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15.4 Obesity and the Metabolic Syndrome

Purging disorder is characterized by recurrent purging behavior after consuming a small amount of food in persons of normal weight who have a distorted view of their weight or body image. Purging behavior includes self-induced vomiting, laxative abuse, enemas, and diuretics. To make the diagnosis, the behavior must not be associated with anorexia nervosa. Purging disorder is differentiated from bulimia nervosa because purging behavior occurs after eating small quantities of food or drink and does not occur as a result of a binge episode. Purging episodes should occur at least once a week over a 3-month period before the diagnosis is made. REFERENCES Brown TA, Keel PK, Striegel RH. Feeding and eating conditions not elsewhere classified (NEC) in DSM-5. Psych Annals. 2012;42:421. De Young KP, Lavender JM, Wilson GT, Wonderlich SA. Binge eating disorder in DSM-5. Psych Annals. 2012;42:410. Friborg, O., Martinussen, M., Kaiser, S., Overgård, KT, Martinsen, EW, Schmierer, P., Rosenvinge, JH, Personality disorders in eating disorder not otherwise specified and binge eating disorder: a meta-analysis of comorbidity studies. J Nerv Ment Dis . 2014;202(2):119–125. Gianini LM, White MA, Masheb RM. Eating pathology, emotion regulation, and emotional overeating in obese adults with binge eating disorder. Eat Behav. 2013;14(3):309–313. Goldschmidt AB, Grange DL, Powers P, Crow SJ, Hill LL, Peterson CB, Crosby RD, Mitchell JE. Eating disorder symptomatology in normal-weight vs. obese individuals with binge eating disorder. Obesity. 2011;19:1515. Perez M, Warren CS. The relationship between quality of life, binge-eating disorder, and obesity status in an ethnically diverse sample. Obesity. 2012;20:879. Pollert GA, Engel SG, Schreiber-Gregory DN, Crosby RD, Cao L, Wonderlich SA, Tanofsky-Kraff M, Mitchell JE. The role of eating and emotion in binge eating disorder and loss of control eating. Int J Eat Disord. 2013;46(3):233–238. Schwitzer AM. Diagnosing, conceptualizing, and treating eating disorders not otherwise specified: A comprehensive practice model. J Counsel Dev. 2012;90:281. Striegel-Moore RH, Franko DL. Should binge eating disorder be included in the DSM-V? A critical review of the state of the evidence. Annu Rev Clin Psychol. 2008;4:305. Tanofsky-Kraff M, Bulik CM, Marcus MD, Striegel RH, Wilfley DE, Wonderlich SA, Hudson JI. Binge eating disorder: The next generation of research. Int J Eat Disord. 2013;46(3):193–207. Vander Wal JS. Night eating syndrome: A critical review of the literature. Clin Psychol Rev. 2012;32:49. Wadden TA, Faulconbridge LF, Jones-Corneille LR, Sarwer DB, Fabricatore AN, Thomas JG, Wilson GT,

Alexander MG, Pulcini ME, Webb VL, Williams NN. Binge eating disorder and the outcome of bariatric surgery at one year: A prospective, observational study. Obes Res. 2011;19:1220. 15.4 Obesity and the Metabolic Syndrome Obesity is a chronic illness manifested by an excess of body fat. It is generally measured by the body mass index (BMI), but a more accurate method is to use body composition analysis also known as biometric impedance analysis (BIA). Excess body fat generally results from a greater amount of calories consumed than are burnt off. In healthy individuals, body fat—which is different from the BMI—varies by gender. It ranges from

