
Eating Disorders (Anorexia Nervosa, Bulimia Nervosa, Binge Eating Disorder)

Diane K. Smith and Christian R. Lemmon

Introduction

The pursuit of thinness among adolescent and young adult females may leave some of them quickly threatening their physiological and psychological wellbeing as the fear of weight gain and possible obesity drives them into maladaptive eating and dieting habits. The intense preoccupation with dieting and weight control among this segment of the population has become so commonplace that it is often viewed as normal behavior and revered by peers who consider themselves less successful at being able to control their weight and shape. Unfortunately, and sometimes tragically, these maladaptive behaviors may inevitably manifest themselves in the form of eating disorders.

Despite the attention afforded to the eating disorders by medicine and the media, anorexia nervosa (AN), bulimia nervosa (BN) and related feeding disturbances remain difficult psychiatric diagnoses to treat. The American Psychiatric Association¹ recently revised their practice guidelines for the treatment of patients with eating disorders, but practitioners who treat these patients, including psychiatrists and other physicians, psychologists, social workers, counselors, dietitians and other health personnel continue to approach these problems from a wide array of treatment perspectives. Part of this lack of consensus concerning the treatment of eating disorders is attributable to the abundance of theories regarding their etiology. As reported by Yager,² "theories regarding the etiology and pathogenesis of the eating disorders have implicated virtually every level of biopsychosocial organization." AN, BN, and related feeding disturbances challenge those who are devoted to their treatment.

Diagnostic Criteria

Eating disorders occur over a continuum of increasingly pathological behavior. Excessive self-evaluation and a preoccupation with weight, shape, and size characterize both AN and BN. Other common characteristics include an intense fear of weight gain and a

relationship with food that borders on obsession. However, there are distinct differences between the two disorders. Following are the diagnostic criteria for the recognized eating disorders AN and BN. A third eating disorder, Binge Eating Disorder (BED), is currently considered a proposed diagnosis for inclusion in the next revision of the Diagnostic and Statistical Manual, Version IV (DSM-IV) that standardizes psychiatric diagnosis. Other diagnoses and feeding disturbances, including Eating Disorder, Not Otherwise Specified (NOS), will be discussed below.

Anorexia Nervosa (AN)

Although AN often starts with only small reductions in total food intake, patients eventually reduce their energy and fat intake to a point where they are consuming only a limited number of foods in a highly ritualistic fashion. The disorder is characterized by severe, self-induced starvation (300 to 600 kcalories/day). However, an actual loss of appetite is quite rare. Individuals with AN refuse to maintain a minimally normal, healthy body weight, show an intense fear of gaining weight or becoming fat, exhibit a disturbance in their perception of their body weight or shape, and experience abnormal menses. Two subtypes of AN exist, with about 50% classified as the restricting type, and the others exhibiting behaviors indicative of binge-eating and purging. (AN and BN are not mutually exclusive diagnoses.) Further distinctions can be made between AN patients who binge eat and purge and those who purge normal meals or snacks but do not engage in binge-eating episodes. DSM-IV criteria for AN are outlined in Table 68.1.

TABLE 68.1

DSM-IV Criteria for Anorexia Nervosa, including Subtypes^a

Refusal to maintain body weight at or above 85% of expected for height and age
 Intense fear of gaining weight or accumulation of body fat despite underweight status
 Body image disturbance which may include the denial or lack of appreciation for the seriousness of one's currently low weight, self-evaluation largely determined by one's shape or weight, or claiming to "feel fat" even though terribly underweight
 In females, primary or secondary amenorrhea

Subtypes

Restricting type — person has not regularly engaged in binge eating or various purging behaviors
 Binge eating/purging type — person regularly engages in binge eating or various purging behaviors

^a Adapted from American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed., American Psychiatric Association, Washington, DC, 1994.

