20 - 15 Feeding and Eating Disorders

01 - 15.1 Anorexia Nervosa

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Feeding and Eating Disorders 15.1 Anorexia Nervosa The term anorexia nervosa is derived from the Greek term for “loss of appetite” and a Latin word implying nervous origin. Anorexia nervosa is a syndrome characterized by three essential criteria. The first is a self-induced starvation to a significant degree—a behavior. The second is a relentless drive for thinness or a morbid fear of fatness—a psychopathology. The third criterion is the presence of medical signs and symptoms resulting from starvation—a physiological symptomatology. Anorexia nervosa is often, but not always, associated with disturbances of body image, the perception that one is distressingly large despite obvious medical starvation. The distortion of body image is disturbing when present, but not pathognomic, invariable, or required for diagnosis. Two subtypes of anorexia nervosa exist: restricting and binge/purge. The theme in all anorexia nervosa subtypes is the highly disproportionate emphasis placed on thinness as a vital source, sometimes the only source, of self-esteem, with weight, and to a lesser degree, shape, becoming the overriding and consuming daylong preoccupation of thoughts, mood, and behaviors. Approximately half of anorexic persons will lose weight by drastically reducing their total food intake. The other half of these patients will not only diet but will also regularly engage in binge eating followed by purging behaviors. Some patients routinely purge after eating small amounts of food. Anorexia nervosa is much more prevalent in females than in males and usually has its onset in adolescence. Hypotheses of an underlying psychological disturbance in young women with the disorder include conflicts surrounding the transition from girlhood to womanhood. Psychological issues related to feelings of helplessness and difficulty establishing autonomy have also been suggested as contributing to the development of the disorder. Bulimic symptoms can occur as a separate disorder (bulimia nervosa, which is discussed in Section 15.2) or as part of anorexia nervosa. Persons with either disorder are excessively preoccupied with weight, food, and body shape. The outcome of anorexia nervosa varies from spontaneous recovery to a waxing and waning course to death. EPIDEMIOLOGY Anorexia nervosa has been reported more frequently over the past several decades, with increasing reports of the disorder in prepubertal girls and in boys. The most common ages of onset of anorexia nervosa are the midteens, but up to 5 percent of anorectic patients have the onset of the disorder in their early 20s. The most common age of onset

is between 14 and 18 years. Anorexia nervosa is estimated to occur in about 0.5 to 1 percent of adolescent girls. It occurs 10 to 20 times more often in females than in males. The prevalence of young women with some symptoms of anorexia nervosa who do not meet the diagnostic criteria is estimated to be close to 5 percent. Although the disorder was initially reported most often among the upper classes, recent epidemiological surveys do not show that distribution. It seems to be most frequent in developed countries, and it may be seen with greatest frequency among young women in professions that require thinness, such as modeling and ballet. COMORBIDITY Table 15.1-

1 lists comorbid psychiatric conditions associated with anorexia nervosa. Overall, anorexia nervosa is associated with depression in 65 percent of cases, social phobia in 35 percent of cases, and obsessive-compulsive disorder in 25 percent of cases. Table 15.1-1 Comorbid Psychiatric Conditions Associated with Anorexia Nervosa

ETIOLOGY Biological, social, and psychological factors are implicated in the causes of anorexia nervosa. Some evidence points to higher concordance rates in monozygotic twins than in dizygotic twins. Sisters of patients with anorexia nervosa are likely to be afflicted, but this association may reflect social influences more than genetic factors. Major mood disorders are more common in family members than in the general population. Neurochemically, diminished norepinephrine turnover and activity are suggested by reduced 3-methoxy-4-hydroxyphenylglycol (MHPG) levels in the urine and the cerebrospinal fluid (CSF) of some patients with anorexia nervosa. An inverse relation is seen between MHPG and depression in these patients; an increase in MHPG is associated with a decrease in depression. Biological Factors Endogenous opioids may contribute to the denial of hunger in patients with anorexia nervosa. Preliminary studies show dramatic weight gains in some patients who are given opiate antagonists. Starvation results in many biochemical changes, some of which are also present in depression, such as hypercortisolemia and nonsuppression by dexamethasone. Thyroid function is suppressed as well. These abnormalities are corrected by realimentation. Starvation may produce amenorrhea, which reflects lowered hormonal levels (luteinizing, follicle-stimulating, and gonadotropin-releasing hormones). Some patients with anorexia nervosa, however, may become amenorrheic before significant weight loss. Several computed tomographic (CT) studies reveal enlarged CSF spaces (enlarged sulci and ventricles) in anorectic patients during starvation, a finding that is reversed by weight gain. In one positron emission tomographic (PET) scan study, caudate nucleus metabolism was higher in the anorectic state than after realimentation. Some authors have proposed a hypothalamic-pituitary axis (neuroendocrine) dysfunction. Some studies have shown evidence for dysfunction in serotonin, dopamine, and norepinephrine, three neurotransmitters involved in regulating eating behavior in the paraventricular nucleus of the hypothalamus. Other humoral factors that may be involved include corticotropin-releasing factor (CRF), neuropeptide Y, gonadotropin-releasing hormone, and thyroid-stimulating hormone. Table 15.1-2 lists the neuroendocrine changes associated with anorexia nervosa. Table 15.1-2 Neuroendocrine Changes in Anorexia Nervosa and Experimental Starvation

Social Factors Patients with anorexia nervosa find support for their practices in society’s emphasis on thinness and exercise. No family constellations are specific to anorexia nervosa, but some evidence indicates that these patients have close, but troubled, relationships with their parents. Families of children who present with eating disorders, especially binge eating or purging subtypes, may exhibit high levels of hostility, chaos, and isolation and low levels of nurturance and empathy. An adolescent with a severe eating disorder may tend to draw attention away from strained marital relationships. Vocational and avocational interests interact with other vulnerability factors to increase the probability of developing eating disorders. In young women, participation in strict ballet schools increases the probability of developing anorexia nervosa at least sevenfold. In high school boys, wrestling is associated with a prevalence of full or partial eating-disorder syndromes during wrestling season of approximately 17 percent, with a minority developing an eating disorder and not improving spontaneously at the end of training. Although these athletic activities probably select for perfectionistic and persevering youth in the first place, pressures regarding weight and

shape generated in these social milieus reinforce the likelihood that these predisposing factors will be channeled toward eating disorders. A gay orientation in men is a proved predisposing factor, not because of sexual orientation or sexual behavior per se, but because norms for slimness, albeit muscular slimness, are very strong in the gay community, only slightly lower than for heterosexual women. In contrast, a lesbian orientation may be slightly protective, because lesbian communities may be more tolerant of higher weights and a more normative natural distribution of body shapes than their heterosexual female counterparts. Psychological and Psychodynamic Factors Anorexia nervosa appears to be a reaction to the demand that adolescents behave more independently and increase their social and sexual functioning. Patients with the disorder substitute their preoccupations, which are similar to obsessions, with eating and weight gain for other, normal adolescent pursuits. These patients typically lack a sense of autonomy and