10 to 13 percent of essential fat to 25 to 31 percent on average of body weight in healthy women. In men, it ranges from 2 to 5 percent of essential fat to 18 to 24 percent on average. The global epidemic of obesity has resulted in an alarming increase in associated morbidity and mortality. Although manifestation of obesity and its comorbid states are largely physical, it has overwhelming psychological ramifications. The diagnosis of obesity using the BMI is discussed below. COMORBIDITY Evidence shows a correlation between obesity and psychiatric disorders. In fact, among treatment-seeking obese patients, there is a higher prevalence of morbid psychiatric illness by 40 to 60 percent. Disorders associated with obesity include eating disorders (particularly binge eating disorder), substance use disorders, psychotic disorders (schizophrenia), mood disorders, anxiety disorders, personality disorders, attentiondeficit/hyperactivity disorder (ADHD), and posttraumatic stress disorder (PTSD). There are two eating disorders that can be associated with obesity: bulimia nervosa and binge eating disorder. Both of these disorders are distinct in their clinical characteristics and have some similarities. They both are associated with significant psychopathology and need to be addressed multimodally to ensure success. It is important to note that not all patients with bulimia nervosa are obese; they may be overweight or of normal weight. See Sections 15.2 and 15.3 for further discussions on bulimia nervosa and binge eating disorder, respectively. EPIDEMIOLOGY Obesity rates continue to grow at epidemic proportions in the United States and other industrialized nations, representing a serious public health threat to millions of people. In the United States, approximately 36 percent of adults are obese. The prevalence of obesity is highest in minority populations, particularly among non-Hispanic black women. More than one half of these individuals, 40 years of age or older, are obese and more than 80 percent are overweight. The prevalence of obesity in adolescents in the United States has also increased from about 15 percent in 2000 to about 35 percent in 2012, while the prevalence rates for overweight children ages 6 to 11 range from 14 to 25 percent; however, there has been a slight decrease in recent years. Obesity also has economic effects. On an individual level, medical spending is approximately 42 percent higher for an obese person compared with that for a person of normal weight. On a national level, costs attributed to both overweight (BMI 25 to 29.9) and obesity (BMI greater than 30) account for 9.1 percent of US health care costs, and if the current trend continues, obesity will account for 16 percent of US health care costs by 2030. ETIOLOGY Persons with no medical etiology accumulate fat by eating more calories than are

expended as energy; thus intake of energy exceeds its dissipation. To reduce body fat, fewer calories must be consumed or more calories must be burnt. An error of no more than 10 percent in either intake or output would lead to a 30-pound change in body weight in 1 year. Satiety Satiety is the feeling that results when hunger is satisfied. Persons stop eating at the end of a meal because they have replenished nutrients that had been depleted. Persons become hungry again when nutrients restored by earlier meals are once again depleted. It seems reasonable that a metabolic signal, derived from food that has been absorbed, is carried by the blood to the brain, where the

signal activates receptor cells, probably in the hypothalamus, to produce satiety. Some studies have shown evidence for dysfunction in serotonin, dopamine, and norepinephrine involvement in regulating eating behavior through the hypothalamus. Other hormonal factors that may be involved include corticotrophin-releasing factor (CRF), neuropeptide Y, gonadotropin-releasing hormone, and thyroid-stimulating hormone. A newly found substance, obestatin, which is made in the stomach, is a hormone that in animal experiments produces satiety and may have potential use as a weight-loss agent in humans. Hunger results from a decrease in the strength of metabolic signals, secondary to the depletion of critical nutrients. Cannabinoid receptors are related to appetite and are stimulated with cannabis (marijuana) use. A cannabinoid inverse antagonist has been developed that blocks appetite. Satiety occurs soon after the beginning of a meal and before the total caloric content of the meal has been absorbed; therefore, satiety is only one regulatory mechanism controlling food intake. Appetite, defined as the desire for food, is also involved. A hungry person may eat to full satisfaction when food is available, but appetite can also induce a person to overeat past the point of satiety. Appetite may be increased by psychological factors such as thoughts or feelings, and an abnormal appetite may result in an abnormal increase in food intake. Eating is also affected by cannabinoid receptors, which, when stimulated, increase appetite. Marijuana acts on that receptor that accounts for the “munchies” associated with marijuana use. A drug called rimonabant (Acomplia) is an inverse agonist to the cannabidiol receptor, meaning that it blocks appetite. It was withdrawn from the market because of adverse effects; however, in theory, inverse cannabinoid receptor agonists may have clinical use. The olfactory system may play a role in satiety. Experiments have shown that strong stimulation of the olfactory bulbs in the nose with food odors by use of an inhaler saturated with a particular smell produces satiety for that food. This may have implications for therapy of obesity. Genetic Factors The existence of numerous forms of inherited obesity in animals and the ease with which adiposity can be produced by selective breeding make it clear that genetic factors can play a role in obesity. These factors must also be presumed to be important in human obesity. About 80 percent of patients who are obese have a family history of obesity. This fact can be accounted for not only by genetic factors but also in part by identification with fat parents and by learned oral methods for coping with anxiety. Nonetheless, studies show that identical twins raised apart can both be obese, an observation that suggests a hereditary role. To date, no specific genetic marker of obesity has been found. Table