Bulimia Nervosa (BN)

BN is characterized by recurrent episodes of eating unusually large quantities of food in a finite period of time, often until the food is gone or the person is uncomfortably or painfully full. Efforts to purge the excess (3000 to 10,000 kcalories) by some compensatory or purging behavior such as vomiting, laxative or diuretic abuse, excessive exercise, and/or restrictive dieting or fasting occur subsequent to the binge eating episode. These behaviors are associated with a sense of loss of control and typically shame, guilt, and embarrassment are associated with the binge eating and purging process. Nevertheless, patients often report that the purging behaviors diminish the intensity of aversive emotions and provide them with a sense of control. Similar to the diagnosis of AN, patients are classified as either the purging type or nonpurging type. DSM-IV criteria for BN are outlined in [Table 68.2](#).

TABLE 68.2

DSM-IV Criteria for Bulimia Nervosa, including Subtypes^a

Recurrent episodes (a minimum average of twice per week for at least 3 months) of binge eating, defined as eating abnormally large amounts of food within a 2-hour period, that are associated with a sense of lack of control over the eating process during the episode
Use of compensatory or purging behavior such as self-induced vomiting, laxative/enema or diuretic abuse, restrictive dieting, fasting, or excessive exercise
Self-evaluation largely determined by one's shape and weight
Bulimic behavior does not occur exclusively as a manifestation of anorexia nervosa

Subtypes

Purging type — person regularly uses purging strategies including self-induced vomiting, laxatives, diuretics, or enemas

Nonpurging type — person regularly uses other purging strategies including fasting, restrictive dieting or excessive exercise, but not currently using strategies listed above

^a Adapted from American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed., American Psychiatric Association, Washington, DC, 1994.

Binge Eating Disorder

BED is more nebulous, and is characterized by recurrent binge-eating episodes without a compensatory effort to eliminate caloric excess. Although described in the classic paper of Stunkard,⁴ it has only recently been widely recognized. In fact, diagnostic criteria for BED were included in the DSM-IV only for research purposes, as it is not yet an approved diagnosis. Although persons diagnosed with BED are seen with a wide range of weights, most are obese. DSM-IV criteria for BED are outlined in Table 68.3.

TABLE 68.3

DSM-IV Proposed Criteria for Binge Eating Disorder^a

Recurrent episodes (a minimum average of twice per week for at least 6 months) of binge eating, defined as eating an abnormally large amount of food within a 2-hour period, that are associated with a sense of lack of control over the eating process during the episode

At least 3 of the following characteristics are present during binge eating:

Eating much more rapidly than usual

Eating until uncomfortably full

Eating large amounts of food despite not feeling physically hungry

Eating alone because of embarrassment over quantity of food consumed

Feeling disgusted, depressed, guilty, or ashamed after the binge

Experience of significant distress about the presence of binge eating

Absence of compensatory behaviors such as self-induced vomiting, laxative or diuretic abuse, restrictive dieting, fasting, or excessive exercise

Binge eating episodes do not occur exclusively as a manifestation of anorexia nervosa or bulimia nervosa.

^a Adapted from American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed., American Psychiatric Association, Washington, DC, 1994.

Other Eating Disorders (BED)

Many patients diagnosed with eating disorders do not satisfy criteria for a formal diagnosis of AN or BN. For example, they may not binge and purge often enough to meet the required minimum average occurrence of such behavior. Similarly, a patient's weight may remain in an acceptable or healthy range despite exhibiting all of the other symptoms of AN. Some patients may display all of the characteristics of AN, but continue to menstruate normally. Others may repeatedly chew their food and spit it out in an effort to avoid the ingestion of unwanted energy. Patients who exhibit these and other similar presentations are clas-

sified as Eating Disorder, NOS.³ However, the failure to meet formal criteria for AN or BN does not mean that such an individual does not have a serious disorder or does not require treatment. Many individuals engage in intermittent maladaptive dieting behaviors such as meal skipping, crash dieting, the avoidance of specific foods, excessive exercise, and abuse of drugs (especially appetite suppressants, caffeine, and other stimulants).