selfhood. Many experience their bodies as somehow under the control of their parents, so that self-starvation may be an effort to gain validation as a unique and special person. Only through acts of extraordinary self-discipline can an anorectic patient develop a sense of autonomy and selfhood. Psychoanalytic clinicians who treat patients with anorexia nervosa generally agree that these young patients have been unable to separate psychologically from their mothers. The body may be perceived as though it were inhabited by the introject of an intrusive and unempathic mother. Starvation may unconsciously mean arresting the growth of this intrusive internal object and thereby destroying it. Often, a projective identification process is involved in the interactions between the patient and the patient’s family. Many anorectic patients feel that oral desires are greedy and unacceptable; therefore, these desires are projectively disavowed. Other theories have focused on fantasies of oral impregnation. Parents respond to the refusal to eat by becoming frantic about whether the patient is actually eating. The patient can then view the parents as the ones who have unacceptable desires and can projectively disavow them; that is, others may be voracious and ruled by desire but not the patient. DIAGNOSIS AND CLINICAL FEATURES The onset of anorexia nervosa usually occurs between the ages of 10 and 30 years. It is present when (1) an individual voluntarily reduces and maintains an unhealthy degree of weight loss or fails to gain weight proportional to growth; (2) an individual experiences an intense fear of becoming fat, has a relentless drive for thinness despite obvious medical starvation, or both; (3) an individual experiences significant starvationrelated medical symptomatology, often, but not exclusively, abnormal reproductive hormone functioning, but also hypothermia, bradycardia, orthostasis, and severely reduced body fat stores; and (4) the behaviors and psychopathology are present for at least 3 months. The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) diagnostic criteria for anorexia nervosa are given in Table 15.1-3. Table 15.1-3 DSM-5 Diagnostic Criteria for Anorexia Nervosa

An intense fear of gaining weight and becoming obese is present in all patients with the disorder and undoubtedly contributes to their lack of interest in, and even resistance to, therapy. Most aberrant behavior directed toward losing weight occurs in secret. Patients with anorexia nervosa usually refuse to eat with their families or in public places. They lose weight by drastically reducing their total food intake, with a disproportionate decrease in high-carbohydrate and fatty foods. As mentioned, the term anorexia is a misnomer, because loss of appetite is usually rare until late in the disorder. Evidence that patients are constantly thinking about food is their passion for collecting recipes and for preparing elaborate meals for others. Some patients cannot continuously control their voluntary restriction of food intake and so have eating binges. These binges usually

occur secretly and often at night and are frequently followed by self-induced vomiting. Patients abuse laxatives and even diuretics to lose weight, and ritualistic exercising, extensive cycling, walking, jogging, and running are common activities.

Patients with the disorder exhibit peculiar behavior about food. They hide food all over the house and frequently carry large quantities of candies in their pockets and purses. While eating meals, they try to dispose of food in their napkins or hide it in their pockets. They cut their meat into very small pieces and spend a great deal of time rearranging the pieces on their plates. If the patients are confronted with their peculiar behavior, they often deny that their behavior is unusual or flatly refuse to discuss it. Obsessive-compulsive behavior, depression, and anxiety are other psychiatric symptoms of anorexia nervosa most frequently noted clinically. Patients tend to be rigid and perfectionist, and somatic complaints, especially epigastric discomfort, are usual. Compulsive stealing, usually of candies and laxatives but occasionally of clothes and other items, may occur. Poor sexual adjustment is frequently described in patients with the disorder. Many adolescent patients with anorexia nervosa have delayed psychosocial sexual development; in adults, a markedly decreased interest in sex often accompanies onset of the disorder. A minority of anorexic patients has a premorbid history of promiscuity, substance abuse, or both but during the disorder show a decreased interest in sex. Patients usually come to medical attention when their weight loss becomes apparent. As the weight loss grows profound, physical signs such as hypothermia (as low as 35°C), dependent edema, bradycardia, hypotension, and lanugo (the appearance of neonatallike hair) appear, and patients show a variety of metabolic changes (Fig. 15.1-1). Some female patients with anorexia nervosa come to medical attention because of amenorrhea, which often appears before their weight loss is noticeable. Some patients induce vomiting or abuse purgatives and diuretics; such behavior causes concern about hypokalemic alkalosis. Impaired water diuresis may be noted. FIGURE 15.1-1 A patient with anorexia nervosa. (Courtesy of Katherine Halmi, M.D.)

Electrocardiographic (ECG) changes, such as T wave flattening or inversion, ST segment depression, and lengthening of the QT interval, have been noted in the emaciated stage of anorexia nervosa. ECG changes may also result from potassium loss, which can lead to death. Gastric dilation is a rare complication of anorexia nervosa. In some patients, aortography has shown a superior mesenteric artery syndrome. Other medical complications of eating disorders are listed in Table 15.1-4. Table 15.1-4 Medical Complications of Eating Disorders SUBTYPES Anorexia nervosa has been divided into two clinical subtypes: the food-restricting category and the purging category. In the food-restricting category, present in approximately 50 percent of cases, food intake is highly restricted (usually with attempts to consume fewer than 300 to 500 calories per day and no fat grams), and the patient may be relentlessly and compulsively overactive, with overuse athletic injuries. In the purging subtype, patients alternate attempts at rigorous dieting with intermittent binge or purge episodes. Purging represents a secondary compensation for the unwanted calories, most often accomplished by self-induced vomiting, frequently by laxative abuse, less frequently by diuretics, and occasionally with emetics. Sometimes, repetitive purging occurs without prior binge eating, after ingesting only relatively few calories. Both types may be socially isolated and have depressive disorder symptoms and diminished sexual interest. Overexercising and perfectionistic traits are also common in

both types. A new diagnostic category in DSM-5 is binge eating disorder (see Section 15.3) characterized by episodic bouts of the intake of excessive amounts of food but without purging or

similar compensatory behavior. Those who practice binge eating and purging share many features with persons who have bulimia nervosa without anorexia nervosa. Those who binge eat and purge tend to have families in which some members are obese, and they themselves have histories of heavier body weights before the disorder than do persons with the restricting type. Binge eating–purging persons are likely to be associated with substance abuse, impulse control disorders, and personality disorders. Persons with restricting anorexia nervosa often have obsessive-compulsive traits with respect to food and other matters. Some persons with anorexia nervosa may purge but not binge. Persons with anorexia nervosa have high rates of comorbid major depressive disorders; major depressive disorder or dysthymic disorder has been reported in up to 50 percent of patients with anorexia nervosa. The suicide rate is higher in persons with the binge eating–purging type of anorexia nervosa than in those with the restricting type. Patients with anorexia nervosa are often secretive, deny their symptoms, and resist treatment. In almost all cases, relatives or intimate acquaintances must confirm a patient's history. The mental status examination usually shows a patient who is alert and knowledgeable on the subject of nutrition and who is preoccupied with food and weight. A patient must have a thorough general physical and neurological examination. If the patient is vomiting, a hypokalemic alkalosis may be present. Because most patients are dehydrated, serum electrolyte levels must be determined initially and periodically. Hospitalization may be necessary to deal with medical complications. A young woman who weighed 10 percent above the average weight but was otherwise healthy, functioning well, and working hard as a university student joined a track team, started training for hours a day, more than her teammates, began to perceive herself as fat and thought that her performance would be enhanced if she lost weight. She started to diet and reduced her weight to 87 percent of the "ideal weight" for her age according to standard tables. At her point of maximum weight loss, her performance actually declined, and she pushed herself even harder in her training regimen. She started to feel apathetic and morbidly afraid of becoming fat. Her food intake became restricted and she stopped eating anything containing fat. Her menstrual periods became skimpy and infrequent but did not cease. (Courtesy of Arnold E. Andersen, M.D., and Joel Yager, M.D.) PATHOLOGY AND LABORATORY EXAMINATION A complete blood count often reveals leukopenia with a relative lymphocytosis in emaciated patients with anorexia nervosa. If binge eating and purging are present,

