
[70] von Mutius E, Schwartz J, Neas LM, Dockery D, Weiss ST. Relation of body mass index to asthma and atopy in children: the National Health and Nutrition Examination Study III. Thorax 2001;56(11):835–8.
[71] Chu NF, Rimm EB, Wang DJ, Liou HS, Shieh SM. Clustering of cardiovascular disease risk factors among obese schoolchildren: the Taipei Children Heart study. Am J Clin Nutr 1998; 67(6):1141–6.
[72] Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart study. Pediatrics 1999;103:1175–82.
[73] Sorof J, Daniels S. Obesity hypertension in children: a problem of epidemic proportions. Hypertension 2002;40(4):441–7.
[74] Bartosh SM, Aronson AJ. Childhood hypertension. An update on etiology, diagnosis, and treatment. Pediatr Clin North Am 1999;46(2):235–52.
[75] Berenson GS, Wattigney WA, Webber LS. Epidemiology of hypertension from childhood to young adulthood in black, white, and Hispanic population samples. Public Health Rep 1996; 111:23–6.
[76] Buiten C, Metzger B. Childhood obesity and risk of cardiovascular disease: a review of the science. Pediatr Nurs 2000;26(1):13–8.

[77] Nakamura M, Fujioka H, Yamada N, Sakuma M, Okada O, Nakanishi N, et al. Clinical characteristics of acute pulmonary thromboembolism in Japan: results of a multi-center registry in the Japanese Society of Pulmonary Embolism Research. Clin Cardiol 2001;24(2):132–8.
[78] Das UN. Is obesity an inflammatory condition? Nutrition 2001;17:953–66.
[79] Hirsch S, de la Maza P, Mendoza L, Petermann M, Glasinovic A, Paulinelli P, et al. Endothelial function in healthy younger and older hyperhomocysteinemic subjects. J Am Geriatr Soc 2002;50(6):1019–23.
[80] Palasciano G, Portincasa P, Vinciguerra V, Velardi A, Tardi S, Baldassarre G, et al. Gallstone prevalence and gallbladder volume in children and adolescents: an epidemiological ultrasonographic survey and relationship to body mass index. Am J Gastroenterol 1989;84(11):1378–82.
[81] Molleston JP, White F, Teckman J, Fitzgerald JF. Obese children with steatohepatitis can develop cirrhosis in childhood. Am J Gastroenterol 2002;97(9):2460–2.
[82] Roberts EA. Steatohepatitis in children. Best Pract Res Clin Gastroenterol 2002;16(5):749–65.
[83] Schwimmer JB, Deutsch R, Rauch JB, Behling C, Newbury R, Lavine JE. Obesity, insulin resistance, and other clinicopathological correlates of pediatric nonalcoholic fatty liver disease. J Pediatr 2003;143(4):500–5.
[84] Lavine JE, Schwimmer JB. Pediatric initiatives within the Nonalcoholic Steatohepatitis-Clinical Research Network (NASH CNR). J Pediatr Gastroenterol Nutr 2003;37(3):220–1.
[85] Adelman RD, Restaino IG, Alon US, Blowey DL. Proteinuria and focal segmental glomerulosclerosis in severely obese adolescents. J Pediatr 2001;138(4):481–5.
[86] Arslanian S. Type 2 diabetes in children: clinical aspects and risk factors. Horm Res 2002; 57:119–28.
[87] Pinhas-Hamiel O, Dolan LM, Daniels SR, Standiford D, Khoury PR, Zeitler P. Increased incidence of noninsulin-dependent diabetes mellitus among adolescents. J Pediatr 1996; 128:608–15.
[88] Sinha R, Fisch G, Teague B, Tamborlane WV, Banyas B, Allen K, et al. Prevalence of impaired glucose tolerance among children and adolescents with marked obesity. N Engl J Med 2002;346(11):802–10.
[89] Harris SB, Perkins BA, Whalen-Brough E. Noninsulin-dependent diabetes mellitus among First Nations children. New entity among First Nations people of north western Ontario. Can Fam Physician 1996;42:869–76.
[90] Uwaifo GI, Elberg J, Yanovski JA. Impaired glucose tolerance in obese children and adolescents. N Engl J Med 2002;347(4):290–2.