15.4-1 lists the genetic factors affecting body weight. Table 15.4-1 Genetic Factors Affecting Body Weight Developmental Factors

Early in life, adipose tissue grows by increases in both cell number and cell size. Once the number of adipocytes has been established, it does not seem to be susceptible to change. Obesity that begins early in life is characterized by adipose tissue with an increased number of adipocytes of increased size. Obesity that begins in adult life, on the other hand, results solely from an increase in the size of the adipocytes. In both instances, weight reduction produces a decrease in cell size. The greater number and size of adipocytes in patients with juvenile-onset diabetes may be a factor in their widely recognized difficulties with weight reduction and the persistence of their obesity. The distribution and amount of fat vary in individuals, and fat in different body areas has different characteristics. Fat cells around the waist, flanks, and abdomen (the socalled potbelly) are more active metabolically than those in the thighs and buttocks. The former pattern is more common in men and has a higher correlation with cardiovascular disease than does the latter pattern. Women, whose fat distribution is in the thighs and buttocks, may become obsessed with nostrums that are

advertised to reduce fat in these areas (so-called cellulite, which is not a medical term), but no externally applied preparation to reduce this fat pattern exists. Men with abdominal fat may attempt to reduce their girth with machines that exercise the abdominal muscles, but exercise has no effect on loss of this type of fat. A hormone called leptin, which is made by fat cells, acts as a fat thermostat. When the blood level of leptin is low, more fat is consumed; when high, less fat is consumed. Further research is needed to determine whether this might lead to new ways of managing obesity. Physical Activity Factors The marked decrease in physical activity in affluent societies seems to be the major factor in the rise of obesity as a public health problem. Physical inactivity restricts energy expenditure and may contribute to increased food intake. Although food intake increases with increasing energy expenditure over a wide range of energy demands, intake does not decrease proportionately when physical activity falls below a certain minimum level. Brain-Damage Factors Destruction of the ventromedial hypothalamus can produce obesity in animals, but this is probably a very rare cause of obesity in humans. There is evidence that the central nervous system, particularly in the lateral and ventromedial hypothalamic areas, adjusts to food intake in response to changing energy requirements so as to maintain fat stores at a baseline determined by a specific set point. This set point varies from one person to another and depends on height and body build. Health Factors In only a small number of cases is obesity the consequence of identifiable illness. Such cases include a variety of rare genetic disorders, such as Prader-Willi syndrome, as well as neuroendocrine abnormalities (Table 15.4-2). Hypothalamic obesity results from damage to the ventromedial region of the hypothalamus (VMH), which has been studied extensively in laboratory animals and is a known center of appetite and weight regulation. In humans, damage to the VMH may result from trauma, surgery, malignancy, or inflammatory disease.

Table 15.4-2 Illnesses that Can Explain Some Cases of Obesity Some forms of depression, particularly seasonal affective disorder, are associated with weight gain. Most persons who live in seasonal climates report increases in appetite and weight during the fall and winter months, with decreases in the spring and summer. Depressed patients usually lose weight, but some will gain weight. Other Clinical Factors A variety of clinical disorders are associated with obesity. Cushing’s disease is associated with a characteristic fat distribution and moon-like face (Fig. 15.4-1). Myxedema is associated with weight gain, although not invariably. Other neuroendocrine disorders include adiposogenital dystrophy (Fröhlich’s syndrome), which is characterized by obesity and sexual and skeletal abnormalities.