Compulsive overeating and night eating have not been formally accepted as eating disorders, but are other maladaptive eating patterns. Compulsive overeating reflects frequent meals and snacks, or continuous eating or “grazing” without the presence of purging behaviors. Usually these individuals become overweight. Night eating is especially common in morbidly obese subjects. It is characterized by delaying the first meal of the day, eating more food after dinner than during that meal, and eating more than half of the day’s food after dinner. The eating is often associated with aversive emotions and stress, and the pattern has persisted for at least two months. Nocturnal sleep-related eating is a sleep disorder. Sufferers are in a state somewhere between sleep and wakefulness and do not have recollection of the eating episodes. Another disorder often confused with BN is psychogenic vomiting, which is involuntary postprandial regurgitation in the absence of the core symptoms of fear of weight gain, binge eating, and body image disturbance.

Epidemiology

Eating disorders occur primarily in adolescent and young adult caucasian women, but the onset can be prior to menarche or later in life. Males and non-caucasians are much less frequently afflicted, but recent research suggests an increasing incidence in non-caucasians and persons across the spectrum of socioeconomic class. About 95% of cases of AN are female, and about 90% of cases of BN are female. However, the prevalence in males may be underestimated because of the wide perception that these are “teenage girl” diseases. Bulimic males are typically less likely to seek treatment than bulimic females. Another common misconception is that eating disorders in males signal probable homosexuality. About 20% of males with eating disorders are homosexual.⁵ The presentation of males with eating disorders is remarkably similar to that of females. Similar methods of weight control are used by both males and females, and there are comparable rates of psychiatric comorbidity and body image dissatisfaction.⁶ A greater percentage of males were overweight before developing their eating disorders.⁵ For BED, there is less gender difference.⁷

Estimates of incidence often vary significantly due to variations in the diagnostic criteria utilized and problems with the underreporting of these disorders that are accompanied by denial and secrecy. Reasonable estimates for lifetime prevalence of AN among females in Western countries approach 1% with a range of 0.5 to 3.7%.¹ The lifetime prevalence for BN among women has yielded higher numbers, with a range of 1.1 to 4.2%.¹ Prevalence rates for more broadly defined forms of eating disorder and Eating Disorder, NOS may be substantially higher. Estimates for BED are 2.0% of the general population,¹ with a 3:2 female to male ratio.⁸ The incidence of BED increases to about 30% of patients who seek treatment for obesity and about 70% of participants in Overeaters Anonymous.⁹ There is no significant racial bias for BED.⁹

Individuals at high risk for the development of eating disorders include dancers, gymnasts, long-distance runners, figure skaters, models, wrestlers, bodybuilders, jockeys, cheerleaders, entertainers, and participants in any other occupations or activities that place

a pronounced emphasis on optimal body weight and shape. Patients with certain medical conditions are at risk for developing eating disorders, and may misuse their prescribed treatments or physical conditions to facilitate weight loss and other purging strategies (Table 68.4).¹⁰

TABLE 68.4

Medical Conditions and Weight Loss Methods^a

Medical Condition	Weight Loss Method
Diabetes	Misuse of insulin or low fat/low carbohydrate diet
Hypothyroidism	Misuse of thyroid hormones
Hyperthyroidism	Noncompliance with antithyroid medications
Cystic fibrosis	Failure to follow prescribed diet or take pancreatic enzymes
Crohn's disease	Noncompliance with sulfasalazine
Pregnancy	Use of pregnancy-related side effects (nausea, vomiting) to facilitate weight control

^a Adapted from Powers PS. In: *Handbook of Treatment for Eating Disorders*, 2nd ed., Garner DM, Garfinkel PE, Eds, The Guilford Press, New York, 1997, pg 424.