[91] Lazar L, Kauli R, Bruchis C, Nordenberg J, Galatzer A, Pertzelan A. Early polycystic ovary-like syndrome in girls with central precocious puberty and exaggerated adrenal response. Eur J Endocrinol 1995;133(4):403–6.
[92] Lewy VD, Danadian K, Witchel SF, Arslanian S. Early metabolic abnormalities in adolescent girls with polycystic ovarian syndrome. J Pediatr 2001;138(1):38–44.
[93] Castro-Magana M. Hypogonadism and obesity. Pediatr Ann 1984;13(6):491–2.
[94] Loder RT, Aronson DD, Greenfield ML. The epidemiology of bilateral slipped capital femoral epiphysis. A study of children in Michigan. J Bone Joint Surg Am 1993;75(8):1141–7.
[95] Dietz Jr WH, Gross WL, Kirkpatrick Jr JA. Blount disease (tibia vara): another skeletal disorder associated with childhood obesity. J Pediatr 1982;101(5):735–7.
[96] Goulding A, Taylor RW, Jones IE, Manning PJ, Williams SM. Spinal overload: a concern for obese children and adolescents? Osteoporos Int 2002;13(10):835–40.
[97] Dowling AM, Steele JR, Baur LA. Does obesity influence foot structure and plantar pressure patterns in prepubescent children? Int J Obes Relat Metab Disord 2001;25(6):845–52.
[98] Rossner S. Childhood obesity and adulthood consequences. Acta Paediatr 1998;87(1):1–5.
[99] Slyper AH. Childhood obesity, adipose tissue distribution, and the pediatric practitioner. Pediatrics 1998;102(1):E4
[100] Strauss R. Childhood obesity. Curr Probl Pediatr 1999;29(1):1–29.
[101] Yanovski JA. Pediatric obesity. Rev Endocr Metab Disord 2001;2(4):371–83.
[102] Deckelbaum RJ, Williams CL. Childhood obesity: the health issue. Obes Res 2001;9: 4239S–43S.

[103] Dixon JB, Dixon AF, O'Brien PE. Improvements in insulin sensitivity and beta-cell function (HOMA) with weight loss in the severely obese. Homeostatic model assessment. Diabet Med 2003;20(2):127–34.
[104] Dixon JB, O'Brien PE. Health outcomes of severely obese type 2 diabetic subjects 1 year after laparoscopic adjustable gastric banding. Diabetes Care 2002;25(2):358–63.
[105] Pories WJ, MacDonald Jr KG, Flickinger EG, Dohm GL, Sinha MK, Barakat HA, et al. Is type II diabetes mellitus (NIDDM) a surgical disease? Ann Surg 1992;215(6):633–42.
[106] Pories WJ, MacDonald Jr KG, Morgan EJ, Sinha MK, Dohm GL, Swanson MS, et al. Surgical treatment of obesity and its effect on diabetes: 10-year follow-up. Am J Clin Nutr 1992;55(Suppl 2):582S–5S.
[107] Sugerman HJ, Fairman RP, Sood RK, Engle K, Wolfe L, Kellum JM. Long-term effects of gastric surgery for treating respiratory insufficiency of obesity. Am J Clin Nutr 1992;55(Suppl 2): 597S–601S.
[108] Sugerman HJ, Fairman RP, Baron PL, Kwentus JA. Gastric surgery for respiratory insufficiency of obesity. Chest 1986;90(1):81–6.
[109] Sugerman HJ, Felton III WL, Salvant Jr JB, Sismanis A, Kellum JM. Effects of surgically induced weight loss on idiopathic intracranial hypertension in morbid obesity. Neurology 1995;45(9):1655–9.
[110] Michaelides EM, Sismanis A, Sugerman HJ, Felton III WL. Pulsatile tinnitus in patients with morbid obesity: the effectiveness of weight reduction surgery. Am J Otol 2000;21(5):682–5.
[111] Calle EE, Thun MJ, Petrelli JM, Rodriguez C, Heath Jr CW. Body mass index and mortality in a prospective cohort of US adults. N Engl J Med 1999;341(15):1097–105.
[112] NIH conference. Gastrointestinal surgery for severe obesity. Consensus Development Conference Panel. Ann Intern Med 1991;115(12):956–61.