serum electrolyte determination reveals hypokalemic alkalosis. Fasting serum glucose concentrations are often low during the emaciated phase, and serum salivary amylase concentrations are often elevated if the patient is vomiting. The ECG may show ST segment and T-wave changes, which are usually secondary to electrolyte disturbances; emaciated patients have hypotension and bradycardia. Young girls may have a high serum cholesterol level. All these values revert to normal with nutritional rehabilitation and cessation of purging behaviors. Endocrine changes that may occur, such as amenorrhea, mild hypothyroidism, and hypersecretion of corticotrophin-releasing hormone, are caused by the underweight condition and revert to normal with weight gain. DIFFERENTIAL DIAGNOSIS The differential diagnosis of anorexia nervosa is complicated by patients' denial of the symptoms, the secrecy surrounding their bizarre eating rituals, and their resistance to seeking treatment. Thus, it may be difficult to identify the mechanism of weight loss and the patient's associated ruminative thoughts about distortions of body image. Clinicians must ascertain that a patient does not have a medical illness that can account for the weight loss (e.g., a brain tumor or cancer). Weight loss, peculiar eating behaviors, and vomiting can occur in several mental disorders. Depressive disorders and anorexia nervosa

have several features in common, such as depressed feelings, crying spells, sleep disturbance, obsessive ruminations, and occasional suicidal thoughts. The two disorders, however, have several distinguishing features. Generally, a patient with a depressive disorder has decreased appetite, whereas a patient with anorexia nervosa claims to have normal appetite and to feel hungry; only in the severe stages of anorexia nervosa do patients actually have decreased appetite. In contrast to depressive agitation, the hyperactivity seen in anorexia nervosa is planned and ritualistic. The preoccupation with recipes, the caloric content of foods, and the preparation of gourmet feasts is typical of patients with anorexia nervosa but is absent in patients with a depressive disorder. In depressive disorders, patients have no intense fear of obesity or disturbance of body image. Weight fluctuations, vomiting, and peculiar food handling may occur in somatization disorder. On rare occasions, a patient fulfills the diagnostic criteria for both somatization disorder and anorexia nervosa; in such a case, both diagnoses should be made. Generally, the weight loss in somatization disorder is not as severe as that in anorexia nervosa, nor does a patient with somatization disorder express a morbid fear of becoming overweight, as is common in those with anorexia nervosa. Amenorrhea for 3 months or longer is unusual in somatization disorder. In patients with schizophrenia, delusions about food are seldom concerned with caloric content. More likely, they believe the food to be poisoned. Patients with schizophrenia are rarely preoccupied with a fear of becoming obese and do not have the hyperactivity that is seen in patients with anorexia nervosa. Patients with schizophrenia have bizarre eating habits but not the entire syndrome of anorexia nervosa.

Anorexia nervosa must be differentiated from bulimia nervosa, a disorder in which episodic binge eating, followed by depressive moods, self-deprecating thoughts, and selfinduced vomiting occur while patients maintain their weight within a normal range. Patients with bulimia nervosa seldom lose 15 percent of their weight, but the two conditions frequently coexist. Rare conditions of unknown etiology are seen in which hyperactivity of the vagus nerve causes changes in eating patterns that are associated with weight loss, sometimes of severe degree. In such cases bradycardia, hypotension, and other parasympathomimetic signs and symptoms are seen. Because the vagus nerve relates to the enteric nervous system, eating may be associated with gastric distress such as nausea or bloating. Patients do not generally lose their appetite. Treatment is symptomatic and anticholinergic drugs can reverse hypotension and bradycardia, which may be lifethreatening. COURSE AND PROGNOSIS The course of anorexia nervosa varies greatly—spontaneous recovery without treatment, recovery after a variety of treatments, a fluctuating course of weight gains followed by relapses, and a gradually deteriorating course resulting in death caused by complications of starvation. One study reviewing subtypes of anorectic patients found that restricting-type anorectic patients seemed less likely to recover than those of the binge eating–purging type. The short-term response of patients to almost all hospital treatment programs is good. Those who have regained sufficient weight, however, often continue their preoccupation with food and body weight, have poor social relationships, and exhibit depression. In general, the prognosis is not good. Studies have shown a range of mortality rates from 5 to 18 percent. Indicators of a favorable outcome are admission of hunger, lessening of denial and immaturity, and improved self-esteem. Such factors as childhood neuroticism, parental conflict, bulimia nervosa, vomiting, laxative abuse, and various behavioral manifestations (e.g., obsessive-compulsive, hysterical, depressive, psychosomatic, neurotic, and denial symptoms) have been related to poor outcome in some studies, but not in others. Ten-year outcome studies in the United States have shown that about one fourth of patients recover completely and another one

half are markedly improved and functioning fairly well. The other one fourth includes an overall 7 percent mortality rate and those who are functioning poorly with a chronic underweight condition. Swedish and English studies over a 20- and 30-year period show a mortality rate of 18 percent. About half of patients with anorexia nervosa eventually will have the symptoms of bulimia, usually within the first year after the onset of anorexia nervosa. TREATMENT In view of the complicated psychological and medical implications of anorexia nervosa, a comprehensive treatment plan, including hospitalization when necessary and both

individual and family therapy, is recommended. Behavioral, interpersonal, and cognitive approaches are used and, in many cases, medication may be indicated. Hospitalization The first consideration in the treatment of anorexia nervosa is to restore patients' nutritional state; dehydration, starvation, and electrolyte imbalances can seriously compromise health and, in some cases, lead to death. The decision to hospitalize a patient is based on the patient's medical condition and the amount of structure needed to ensure patient cooperation. In general, patients with anorexia nervosa who are 20 percent below the expected weight for their height are recommended for inpatient programs, and patients who are 30 percent below their expected weight require psychiatric hospitalization for 2 to 6 months. Inpatient psychiatric programs for patients with anorexia nervosa generally use a combination of a behavioral management approach, individual psychotherapy, family education and therapy, and, in some cases, psychotropic medications. Successful treatment is promoted by the ability of staff members to maintain a firm yet supportive approach to patients, often through a combination of positive reinforcers (praise) and negative reinforcers (restriction of exercise). The program must have some flexibility for individualizing treatment to meet patients' needs and cognitive abilities. Patients must become willing participants for treatment to succeed in the long run. Most patients are uninterested in psychiatric treatment and even resist it; they are brought to a doctor's office unwillingly by agonizing relatives or friends. The patients rarely accept the recommendation of hospitalization without arguing and criticizing the proposed program. Emphasizing the benefits, such as relief of insomnia and depressive signs and symptoms, may help persuade the patients to admit themselves willingly to the hospital. Relatives' support and confidence in the physicians and treatment team are essential when firm recommendations must be carried out. Patients' families should be warned that the patients will resist admission and, for the first several weeks of treatment, will make many dramatic pleas for their families' support to obtain release from the hospital program. Compulsory admission or commitment should be obtained only when the risk of death from the complications of malnutrition is likely. On rare occasions, patients prove that the doctor's statements about the probable failure of outpatient treatment are wrong. They may gain a specified amount of weight by the time of each outpatient visit, but such behavior is uncommon, and a period of inpatient care is usually necessary. Hospital Management. The following considerations apply to the general management of patients with anorexia nervosa during a hospitalized treatment program. Patients should be weighed daily, early in the morning after emptying the bladder. The daily fluid intake and urine output should be recorded. If vomiting is occurring, hospital staff members must monitor serum electrolyte levels regularly and watch for the development of hypokalemia. Because food is often regurgitated after