FIGURE 15.4-1 Cushing’s syndrome. Plethoric, “moon-faced” with “sun-fish” mouth. (From Douthwait AH, ed. French’s Index of Differential Diagnosis. 7th ed. Baltimore: Williams & Wilkins; 1954:513, with permission.) Psychotropic Drugs Long-term use of steroid medications is associated with significant weight gain, as is the use of several psychotropic agents. Patients treated for major depression, psychotic disturbances, and bipolar disorder typically gain 3 to 10 kg, with even larger gains with chronic use. This can produce the so-called metabolic syndrome discussed below. Psychological Factors Although psychological factors are evidently crucial to the development of obesity, how such psychological factors result in obesity is not known. The food-regulating mechanism is susceptible to environmental influence, and cultural, family, and psychodynamic factors have all been shown to contribute to the development of obesity.

Although many investigators have proposed that specific family histories, precipitating factors, personality structures, or unconscious conflicts cause obesity, overweight persons may suffer from every conceivable psychiatric disorder and come from a variety of disturbed backgrounds. Many obese patients are emotionally disturbed persons who, because of the availability of the overeating mechanism in their environments, have learned to use hyperphagia as a means of coping with psychological problems. Some patients may show signs of serious mental disorder when they attain normal weight because they no longer have that coping mechanism. DIAGNOSIS AND CLINICAL FEATURES The diagnosis of obesity, if done in a sophisticated way, involves the assessment of body fat. As this is rarely practical, the use of height and weight to calculate BMI is recommended. Figure 15.4-2 presents a chart for determining BMI from height and weight.

FIGURE 15.4-2 Body mass index (BMI) chart. To determine BMI, find the patient's weight on the left of the graph and their height on the top of the graph. Follow the two categories toward the middle of the graph until they intersect. This point represents the patient's BMI. In most cases of obesity, it is not possible to identify the precise etiology, given the multitude of possible causes and their interactions. Instances of secondary obesity (described in Table 15.4-3) are rare but should not be overlooked. Table 15.4-3 Psychiatric Medications and Changes in Body Weight

The habitual eating patterns of many obese persons often seem similar to patterns found in experimental obesity. Impaired satiety is a particularly important problem. Obese persons seem inordinately susceptible to food cues in their environment, to the palatability of foods, and to the inability to stop eating if food is available. Obese persons are usually susceptible to all kinds of external stimuli to eating, but they remain relatively unresponsive to the usual internal signals of hunger. Some are unable to distinguish between hunger and other kinds of dysphoria. DIFFERENTIAL DIAGNOSIS Other Syndromes The night eating syndrome, in which persons eat excessively after they have had their evening meal, seems to be precipitated by stressful life circumstances and, once present, tends to recur daily until the stress is alleviated. Night eating may also occur as a result of using sedatives to sleep, which may produce sleepwalking and eating. This has been reported with the use of zolpidem (Ambien) in patients. (See Section 15.3 for further

discussion on night eating syndrome.) Binge eating disorder is characterized by sudden, compulsive ingestion of very large amounts of food in a short time, usually with great subsequent agitation and selfcondemnation. Binge eating also appears to represent a reaction to stress. In contrast to night eating syndrome, however, these bouts of overeating are not periodic, and they are far more often linked to specific precipitating circumstances. Pickwickian syndrome is said to exist when a person is 100 percent over desirable weight and has associated respiratory and cardiovascular pathology. Body Dysmorphic Disorder (Dysmorphophobia) Some obese persons feel that their bodies are grotesque and loathsome and that others view them with hostility and contempt. This feeling is closely associated with selfconsciousness and impaired social functioning. Emotionally healthy obese persons have no body image disturbances, and only a minority of neurotic obese persons have such disturbances. The disorder is confined mainly to persons who have been obese since childhood; even among them, less than half suffer from it. (See Section 10.2 for a complete discussion of body dysmorphic disorder.) COURSE AND PROGNOSIS Effects on Health Obesity has adverse effects on health and is associated with a broad range of illnesses (Table 15.4-4). There is a strong correlation between obesity and cardiovascular disorders. Hypertension (blood pressure higher than 140/90 mm Hg) is three times higher for persons who are

overweight, and hypercholesterolemia (blood cholesterol over 240 mg/dL) is twice as common. Studies show that blood pressure and cholesterol levels can be reduced by weight reduction. Diabetes, which has clear genetic determinations, can often be reversed with weight reduction, especially type 2 diabetes (mature-onset or noninsulin-dependent diabetes mellitus). Table 15.4-4 Health Disorders Thought to Be Caused or Exacerbated by Obesity