[113] Inge TH, Krebs NF, Garcia VF, Skelton JA, Guice KS, Strauss RS, et al. Bariatric surgery for severely overweight adolescents: concerns and recommendations. Pediatrics 2004;114:217–23.
[114] Rand CS, Macgregor AM. Adolescents having obesity surgery: a 6-year follow-up. South Med J 1994;87(12):1208–13.
[115] Gastrointestinal surgery for severe obesity: National Institutes of Health Consensus Development Conference statement. Am J Clin Nutr 1992;55(Suppl 2):615S–9S.
[116] Guidelines for laparoscopic and open surgical treatment of morbid obesity. American Society for Bariatric Surgery. Society of American Gastrointestinal Endoscopic Surgeons. Obes Surg 2000;10(4):378–9.
[117] Tanner JM. Growth at adolescence. 2nd edition. Oxford (UK): Blackwell Scientific Publications; 1962.
[118] Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. Arch Dis Child 1969;44(235):291–303.
[119] Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. Arch Dis Child 1970;45(239):13–23.
[120] Greulich W, Pyle SI, editors. Radiographic atlas of skeletal development of the hand and wrist. Palo Alto (CA): Stanford University Press; 1983.
[121] Tanner JM, Whitehouse RH, editors. Assessment of Skeletal Maturity and Prediction of Adult Height. (TW2 method). San Diego (CA): Academic Press; 1983.
[122] Dixon JB, Dixon ME, O'Brien PE. Depression in association with severe obesity: changes with weight loss. Arch Intern Med 2003;163(17):2058–65.
[123] Kolotkin RL, Meter K, Williams GR. Quality of life and obesity. Obes Rev 2001;2(4):219–29.
[124] Kolotkin RL, Crosby RD, Williams GR, Hartley GG, Nicol S. The relationship between health-related quality of life and weight loss. Obes Res 2001;9(9):564–71.
[125] Bocchieri LE, Meana M, Fisher BL. A review of psychosocial outcomes of surgery for morbid obesity. J Psychosom Res 2002;52(3):155–65.
[126] Chapman A, Game P, O'Brien P, Maddern G, Kiroff G, Foster B, et al. Systematic review of laparoscopic adjustable gastric banding for the treatment of obesity: Update and reappraisal. 8915. Australian Safety & Efficacy Register of New Interventional Procedures–Surgical,

Report no. 31, second edition. Available at: http://www.surgeons.org/asernip-s/publications_obesity.htm. Accessed: July 11, 2004.
[127] Dixon JB, Chapman L, O'Brien P. Marked improvement in asthma after lap band surgery for morbid obesity. Obes Surg 1999;9(4):385–9.
[128] Dixon JB, Dixon ME, O'Brien PE. Quality of life after lap-band placement: influence of time, weight loss, and comorbidities. Obes Res 2001;9(11):713–21.
[129] Dixon JB, O'Brien PE. Changes in comorbidities and improvements in quality of life after lap band placement. Am J Surg 2002;184(6B):S51–4.
[130] Dixon JB, O'Brien PE. Lipid profile in the severely obese: changes with weight loss after lap band surgery. Obes Res 2002;10(9):903–10.
[131] Dixon JB, Schachter LM, O'Brien PE. Sleep disturbance and obesity: changes following surgically induced weight loss. Arch Intern Med 2001;161(1):102–6.
[132] Fisher BL, Barber AE. Gastric bypass procedures. Eur J Gastroenterol Hepatol 1999;11(2):93–7.
[133] Pope GD, Birkmeyer JD, Finlayson SR. National trends in utilization and in-hospital outcomes of bariatric surgery. J Gastrointest Surg 2002;6(6):855–61.
[134] Pories WJ, Swanson MS, MacDonald KG, Long SB, Morris PG, Brown BM, et al. Who would have thought it? An operation proves to be the most effective therapy for adult-onset diabetes mellitus. Ann Surg 1995;222(3):339–50.
[135] Brolin RE. Gastric bypass. Surg Clin North Am 2001;81(5):1077–95.
[136] Balsiger BM, Murr MM, Poggio JL, Sarr MG. Bariatric surgery. Surgery for weight control in patients with morbid obesity. Med Clin North Am 2000;84(2):477–89.