meals, the staff may be able to control vomiting by making the bathroom inaccessible for at least 2 hours after meals or by having an attendant in the bathroom to prevent the opportunity for vomiting. Constipation in these patients is relieved when they begin to eat normally. Stool

softeners may occasionally be given, but never laxatives. If diarrhea occurs, it usually means that patients are surreptitiously taking laxatives. Because of the rare complication of stomach dilation and the possibility of circulatory overload when patients immediately start eating an enormous number of calories, the hospital staff should give patients about 500 calories over the amount required to maintain their present weight (usually 1,500 to 2,000 calories a day). It is wise to give these calories in six equal feedings throughout the day, so that patients need not eat a large amount of food at one sitting. Giving patients a liquid food supplement such as Sustagen may be advisable, because they may be less apprehensive about gaining weight slowly with the formula than by eating food. After patients are discharged from the hospital, clinicians usually find it necessary to continue outpatient supervision of the problems identified in the patients and their families. Psychotherapy Cognitive-Behavioral Therapy. Cognitive and behavioral therapy principles can be applied in both inpatient and outpatient settings and have been found effective for inducing weight gain. Monitoring is an essential component of cognitive-behavioral therapy. Patients are taught to monitor their food intake, their feelings and emotions, their binging and purging behaviors, and their problems in interpersonal relationships. Patients are taught cognitive restructuring to identify automatic thoughts and to challenge their core beliefs. Problem solving is a specific method whereby patients learn how to think through and devise strategies to cope with their food-related and interpersonal problems. Patients' vulnerability to rely on anorectic behavior as a means of coping can be addressed if they can learn to use these techniques effectively. Dynamic Psychotherapy. Dynamic expressive-supportive psychotherapy is sometimes used in the treatment of patients with anorexia nervosa, but their resistance may make the process difficult and painstaking. Because patients view their symptoms as constituting the core of their specialness, therapists must avoid excessive investment in trying to change their eating behavior. The opening phase of the psychotherapy process must be geared toward building a therapeutic alliance. Patients may experience early interpretations as though someone else were telling them what they really feel and thereby minimizing and invalidating their own experiences. Therapists who empathize with patients' points of view and take an active interest in what their patients think and feel, however, convey to patients that their autonomy is respected. Above all, psychotherapists must be flexible, persistent, and durable in the face of patients' tendencies to defeat any efforts to help them. Family Therapy. A family analysis should be done for all patients with anorexia

nervosa who are living with their families, which is used as a basis for a clinical judgment on what type of family therapy or counseling is advisable. In some cases, family therapy is not possible; however, issues of family relationships can then be addressed in individual therapy. Sometimes, brief counseling sessions with immediate family members is the extent of family therapy required. In one controlled family therapy study in London, anorectic patients under the age of 18 benefited from family therapy, whereas patients over the age of 18 did worse in family therapy than with the control therapy. No controlled studies have been reported on the combination of individual and family therapy; however, in actual practice, most clinicians provide individual therapy and some form of family counseling in managing patients with anorexia nervosa. Pharmacotherapy Pharmacological studies have not yet identified any medication that yields definitive improvement of the core symptoms of anorexia nervosa. Some reports support the use of cyproheptadine (Periactin), a drug with antihistaminic and antiserotonergic properties, for patients with the restricting type of anorexia nervosa. Amitriptyline (Elavil) has also been reported to have some benefit. Other medications that have been tried by patients with anorexia nervosa with variable results include clomipramine (Anafranil), pimozide (Orap), and chlorpromazine (Thorazine). Trials of

fluoxetine (Prozac) have resulted in some reports of weight gain, and serotonergic agents may yield positive responses in some cases. In patients with anorexia nervosa and coexisting depressive disorders, the depressive condition should be treated. Concern exists about the use of tricyclic drugs in low-weight, depressed patients with anorexia nervosa, who may be vulnerable to hypotension, cardiac arrhythmia, and dehydration. Once an adequate nutritional status has been attained, the risk of serious adverse effects from the tricyclic drugs may decrease; in some patients, the depression improves with weight gain and normalized nutritional status. REFERENCES

Andersen AE, Yager J. Eating disorders. In: Sadock BJ, Sadock VA, Ruiz P, eds. Kaplan & Sadock's Comprehensive Textbook of Psychiatry. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 2009:2128. Birmingham CL, Treasure J. Medical Management of Eating Disorders. 2nd ed. New York: Cambridge University Press; 2010. Blechert J, Ansorge U, Tuschen-Caffier B. A body-related dot-probe task reveals distinct attentional patterns for bulimia nervosa and anorexia nervosa. J Abnorm Psychol. 2010;119:575. Brown LM, Clegg DJ. Estrogen and leptin regulation of endocrinological features of anorexia nervosa. Neuropsychopharmacol Rev. 2013;38:237. Engel SG, Wonderlich SA, Crosby RD, Mitchell JE, Crow S, Peterson CB, Le Grange D, Simonich HK, Cao L, Lavender JM, Gordon KH. The role of affect in the maintenance of anorexia nervosa: Evidence from a naturalistic assessment of momentary behaviors and emotion. J Abnorm Psychol. 2013;122(3):709–719. Fallon P, Wisniewski L. A system of evidenced-based techniques and collaborative clinical interventions with a chronically

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ill patient. Int J Eat Disord. 2013;46(5):501–506. Fazeli PK, Misra M, Goldstein M, Miller KK, Klibanski A. Fibroblast growth factor-21 may mediate growth hormone resistance in anorexia nervosa. J Clin Endocrinol Metab. 2010;95:369. Fladung AK, Grön G, Grammer K, Herrnberger B, Schilly E, Grasteit S, Wolf RC, Walter H, von Wietersheim J. A neural signature of anorexia nervosa in the ventral striatal reward system. Am J Psych. 2009;167:206. Frank GKW, Reynolds JR, Shott ME, Jappe L, Yang TT, Tregellas JR, O'Reilly RC. Anorexia nervosa and obesity are associated with opposite brain reward response. Neuropsychopharmacology. 2012;37:2031. Friederich HC, Herzog W. Cognitive-behavioral flexibility in anorexia nervosa. In: Adan RAH, Kaye WH, eds. Behavioral Neurobiology of Eating Disorders. New York: Springer; 2011:111. Germain N, Galusca B, Grouselle D, Frere D, Billard S, Epelbaum J, Estour B. Ghrelin and obestatin circadian levels differentiate bingeing-purging from restrictive anorexia nervosa. J Clin Endocrinol Metab. 2010;95:3057. Hay P. A systematic review of evidence for psychological treatments in eating disorders: 2005–2012. Int J Eat Disord. 2013;46(5):462–469. Kishi T, Kafantaris V, Sunday S, Sheridan EM, Correll CU. Are antipsychotics effective for the treatment of anorexia nervosa? Results from a systematic review and meta-analysis. J Clin Psychiatry. 2012;73:e757. Kumar KK, Tung S, Iqbal J. Bone loss in anorexia nervosa: Leptin, serotonin, and the sympathetic nervous system. Ann New York Acad Sci. 2010;1211:51. Locke J, Grange DL. Treatment Manual for Anorexia Nervosa. 2nd ed. New York: Guilford; 2013. Lopez C, Davies H, Tchanturia K. Neuropsychological inefficiences in anorexia nervosa targeted in clinical practice: The development of a module of cognitive remediation therapy. In: Fox J, Goss K, eds. Eating and Its Disorders. Hoboken, NJ: Wiley; 2012:185. Versini A, Ramoz N, Strat YL, Scherag S, Ehrlich S, Boni C, Hinney A, Hebebrand J, Romo L, Guelfi JD, Gorwood P. Estrogen receptor 1 gene (ESR1) is associated with restrictive anorexia nervosa. Neuropsychopharmacology. 2010;35:1818. Zipfel S, et al. Focal psychodynamic therapy, cognitive behaviour therapy, and optimised treatment as usual in outpatients with anorexia nervosa (ANTOP study): randomised controlled trial. Lancet. 2014;383(9912):127–137. 15.2 Bulimia Nervosa Bulimia nervosa is characterized by episodes of binge eating combined with inappropriate ways of stopping weight gain. Physical discomfort—for example, abdominal pain or nausea—terminates the binge eating, which is often followed by feelings of guilt, depression, or self-disgust. Unlike patients with anorexia nervosa, those with bulimia nervosa typically maintain a normal body weight. The term bulimia nervosa derives from the terms for "ox-hunger" in Greek and "nervous involvement" in Latin. For some patients, bulimia nervosa may represent a failed attempt at anorexia nervosa, sharing the goal of becoming very thin, but occurring in an individual less able to sustain prolonged semistarvation or severe hunger as consistently as classic restricting anorexia nervosa patients. For others, eating binges represent "breakthrough eating" episodes of giving in to hunger pangs generated by efforts to restrict eating so as to maintain a socially desirable level of thinness. Still others use binge eating as a means to self-medicate during times of emotional distress.