According to the National Institutes of Health data, obese men, regardless of smoking habits, have a higher mortality from colon, rectal, and prostate cancer than men of normal weight. Obese women have a higher mortality from cancer of the gallbladder, biliary passages, breast (postmenopause), uterus (including cervix and endometrium), and ovaries than women of normal weight. Longevity

Reliable studies indicate that the more overweight a person is, the higher is that person's risk for death. A person who reduces weight to acceptable levels has a mortality decline to normal rates. Weight reduction may be lifesaving for patients with extreme obesity, defined as weight that is twice the desirable weight. Such patients may have cardiorespiratory failure, especially when asleep (sleep apnea). A number of studies have demonstrated that decreasing caloric intake by 30 percent or more in young or middle-aged laboratory animals prevents or retards age-related chronic diseases and significantly prolongs the maximal lifespan. The mechanisms through which this effect is mediated are not known, but they may include reductions in metabolic rate, oxidative stress, and inflammation; improved insulin sensitivity; and changes in neuroendocrine and sympathetic nervous system function. Whether long-term calorie restriction with adequate nutrition slows aging in humans is not yet known. Prognosis The prognosis for weight reduction is poor, and the course of obesity tends toward inexorable progression. Of patients who lose significant amounts of weight, 90 percent regain it eventually. The prognosis is particularly poor for those who become obese in childhood. Juvenile-onset obesity tends to be more severe, more resistant to treatment, and more likely to be associated with emotional disturbance than is adult obesity. Discrimination Toward the Obese. Overweight and obese individuals are subject to significant prejudice and discrimination in the United States and other industrialized nations. In a culture in which beauty ideals are thin and highly unrealistic, overweight people are blamed for their condition and are the subject of teasing, bias, and discrimination (sometimes called "fatism"). Income and earning power are suppressed in overweight people, and untoward social conditions, such as absence of romantic relationships, are more common. Furthermore, obese individuals face limited access to health care and may receive biased diagnoses and treatment from medical and mental health providers. TREATMENT As mentioned above, many patients routinely treated for obesity may develop anxiety or depression. A high incidence of emotional disturbances has been reported among obese persons undergoing long-term, in-hospital treatment by fasting or severe calorie restriction. Obese persons with extensive psychopathology, those with a history of emotional disturbance during dieting, and those in the midst of a life crisis should attempt weight reduction cautiously and under careful supervision. Diet The basis of weight reduction is simple—establish a caloric deficit by bringing intake below output. The simplest way to reduce caloric intake is by means of a low-calorie diet. This strategy requires an adequate amount of protein intake with balanced

carbohydrate and fat intake and should be done under medical supervision. The best long-term effects are achieved with a balanced diet that contains readily available foods. For most persons,

the most satisfactory reducing diet consists of their usual foods in amounts determined with the aid of tables of food values, which are available in standard books on dieting. Such a diet gives the best chance of long-term maintenance of weight loss. Total unmodified fasts are used for short-term weight loss, but they have associated morbidity including orthostatic hypotension, sodium diuresis, and impaired nitrogen balance. Ketogenic diets are high-protein, high-fat diets used to promote weight loss. They have high cholesterol content and produce ketosis, which is associated with nausea, hypotension, and lethargy. Many obese persons find it tempting to use a novel or even bizarre diet. Table 15.4-5 contains details and comparisons of various types of diets. Whatever effectiveness these diets may have in large part results from their monotony. When a dieter stops the diet and returns to the usual fare, the incentives to overeat are multiplied. In general, the best method of weight loss is a balanced diet of 1,100 to 1,200 calories. Such a diet can be followed for long periods but should be supplemented with vitamins, particularly iron, folic acid, zinc, and vitamin B6. Table 15.4-5 Types of Diets Exercise Increased physical activity is an important part of a weight-reduction regimen. Because