[137] Albrecht RJ, Pories WJ. Surgical intervention for the severely obese. Baillieres Best Pract Res Clin Endocrinol Metab 1999;13(1):149–72.
[138] Nguyen NT, Rivers R, Wolfe BM. Early gastrointestinal hemorrhage after laparoscopic gastric bypass. Obes Surg 2003;13(1):62–5.
[139] Buckwalter JA, Herbst Jr CA. Leaks occurring after gastric bariatric operations. Surgery 1988;103(2):156–60.
[140] Sanyal AJ, Sugerman HJ, Kellum JM, Engle KM, Wolfe L. Stomal complications of gastric bypass: incidence and outcome of therapy. Am J Gastroenterol 1992;87(9):1165–9.
[141] Gollobin C, Marcus WY. Bariatric beriberi. Obes Surg 2002;12(3):309–11.
[142] Chaves LC, Faintuch J, Kahwage S, Alencar FA. A cluster of polyneuropathy and Wernicke-Korsakoff syndrome in a bariatric unit. Obes Surg 2002;12(3):328–34.
[143] Amaral JF, Thompson WR, Caldwell MD, Martin HF, Randall HT. Prospective hematologic evaluation of gastric exclusion surgery for morbid obesity. Ann Surg 1985;201(2):186–93.
[144] Halverson JD. Vitamin and mineral deficiencies following obesity surgery. Gastroenterol Clin North Am 1987;16(2):307–15.
[145] Halverson JD. Micronutrient deficiencies after gastric bypass for morbid obesity. Am Surg 1986;52(11):594–8
[146] MacLean LD, Rhode BM, Shizgal HM. Nutrition following gastric operations for morbid obesity. Ann Surg 1983;198(3):347–55.
[147] Mason EE. Starvation injury after gastric reduction for obesity. World J Surg 1998;22(9):1002–7.
[148] Schilling RF, Gohdes PN, Hardie GH. Vitamin B12 deficiency after gastric bypass surgery for obesity. Ann Intern Med 1984;101(4):501–2.
[149] Boylan LM, Sugerman HJ, Driskell JA. Vitamin E, vitamin B-6, vitamin B-12, and folate status of gastric bypass surgery patients. J Am Diet Assoc 1988;88(5):579–85.
[150] Kalkwarf HJ, Khoury JC, Lanphear BP. Milk intake during childhood and adolescence, adult bone density, and osteoporotic fractures in US women. Am J Clin Nutr 2003;77(1):257–65.
[151] Wysocki T, Greco P, Harris MA, Bubb J, White NH. Behavior therapy for families of adolescents with diabetes: maintenance of treatment effects. Diabetes Care 2001;24(3):441–6.
[152] Hall JG, Solehdin F. Folate and its various ramifications. Adv Pediatr 1998;45:1–35.
[153] Bergfeld WF, Mulinari-Brenner F. Shedding: how to manage a common cause of hair loss. Cleve Clin J Med 2001;68(3):256–61.

[154] Neve HJ, Bhatti WA, Soulsby C, Kincey J, Taylor TV. Reversal of hair loss following vertical gastroplasty when treated with zinc sulphate. Obes Surg 1996;6(1):63–5
[155] Munoz-Farjas E, Jerico I, Pascual-Millan LF, Mauri JA, Morales-Asin F. Neuropathic beriberi as a complication of surgery of morbid obesity. Rev Neurol 1996;24(128):456–8.
[156] Towbin A, Inge TH, Garcia VF, Roerig HR, Clements RH, Harmon CM, et al. Beriberi after gastric bypass surgery. J Pediatrics 2004;145(2):263–7.
[157] Breaux CW. Obesity Surgery in Children. Obes Surg 1995;5(3):279–84.
[158] Stanford A, Glascock JM, Eid GM, Kane T, Ford HR, Ikramuddin S, et al. Laparoscopic roux-en-Y gastric bypass in morbidly obese adolescents. J Pediatr Surg 2003;38(3):430–3.
[159] Greenstein RJ, Rabner JG. Is adolescent gastric-restrictive antiobesity surgery warranted? Obes Surg 1995;5(2):138–44.
[160] Abu-Abeid S, Gavert N, Klausner JM, Szold A. Bariatric surgery in adolescence. J Pediatr Surg 2003;38(9):1379–82.