Regardless of the reason, eating binges provoke panic as individuals feel that their eating has been out of control. The unwanted binges lead to secondary attempts to avoid the feared weight gain by a variety of compensatory behaviors, such as purging or excessive exercise. EPIDEMIOLOGY Bulimia nervosa is more prevalent than anorexia nervosa. Estimates of bulimia nervosa range from 1 to 4 percent of young women. As with anorexia nervosa, bulimia nervosa is more common in women than in men, but its onset is often later in adolescence than that of anorexia nervosa. The onset may also occur in early adulthood. Approximately 20 percent of college women experience transient bulimic symptoms at some point during their college years. Although bulimia nervosa is often present in normal-weight young women, they sometimes have a history of obesity. In industrialized countries the prevalence is about 1 percent of the general population. In the United States, bulimia nervosa may be more prevalent among Hispanics and blacks than non-Hispanic whites. ETIOLOGY Biological Factors Some investigators have attempted to associate cycles of binging and purging with various neurotransmitters. Because antidepressants often benefit patients with bulimia nervosa and because serotonin has been linked to satiety, serotonin and norepinephrine have been implicated. Because plasma endorphin levels are raised in some bulimia nervosa patients who vomit, the feeling of well-being after vomiting that some of these patients experience may be mediated by raised endorphin levels. Increased frequency of bulimia nervosa is found in first-degree relatives of persons with the disorder. Recent research using functional magnetic resonance imaging (MRI) suggests that overeating in bulimia nervosa may result from an exaggerated perception of hunger signals related to sweet taste mediated by the right anterior insula area of the brain. Social Factors Patients with bulimia nervosa, as with those with anorexia nervosa, tend to be high achievers and to respond to societal pressures to be slender. As with anorexia nervosa patients, many patients with bulimia nervosa are depressed and have increased familial depression, but the families of patients with bulimia nervosa are generally less close and more conflictual than the families of those with anorexia nervosa. Patients with bulimia nervosa describe their parents as neglectful and rejecting. Psychological Factors Patients with bulimia nervosa, as with those with anorexia nervosa, have difficulties with adolescent demands, but patients with bulimia nervosa are more outgoing, angry, and impulsive than those with anorexia nervosa. Alcohol dependence, shoplifting, and emotional lability (including suicide attempts) are associated with bulimia nervosa. These patients generally experience their uncontrolled eating as more ego-dystonic than do patients with anorexia nervosa and so

seek help more readily. Patients with bulimia nervosa lack superego control and the ego strength of their counterparts with anorexia nervosa. Their difficulties in controlling their impulses are often manifested by substance dependence and self-destructive sexual relationships in addition to the binge eating and purging that characterize the disorder. Many patients with bulimia nervosa have histories of difficulties separating from caretakers, as manifested by the absence of transitional objects during their early childhood years. Some clinicians have observed that patients with bulimia nervosa use their own bodies as transitional objects. The struggle for separation from a maternal figure is played out in the ambivalence toward food; eating may represent a wish to fuse with the caretaker, and regurgitating may unconsciously express a wish for separation. DIAGNOSIS AND CLINICAL FEATURES Bulimia nervosa is present when (1) episodes of binge eating occurs relatively frequently (once a week or more) for at least 3 months; (2) compensatory behaviors are practiced after binge eating to prevent weight gain, primarily self-induced vomiting, laxative abuse, diuretics, enemas, abuse of emetics (80 percent of cases), and, less commonly, severe dieting and strenuous exercise (20 percent of cases); (3) weight is not severely lowered as in anorexia nervosa; and (4)

the patient has a morbid fear of fatness, a relentless drive for thinness, or both and a disproportionate amount of selfevaluation that depends on body weight and shape. When making a diagnosis of bulimia nervosa, clinicians should explore the possibility that the patient has experienced a brief or prolonged prior bout of anorexia nervosa, which is present in approximately half of those with bulimia nervosa. Binging usually precedes vomiting by about 1 year. The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) diagnostic criteria for bulimia nervosa are listed in Table 15.2-1. Table 15.2-1 DSM-5 Diagnostic Criteria for Bulimia Nervosa

Vomiting is common and is usually induced by sticking a finger down the throat, although some patients are able to vomit at will. Vomiting decreases the abdominal pain and the feeling of being bloated and allows patients to continue eating without fear of gaining weight. The acid content of vomitus can damage tooth enamel, a not uncommon finding in patients with the disorder. Depression, sometimes called postbinge anguish, often follows the episode. During binges, patients eat food that is sweet, high in calories, and generally soft or smooth textured, such as cakes and pastry. Some patients prefer bulky foods without regard to taste. The food is eaten secretly and rapidly and is sometimes not even chewed. Annie is a 26-year-old Dutch woman. She works as a nurse in a city hospital and lives alone. Annie would wake up at night, go to her kitchen, and start eating whatever food she could lay her hands on. She stopped only after an hour or two when she could find no more food. The bouts of overeating went on for 5 years until she consulted her general practitioner, who referred her to outpatient psychiatric treatment for a depression related to the eating spells. Annie’s spells of uncontrollable overeating were preceded by a feeling of severe tension and were followed by relaxation, although this was coupled with shame and despair. During the year before her referral, the frequency of the overeating spells had increased to two or three times a week. They usually appeared at night after just a few hours of sleep. After eating her way through whatever she could find, she would feel bloated but would not vomit. She tried to get rid of the food by taking large quantities of laxatives. Her weight was

unstable, but she managed to keep it within normal limits simply by fasting between the overeating spells. Annie despised obesity but had never really been slim. Her bouts of overeating made her feel increasingly low-spirited and despairing. She had even considered committing suicide by taking an overdose of the sleeping tablets that her general practitioner had prescribed because of her interrupted sleep. Annie managed to do her job adequately and had taken only a few days of sick leave. Annie was brought up in a village, where her father was a schoolteacher. After secondary school she trained as a nurse and had various jobs on geriatric wards. Annie had always been very sensitive and fearful of criticism and had low self-esteem. She tried hard to live up to expectations and felt frustrated by minor criticisms. She had been in love more than once, but never dared to become engaged because she feared rejection and possibly also because she feared a sexual relationship. She had only a few close friends because she had difficulty engaging in close relationships. She often felt tense and diffident in company. She avoided going to meetings or parties because she feared being criticized or rejected. On examination Annie appeared quiet and reticent. Her mood was mildly depressed, and she cried silently as she described her difficulties. No psychotic features were suspected. She was otherwise healthy and of average weight. She perceived her own weight to be slightly higher than the weight she would prefer. She said she was afraid of becoming obese. (Courtesy of International Statistical Classification of Diseases and Related Health Problems, 10th ed. Casebook) Most patients with bulimia nervosa are