caloric expenditure in most forms of physical activity is directly proportional to body weight, obese persons expend more calories than persons of normal weight with the same amount of activity. Furthermore, increased physical activity may actually decrease food intake by formerly sedentary persons. This combination of increased caloric expenditure and decreased food intake makes an increase in physical activity a highly desirable feature of any weight-reduction program. Exercise also helps maintain weight loss. It is essential in the treatment of the metabolic syndrome. Lifestyle Change A lifestyle change empowers the patient to set goals of weight management. Simple lifestyle modification strategies that patients should be encouraged to follow include: Personal behavior during a meal: Eat slowly and savor each mouthful Chew each bite 30 times before swallowing Put the fork down between bites Delay eating for 2 to 3 minutes and converse Postpone a snack for 10 minutes Serve food on a smaller plate Divide portions in half so another portion may be permitted Reduce eating cues: Eat only at one designated place Leave the table as soon as eating is done Do not combine eating with other activities (e.g., reading or watching television) Do not put bowls of food on the table Stock home with healthier food choices Shop for groceries from a list after a full meal Plan meals Keep a food diary to link eating with hunger and nonhunger episodes Substitute other activities for snacking Pharmacotherapy Various drugs, some more effective than others, are used to treat obesity. Table 15.4-6 lists the drugs currently available for this use. Drug treatment is effective because it suppresses appetite, but tolerance to this effect may develop after several weeks of use. An initial trial period of 4 weeks with a specific drug can be used; then, if the patient responds with weight loss, the drug can be continued to see whether tolerance develops. If a drug remains effective, it can be dispensed for a longer time until the desired weight

is achieved. Table 15.4-6 Common Drugs for the Treatment of Obesity Orlistat. One weight-loss medication approved by the Food and Drug Administration (FDA) for long-term use is orlistat (Xenical), which is a selective gastric and pancreatic lipase inhibitor that reduces the absorption of dietary fat (which is then excreted in stool). In clinical trials, orlistat (120 mg, three times a day), in combination with a low-calorie diet, induced losses of approximately 10 percent of initial weight in the first 6 months, which were generally well maintained for periods up to 24 months. Because of its peripheral mechanism of action, orlistat is generally free of the central nervous system effects (i.e., increased pulse, dry mouth, insomnia) that are associated with most weight-loss medications.

The principal adverse effects of orlistat are gastrointestinal; patients must consume 30 percent or fewer calories from fat to prevent adverse events that include oily stool, flatulence with discharge, and fecal urgency. A lower dosed over-the-counter formulation of orlistat (Alli) was approved by the FDA in 2007. Sibutramine. Sibutramine (Meridia) is a β-phenylethylamine that inhibits the reuptake of serotonin and norepinephrine (and dopamine to a limited extent). It was approved by the FDA in 1997 for weight loss and the maintenance of weight loss (i.e., long-term use). Lorcaserin. Lorcaserin (Belviq) has been approved by the FDA for the treatment of obesity in adults. Lorcaserin is a selective serotonin agonist that suppresses appetite and reduces food intake. One double-blind, placebo-controlled trial showed that obese patients lost about 4 percent more of their body weight in 1 year while on lorcaserin compared with controls. Additionally, weight loss was maintained in 70 percent of patients who took lorcaserin for 2 years. Another trial showed that obese patients who took lorcaserin 10 mg 1 to 2 times per day in conjunction with nutritional and exercise