within their normal weight range, but some may be underweight or overweight. These patients are concerned about their body image and their appearance, worried about how others see them, and concerned about their sexual attractiveness. Most are sexually active, compared with anorexia nervosa patients, who are not interested in sex. Pica and struggles during meals are sometimes revealed in the histories of patients with bulimia nervosa. Bulimia nervosa occurs in persons with high rates of mood disorders and impulse control disorders. Bulimia nervosa is also reported to occur in those at risk for substancerelated disorders and a variety of personality disorders. Patients with bulimia nervosa also have increased rates of anxiety disorders, bipolar I disorder, dissociative disorders, and histories of sexual abuse. Subtypes Evidence indicates that bulimic persons who purge differ from binge eaters who do not purge in that the latter tend to have less body-image disturbance and less anxiety concerning eating. Those with bulimia nervosa who do not purge tend to be obese. Distinct physiological differences also exist between patients with bulimia who purge and those who do not. Because of all these differences, the diagnosis of bulimia nervous is sometimes subtyped into a purging type, for those who regularly engage in self-induced vomiting or the use of laxatives or diuretics, and a nonpurging type, for those who use strict dieting, fasting, or vigorous exercise but do not regularly engage in purging. Patients who purge may have a different course from that of patients who binge and then diet or exercise.

Patients with purging type may be at risk for certain medical complications such as hypokalemia from vomiting or laxative abuse and hypochloremic alkalosis. Those who vomit repeatedly are at risk for gastric and esophageal tears, although these complications are rare. PATHOLOGY AND LABORATORY EXAMINATIONS Bulimia nervosa can result in electrolyte abnormalities and various degrees of starvation, although it may not be as obvious as in low-weight patients with anorexia nervosa. Thus, even normal-weight patients with bulimia nervosa should have laboratory studies of electrolytes and metabolism. In general, thyroid function remains intact in bulimia nervosa, but patients may show nonsuppression on a dexamethasonesuppression test. Dehydration and electrolyte disturbances are likely to occur in patients with bulimia nervosa who purge regularly. These patients commonly exhibit hypomagnesemia and hyperamylasemia. Although not a core diagnostic feature, many patients with bulimia nervosa have menstrual disturbances. Hypotension and bradycardia occur in some patients. DIFFERENTIAL DIAGNOSIS The diagnosis of bulimia nervosa cannot be made if the binge-eating and purging behaviors occur exclusively during episodes of anorexia nervosa. In such cases, the diagnosis is anorexia nervosa, binge eating-purging type. Clinicians must ascertain that patients have no neurological disease, such as epilepticequivalent seizures, central nervous system tumors, Klüver-Bucy syndrome, or KleineLevin syndrome. The pathological features manifested by Klüver-Bucy syndrome are visual agnosia, compulsive licking and biting, examination of objects by the mouth, inability to ignore any stimulus, placidity, altered sexual behavior (hypersexuality), and altered dietary habits, especially hyperphagia. The syndrome is exceedingly rare and is unlikely to cause a problem in differential diagnosis. Kleine-Levin syndrome consists of periodic hypersomnia lasting for 2 to 3 weeks and hyperphagia. As in bulimia nervosa, the onset is usually during adolescence, but the syndrome is more common in men than in women. Patients with bulimia nervosa who have concurrent seasonal affective disorder and patterns of atypical depression (with overeating and oversleeping in low-light months) may manifest seasonal worsening of both bulimia nervosa and depressive features. In these cases, binges are typically much more severe during winter months. Bright light therapy (10,000 lux for 30 minutes, in early morning, at 18 to 22 inches from the eyes) may be a useful component of comprehensive treatment of an eating disorder with seasonal affective disorder.

Some patients with bulimia nervosa—perhaps 15 percent—have multiple comorbid impulsive behaviors, including substance abuse, and lack of ability to control themselves in such diverse areas as money management (resulting in impulse buying and compulsive shopping) and sexual relationships (often resulting in brief, passionate attachments and promiscuity). They exhibit self-mutilation, chaotic emotions, and

chaotic sleeping patterns. They often meet criteria for borderline personality disorder and other mixed personality disorders and, not infrequently, bipolar II disorder. COURSE AND PROGNOSIS Bulimia nervosa is characterized by higher rates of partial and full recovery compared with anorexia nervosa. As noted in the treatment section, those treated fare much better than those who are untreated. Patients who are untreated tend to remain chronic or may show small, but generally unimpressive, degrees of improvement with time. In a 10-year follow-up study of patients who had previously participated in treatment programs, the number of women who continued to meet the full criteria for bulimia nervosa declined as the duration of follow-up increased. Approximately 30 percent continued to engage in recurrent binge-eating or purging behaviors. A history of substance use problems and a longer duration of the disorder at presentation predicted worse outcome. Approximately 40 percent of women were fully recovered at follow-up. The mortality rate for bulimia nervosa has been estimated at 2 percent per decade according to DSM-5. TREATMENT Most patients with uncomplicated bulimia nervosa do not require hospitalization. In general, patients with bulimia nervosa are not as secretive about their symptoms as patients with anorexia nervosa. Therefore, outpatient treatment is usually not difficult, but psychotherapy is frequently stormy and may be prolonged. Some obese patients with bulimia nervosa who have had prolonged psychotherapy do surprisingly well. In some cases—when eating binges are out of control, outpatient treatment does not work, or a patient exhibits such additional psychiatric symptoms as suicidality and substance abuse—hospitalization may become necessary. In addition, electrolyte and metabolic disturbances resulting from severe purging may necessitate hospitalization. Psychotherapy Cognitive-Behavioral Therapy. Cognitive-behavioral therapy (CBT) should be considered the benchmark, first-line treatment for bulimia nervosa. The data supporting the efficacy of CBT are based on strict adherence to rigorously implemented, highly detailed, manual-guided treatments that include about 18 to 20 sessions over 5 to 6 months. CBT implements a number of cognitive and behavioral procedures to (1) interrupt the self-maintaining behavioral cycle of binging and dieting and (2) alter the individual’s dysfunctional cognitions; beliefs about food, weight, body image; and overall self-concept. Dynamic Psychotherapy. Psychodynamic treatment of patients with bulimia nervosa has been of limited success. Psychodynamic formulations revealed a tendency to concretize introjective and projective defense mechanisms. In a manner analogous to

splitting, patients divide food into two categories: items that are nutritious and those that are unhealthy. Food that is designated nutritious may be ingested and retained because it unconsciously symbolizes good introjects. But junk food is unconsciously associated with bad introjects and, therefore, is expelled by vomiting, with the unconscious fantasy that all destructiveness, hate, and badness are being evacuated. Patients can temporarily feel good after vomiting because of the fantasized evacuation, but the associated feeling of “being all good” is short-lived because it is based on an unstable combination of splitting and projection. Other Modalities. Controlled trials have shown that a variety of novel ways of administering and facilitating cognitive-behavioral therapy are effective for bulimia nervosa. Some have been