programs lost 6 percent of their body weight after 1 year. The recommended dosage is 10 mg twice a day. If the patient does not see a 5 percent reduction of their body weight within 12 weeks of treatment, lorcaserin treatment should be discontinued. Side effects of lorcaserin include headaches, dizziness, fatigue, nausea, dry mouth, and constipation. Rare but serious side effects include a chemical imbalance (serotonin syndrome), suicidal thoughts, psychiatric problems, and problems with memory or comprehension. Pregnant women should not take lorcaserin. Phentermine-topiramate. Phentermine-topiramate (Qsymia) has been approved by the FDA for weight management treatment in conjunction with diet and exercise. It combines lower doses of immediate-release phentermine, a weight-loss drug prescribed for short-term use, and controlled-release topiramate, an anticonvulsant. Patients should start at the lowest dose (3.75 mg phentermine/23 mg topiramate extended release), then increase to the recommended dose (7.5 mg/46 mg). In some circumstances, patients may have their dose increased to the highest dose (15 mg/92 mg). In clinical trials, patients have shown an average weight loss ranging from 7 percent (lowest dose) to 9 percent (recommended dose) over those taking a placebo. Side effects include paraesthesia, dry mouth, altered taste, increased heart rate, possible birth defects, and psychiatric problems (depression, suicidal thoughts, impaired memory, and concentration). If the patients do not see a 3 percent reduction in their body weight after 12 weeks on the recommended dose, the dosage may be increased to the highest dose. If patients do not see a 5 percent reduction in their body weight after 12 weeks on the highest dose, treatment with Qsymia should be discontinued. Surgery Gastroplasty. Vertical banded gastroplasty (VBG) is a restrictive-only operation that involves creating a small gastric reservoir or pouch measuring 15 to 20 mL in volume, which then empties into the residual stomach through a calibrated or banded outlet. On average, patients lose 40 to 50 pounds of excess body weight over the first 1 to 2 years postoperatively. Vomiting, electrolyte imbalance, and obstruction may occur. A syndrome called dumping, which consists of palpitations, weakness, and sweating, may follow surgical procedures in some patients if they ingest large amounts of carbohydrates in a single meal. Due to such complications, VBG is now only performed in a few centers in the United States. Gastric Bypass. Since the early 1990s, gastric bypass (Fig. 15.4-3) has greatly replaced VBG as the operation of choice. The procedure involves dividing the stomach into two pouches—a small upper pouch and a larger lower “remnant” pouch—and then rearranging the small intestine to connect to both. The expected weight loss averages 70 percent of excess body weight with the maximum weight loss occurring by 3 years postoperatively (Fig. 15.4-4). The main complications of gastric bypass surgery are primarily seen

during the perioperative period. Mortality is less than 0.5 percent and is

mainly due to pulmonary emboli or sepsis secondary to anastomotic leakage. Vitamin B12 and iron deficiencies may present and may require oral supplementation. All patients need to take multivitamins postoperatively and need to be followed at regular intervals for nutritional assessment. About 10 to 15 percent of patients will either fail to achieve significant weight loss or regain a significant amount of their loss after 2 or 3 years. This is usually due to consumption of carbohydrates, such as potato chips or other snack foods. Psychological treatment of abnormal eating behaviors is essential to prevent weight gain. FIGURE 15.4-3 Roux-en Y gastric bypass. (From Sadock BJ, Sadock VA, Ruiz P, eds. Kaplan & Sadock's Comprehensive Textbook of Psychiatry. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 2009, with permission.)

FIGURE 15.4-4 A. Preoperative photograph of a woman who lost 140 pounds after laparoscopic gastric bypass surgery with significant deflation and poor skin quality. B. Postoperative photograph after extended vertical thigh lift demonstrates tightening of the skin and dramatic improvement of the thigh appearance. (From Sadock BJ, Sadock VA, Ruiz P, eds. Kaplan & Sadock's Comprehensive Textbook of Psychiatry. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 2009, with permission.) Gastric Banding. Laparoscopic adjustable gastric band was approved by the FDA in 2002 and is one of the least invasive operations for obesity because it does not involve cutting the stomach or the intestine. It involves placing a band around the upper part of the stomach, creating a smaller stomach above the band and a larger stomach below the band (Fig. 15.4-5). The smaller stomach allows the patient to feel fuller quicker, thus reducing the amount of food intake. Average weight loss is approximately 37 to 50 percent of excess body weight. Complications involve band movement, erosion, malfunction, and slippage (stomach herniating through the band). Improvements in the design of the band and newer placement techniques appear to be reducing complications.

FIGURE 15.4-5 Laparoscopic adjustable gastric band. (From Sadock BJ, Sadock VA, Ruiz P, eds. Kaplan & Sadock's Comprehensive Textbook of Psychiatry. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 2009, with permission.) Other Methods. The surgical removal of fat (lipectomy) has no effect on weight loss in the long run nor does liposuction, which has value only for cosmetic reasons. Bariatric surgery is now recommended in individuals who have serious obesity-related health complications and a BMI of greater than 35 kg/m2 (or a BMI greater than 40 kg/m2 in the absence of major health complications). Before surgery, candidates should have tried to lose weight using the safer, more traditional options of diet, exercise, and weight loss medication. Psychotherapy The psychological problems of obese persons vary, and there is no particular personality type that is more prone to obesity. Some patients may respond to insight-oriented psychodynamic therapy with weight loss, but this treatment has not had much success. Uncovering the unconscious causes of overeating may not alter the behavior of persons who overeat in response to stress, although it may serve to augment other treatment methods. Years after successful psychotherapy many persons who overeat under stress continue to do so. Obese persons seem particularly vulnerable to overdependency on a therapist, and the inordinate regression that may occur during the uncovering psychotherapies should be carefully monitored. Behavior modification has been the most successful of the therapeutic approaches for obesity and is considered the method of choice. Patients are taught to recognize external cues that are associated with eating and to keep diaries of foods consumed in particular circumstances, such as

at the movies or while watching television, or during certain emotional states, such as anxiety or depression. Patients are also taught to develop new eating patterns, such as eating slowly, chewing food well, not reading while eating, and not eating between meals or when not seated. Operant conditioning therapies that use rewards such as praise or new clothes to reinforce weight loss have also been successful.

Group therapy helps to maintain motivation, to promote identification among members who have lost weight, and to provide education about nutrition. Comprehensive Approach The National Heart, Lung, and Blood Institute formulated key recommendations for patients and the public regarding weight loss. These are listed in Table 15.4-7. Table 15.4-7 Key Recommendations for Healthy Weight METABOLIC SYNDROME The metabolic syndrome consists of a cluster of metabolic abnormalities associated with obesity and that contribute to an increased risk of cardiovascular disease and type 2 diabetes. The syndrome is diagnosed when a patient has three or more of the following five risk factors: (1) abdominal obesity, (2) high triglyceride level, (3) low HDL cholesterol level, (4) hypertension, and (5) an elevated fasting blood glucose level. Table 15.4-8 lists the criteria as set forth by the World Health Organization (WHO). The syndrome is believed to occur in about 30 percent of the American population, but is also well known in other industrialized countries around the world.

Table 15.4-8 World Health Organization Clinical Criteria for Metabolic Syndrome The cause of the syndrome is unknown, but obesity, insulin resistance, and a genetic vulnerability are involved. Treatment involves weight loss, exercise, and the use of statins and antihypertensives as needed to lower lipid levels and blood pressure, respectively. Because of the increased risk of mortality, it is important that the syndrome be recognized early and treated. Second-generation (atypical) antipsychotic medications have been implicated as a cause of metabolic syndrome. In patients with schizophrenia, treatment with these medications can cause a rapid increase in body weight in the first few months of therapy, which may continue on for more than a year. In addition, insulin resistance leading to type 2 diabetes has been associated with an atherogenic lipid profile. Clozapine (Clozaril) and olanzapine (Zyprexa) are the two drugs most implicated, but other atypical antipsychotics may also be involved. Patients prescribed second-generation antipsychotic medications should be monitored periodically with hemoglobin A1c, fasting blood glucose levels at the beginning of treatment and during its course. Lipid profiles should also be obtained. Table 15.4-9 lists screening procedures for patients taking these medications. Table 15.4-9 Screen Patients Before Prescribing Antipsychotics

Psychological reactions to the metabolic syndrome depend on the signs and symptoms experienced by the patient. Those who suffer primarily from obesity must deal with selfesteem issues from being overweight as well as the stress of participating in weight loss programs. In many cases of obesity, eating is a way of satisfying deep-seated dependency needs. As weight is lost, some patients become depressed or anxious. Cases of psychosis have been reported in a few markedly obese patients during or after the process of losing a vast amount of weight. Other metabolic discrepancies, particularly variations in blood sugar, may be accompanied by irritability or other mood changes. Finally, fatigue is a common occurrence in patients with this syndrome. As the condition improves, especially if exercise is part of the regimen, fatigue eventually diminishes; but patients may be misdiagnosed as having a dysthymic disorder or chronic fatigue syndrome if metabolic causes of fatigue are not considered. REFERENCES Abraham S, Rubino D, Sinaii N,

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