incorporated in “stepped-care” programs and including Internet-based platforms, computer facilitated programs, email enhanced programs, and administration of cognitive-behavioral therapy via telemedicine to remote areas. Pharmacotherapy Antidepressant medications have been shown to be helpful in treating bulimia. This includes the selective serotonin reuptake inhibitors (SSRIs), such as fluoxetine (Prozac). This may be based on elevating central 5-hydroxytryptamine levels. Antidepressant medications can reduce binge eating and purging independent of the presence of a mood disorder. Thus, antidepressants have been used successfully for particularly difficult binge-purge cycles that do not respond to psychotherapy alone. Imipramine (Tofranil), desipramine (Norpramin), trazodone (Desyrel), and monoamine oxidase inhibitors (MAOIs) have been helpful. In general, most of the antidepressants have been effective at dosages usually given in the treatment of depressive disorders. Dosages of fluoxetine that are effective in decreasing binge eating, however, may be higher (60 to 80 mg a day) than those used for depressive disorders. Medication is helpful in patients with comorbid depressive disorders and bulimia nervosa. Carbamazepine (Tegretol) and lithium (Eskalith) have not shown impressive results as treatments for binge eating, but they have been used in the treatment of patients with bulimia nervosa with comorbid mood disorders, such as bipolar I disorder. Evidence indicates that the use of antidepressants alone results in a 22 percent rate of abstinence from binging and purging; other studies show that CBT and medications are the most effective combination. REFERENCES Andersen AE, Yager J. Eating disorders. In: Sadock BJ, Sadock VA, Ruiz P, eds. Kaplan & Sadock’s Comprehensive Textbook of Psychiatry. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 2009:2128. Glasner-Edwards S, Mooney LJ, Marinelli-Casey P, Ang A, Rawson R. Bulimia nervosa among methamphetamine dependent adults: Association with outcomes 3 years after treatment. Eat Disord. 2011;19:259. Hildebrandt T, Alfano L, Tricamo M, Pfaff DW. Conceptualizing the role of estrogens and serotonin in the development and maintenance of bulimia nervosa. Clin Psychol Rev. 2010;30:655.

03 - 15.3 Binge Eating Disorder and Other Eating D

15.3 Binge Eating Disorder and Other Eating Disorders

Levitan RD, Kaplan AS, Davis C, Lam RW, Kennedy JL. A season-of-birth/DRD4 interaction predicts maximal body mass index in women with bulimia nervosa. Neuropsychopharmacology. 2010;35:1729. Lowe MR, Witt AA, Grossman SL. Dieting in bulimia nervosa is associated with increased food restriction and psychopathology but decreased binge eating. Eat Behav. 2013;14(3):342–347. Oberndorfer TA, Frank GKW. Altered insula response to sweet taste processing after recovery from anorexia and bulimia nervosa. Am J Psychiatry. 2013;170(10):1143–1151. Poulsen, S., Lunn, S., Daniel, SI, Folke, S., Mathiesen, BB, Katznelson, H., Fairburn, CG. A randomized controlled trial of psychoanalytic psychotherapy or cognitive-behavioral therapy for bulimia nervosa. Am J Psychiatry. 2014;171(1):109– 116. Sandberg K, Erford BT. Choosing assessment instruments for bulimia practice and outcome research. J Counsel Dev. 2013;91(3):359–366. Wolfe BE, Hannon-Engel SL, Mitchell JE. Bulimia nervosa in DSM-5. Psych Annals. 2012;42:406. Zimmerli EJ, Devlin MJ, Kissileff HR, Walsh BT. The development of satiation in bulimia nervosa. Physiol Behav. 2010;100:346. Zunker C, Peterson CB, Crosby RD, Cao L, Engel SG, Mitchell JE, Wonderlich SA. Ecological momentary assessment of bulimia nervosa: Does dietary restriction predict binge eating? Behav Res Ther. 2011;49(10):714. 15.3 Binge Eating Disorder and Other Eating Disorders BINGE EATING DISORDER Individuals with binge eating disorder engage in recurrent binge eating during which they eat an abnormally large amount of food over a short time. Unlike bulimia nervosa, patients with binge eating disorder do not compensate in any way after a binge episode (e.g., laxative use). Binge episodes often occur in private, generally include foods of dense caloric content, and, during the binge, the person feels he or she cannot control his or her eating. Epidemiology Binge eating disorder is the most common eating disorder. It appears in approximately 25 percent of patients who seek medical care for obesity and in 50 to 75 percent of those with severe obesity (body mass index [BMI] greater than 40). It is more common in females (4 percent) than in males (2 percent). Etiology The cause of binge eating disorder is unknown. Impulsive and extroverted personality styles are linked to the disorder as are persons who place themselves on a very low calorie diet. Binge eating may also occur during periods of stress. It may be used to reduce anxiety or alleviate depressive moods. Diagnosis and Clinical Features

To be diagnosed with binge eating disorder the binges must be characterized by four features: (1) eating more rapidly than normal and to the point of being uncomfortably full, (2) eating large amounts of food even when not hungry, (3) eating alone, and (4) feeling guilty or otherwise upset about the episode. Binges must occur at least once a week for at least 3 months. The guidelines from the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders are presented in Table 15.3-1. Table 15.3-1 DSM-5 Diagnostic Criteria for Binge Eating Disorder

Approximately half of individuals with binge eating disorder are obese. Additionally, obese individuals with binge eating disorder have an earlier onset of obesity than those

without the disorder. Patients with binge eating disorder are also more likely to have an unstable weight history with frequent episodes of weight cycling (the gaining or losing of more than 10 kg). The disorder may be associated with insomnia, early menarche, neck or shoulder and lower back pain, chronic muscle pain, and metabolic disorders. Differential Diagnosis Binge eating disorder and bulimia nervosa share the same core feature of recurrent binge eating. Binge eating disorder is distinct from bulimia nervosa, however, in that binge eating disorder patients do not report recurrent compensatory behavior such as vomiting, laxative abuse, or excessive dieting. Binge eating disorder is distinct from anorexia nervosa in that patients do not exhibit an excessive drive for thinness and are of normal weight or are obese. The prevalence of binge eating disorder is higher in overweight populations (3 percent) than in the general population (approximately 2 percent). However there are some distinctions. Obese patients with binge eating disorder have a greater caloric intake during binging and nonbinging episodes, greater eating disorder pathology (i.e., more emotional eating, chaotic eating habits), and higher rates of comorbid psychiatric disorders. Binge eating disorder is also more prevalent in families than obesity. Course and Prognosis Little is known about the course of binge eating disorder. Severe obesity is a long-term effect in over 3 percent of patients with the disorder. One prospective study of women in the community with binge eating disorder suggested that by 5 years of follow-up fewer than one fifth of the sample still had clinically significant eating disorder symptoms. Treatment Psychotherapy. Cognitive-behavioral therapy (CBT) is the most effective psychological treatment for binge eating disorder. CBT has been shown to lead to decreases in binge eating and associated problems (e.g., depression); however, studies have not shown marked weight loss as a result of CBT, and CBT combined with psychopharmacological treatments such as selective serotonin reuptake inhibitors (SSRIs) show better results than CBT alone. Exercise has also shown a reduction in binge eating when combined with CBT. Interpersonal psychotherapy (IPT) has also shown to be effective in the treatment of binge eating disorder; however, therapy focuses more on the interpersonal problems that contribute to the disorder rather than disturbances in eating behavior. Self-Help Groups. Self-help groups such as Overeaters Anonymous (OA) have proven to be helpful in patients with binge eating disorder. For the treatment of moderate obesity, organizations such as Weight Watchers can be extremely helpful and

do not involve common fads or quick fixes. Psychopharmacotherapy. Symptoms of binge eating may benefit from medication treatment with several SSRIs, desipramine (Norpramin), imipramine (Tofranil), topiramate (Topamax), and sibutramine (Meridia). SSRI medications that have demonstrated improvement in mood as well as binge eating include fluvoxamine (Luvox), citalopram (Celexa), and sertraline (Zoloft). Some studies showed that highdose SSRI treatment (e.g., fluoxetine [Paxil] at 60 to 100 mg) often initially resulted in weight loss. However, the weight

loss was ordinarily short lived, even when medication was continued, and weight always returned when medication was discontinued. Amphetamine and amphetamine-like drugs may help but are of little use over the long term. Most, but not all, studies show that medication added to CBT is more effective than medication alone. For example, studies indicate that CBT did better than fluvoxamine or desipramine as a monotherapy for binge eating disorder; however, when CBT was used in combination with these agents, more improvement was seen in terms of weight loss compared with CBT alone. OTHER SPECIFIED FEEDING OR EATING DISORDER The diagnostic category of “other specified feeding or eating disorder” can be used for eating conditions that may cause significant distress but do not meet the full criteria for a classified eating disorder. Conditions included in this category include night eating syndrome, purging disorder, and subthreshold forms of anorexia nervosa, bulimia nervosa, and binge eating disorder. Night Eating Syndrome Night eating syndrome is characterized by the consumption of large amounts of food after the evening meal. Individuals generally have little appetite during the day and suffer from insomnia. Epidemiology. Night eating syndrome occurs in approximately 2 percent of the general population; however, it has a higher prevalence among patients with insomnia, obesity (10 to 15 percent), eating disorders, and other psychiatric disorders. The disorder usually begins in early adulthood. Etiology. Little is known about the cause of night eating disorder; however, the hormones melatonin, leptin, ghrelin, and cortisol have been studied in relation to the disorder. Night eating syndrome also appears to run in families; patients with night eating syndrome are five times more likely to have a first-degree relative with night eating syndrome. Diagnosis and Clinical Features

The diagnosis of night eating disorder includes recurrent episodes of hyperphagia or night eating; the lack of desire for food in the morning; and insomnia. Symptoms must persist for at least 3 months and cannot be secondary to another medical or mental condition. Patients with night eating syndrome usually consume a large portion of their daily calorie intake after the evening meal. They are also more likely to wake up during the night and to eat upon awakening. Nocturnal eating tends to occur during non-rapid eye movement (REM) sleep and is usually short in duration. Patients are also prone to low sleep efficiency. Patients believe that they can only sleep if they eat. Depressed mood is common among these patients, especially during the evening and night hours. Differential Diagnosis. Night eating disorder is common among patients with other eating disorders, particularly bulimia nervosa and binge eating disorder. Although night eating can be found in bulimia nervosa and binge eating disorder, it is the characteristic sign of night eating disorder. Also, the amount of food consumed during eating episodes is usually lower in night eating disorder than in bulimia nervosa and binge eating disorder. Unlike other eating disorders, patients with night eating syndrome are not overly concerned about body image and weight. Patients with night eating disorder are also at higher risk for obesity and metabolic syndrome. Sleep-related eating disorder is characterized by recurrent episodes of involuntary eating during the night. These episodes can lead to serious consequences such as the ingesting of nonedible foods or substances, dangerous behaviors while searching for or preparing food, and sleep-related injury. The eating episodes usually occur after the patient has gone to sleep and may occur while the patient is unconscious or asleep. Sleep-related eating disorder also has a high comorbidity with sleepwalking, restless leg syndrome, and obstructive sleep apnea, conditions that are rarely found among night eating syndrome patients. Episodes of sleep-related eating disorder have been reported after the use certain medications, including zolpidem (Ambien), triazolam (Halcion), olanzapine (Zyprexa), and risperidone (Risperdal). Course and Prognosis. The age of onset for night eating syndrome ranges from the late teens to late 20s and has a long-lasting course with periods of remission with

treatment. Patients who experience poor sleep quality are more likely to develop diabetes, obesity, hypertension, and cardiovascular disease. Treatment. Various studies have shown positive results in patients treated with SSRIs who showed improvement in nighttime awakenings, nocturnal eating, and postevening caloric intake. Weight loss and a reduction in nocturnal eating have been associated with an addition of topiramate to medication regimens. In patients with comorbid major depression and night eating syndrome, bright light therapy has shown to decrease depressed mood. CBT has also been helpful. Purging Disorder

04 - 15.4 Obesity and the Metabolic Syndrome

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, Ramsey S, Nieman L. Cortisol, obesity, and the metabolic syndrome: A cross-sectional study of obese subjects and review of the literature. Obesity. 2013;21(1):E105–E117. Adams TD, Davidson

LE, Litwin SE, Kolotkin RL, LaMonte MJ, Pendleton RC, Strong MB, Vinik R, Wanner NA, Hopkins PN, Gress RE, Walker JM, Cloward TV, Nuttall RT, Hammoud A, Greenwood JLJ, Crosby RD, McKinlay R, Simper SC, Smith SC, Hunt, SC. Health benefits of gastric bypass surgery after 6 years. JAMA. 2012;308(11):1122. Chugh PK, Sharma S. Recent advances in the pathophysiology and pharmacological treatment of obesity. J Clin Pharm Ther. 2012;37:525. Jurd R. TiNS special issue: Neural control of appetite. Trend Neurosci. 2013;36(2):63–64. Kabra DG, Kabra UD, Tschöp MH, Hofmann S. Pharmacological treatment of obesity. In: Shiromani P, Horvath T, Redline S, Van Cauter E, eds. Sleep Loss and Obesity: Intersecting Epidemics. New York: Springer; 2012:203. Landsberg L, Aronne LJ, Beilin LJ, Burke V, Igel LI, Lloyd-Jones D, Sowers J. Obesity-related hypertension: Pathogenesis, cardiovascular risk, and treatment—A position paper of the Obesity Society and the American Society of Hypertension. Obesity. 2013;21(1):8–24. Marcus MD, Wildes JE. Obesity in DSM-5. Psych Annal. 2012;42:431. Miller LE. Lorcaserin for weight loss: Insights into US Food and Drug Administration approval. J Acad Nutr Diet. 2013;113:25. Neovius M, Narbro K, Keating C, Peltonen M, Sjöholm K, Ågren G, Sjöström L, Carlsson L. Health care use during 20 years following bariatric surgery. JAMA. 2012;308(11):1132.

Palfreyman Z, Haycraft E, Meyer C. Unintentional role models: Links between maternal eating psychopathology and the modelling of eating behaviours. Eur Eat Disord Rev. 2013;21(3):195–201. Pike KM. Classification, culture, and complexity: A global look at the diagnosis of eating disorders: Commentary on Wildes and Marcus: Incorporating dimensions into the classification of eating disorders. Int J Eat Disord. 2013;46(5):408–411. Shen XL, Jia FJ, Song N, Xie JX, Jiang H. Protection of MES23. 5 dopaminergic cells by obestatin is mediated by proliferative rather than anti-apoptotic action. Neurosci Bull. 2014; 30(1), 118–124. Vaidya V, Steele KE, Schweitzer M, Shermack MA. Obesity. In: Sadock BJ, Sadock VA, Ruiz P, eds. Kaplan & Sadock’s Comprehensive Textbook of Psychiatry. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 2009:2273.