Etiology

At present no universally accepted theory establishes the etiology of the eating disorders; complex and unique interactions of variables may occur in the affected individual. Disturbances in these interactions may represent primary or secondary phenomena. The current trend is to conceptualize the disorders from a biopsychosocial perspective of multiple converging physiological, psychological, and environmental factors. Table 68.5 summarizes the more accepted etiologic theories.

TABLE 68.5

Etiological Theories of the Eating Disorders

Biological
Genetic
Psychological
Feminist/Social/Cultural
Familial

Numerous biologic theories have been proposed to explain the etiology of the eating disorders; however, no single biologic abnormality unequivocally accounts for the eating disorders. For underweight subjects, most aberrations appear to be the result rather than the cause of the weight loss or malnutrition, and most abate after healthy weight and adequate nutrition are restored.¹¹ The primary structure controlling ingestive behavior is the hypothalamus of the brain, which also regulates metabolism and end organ function. Other brain structures involved include the limbic system, amygdala, orbitofrontal cortex, and multiple brain stem nuclei. Neuroimaging methods have detected nonspecific abnormalities outside of the secondary changes that accompany malnutrition and hormonal changes.¹² Multiple alterations in neurotransmitters and neuroendocrine axes have been observed in both AN and BN.¹³⁻¹⁵ For example, abnormal results have been found in eating disorder patients for levels of luteinizing hormone, follicle-stimulating hormone, gona-

dotropin, growth hormone, and cortisol and activity of cholecystokinin, opioids, norepinephrine, and serotonin.¹¹ Once an eating disorder is established, secondary changes in brain chemistry may perpetuate the disease. The fact that amenorrhea is often found in normal-weight bulimics and anorexics before the profound weight loss also suggests some sort of biologic deficit to explain the eating disorders.

Another widely considered theory suggests that the eating disorders, especially BN, are a form of mood disorder.² The sufferer of an eating disorder may be using food to self-medicate negative emotions via alterations in brain chemicals. This theory is supported by the high comorbidity of mood disorders found among eating disorder patients, the positive response of patients to antidepressant medications, and the increased prevalence of mood disorders in relatives of eating disorder patients. These findings further emphasize the importance of proper assessment and treatment of depressive symptoms among the eating disorder population.

A genetic predisposition for eating disorders has been suggested. Co-twins of twins with AN were at higher risk for eating disorders.¹⁶ Woodside¹⁷ demonstrated a 45% concordance rate for AN in identical twins, but only a 6.7% concordance in fraternal twins. While the concordance is higher in monozygotic than in dizygotic twins (47.3 versus 31.5%) for BN, the differences were not statistically significant.¹⁷ In a very complete review of the genetic literature, Lilenfeld and Kaye¹⁸ suggested that certain familial tendencies or "vulnerability factors," such as impulsivity, restraint, affective instability, and obsessiveness contribute to the development of eating disorders. Others have suggested that a genetically determined vulnerability within the hypothalamic system might contribute to eating disorders.

Many psychological theories have been developed to explain the etiology of eating disorders. Bruch¹⁹ suggested that eating disorders result from disturbances in body image, limited self-esteem, and problems in interoceptive awareness. Another popular theory, especially for AN, relates to conflicts that may develop during adolescence and the sexual maturation process. It has been hypothesized that the physical and emotional regressions that take place in the development of AN are the result of problems encountered during the struggle with independence, the formation of an adult identity, and the realization of sexual urges and sometimes competing parental, personal, and peer pressures.

Learning theorists view eating disorders as evolving out of classical or operant conditioning such that maladaptive learned responses are formed by efforts to reduce anxiety and other aversive emotions. Unwanted emotions and stress are reduced by engaging in restrictive dieting or binge-eating behavior. The guilt, shame, and other feelings that result from binge eating are then reduced by the use of some form of compensatory behavior. Negative reinforcement influences eating disorder behaviors to a large extent. Conditioning principles also may be used to explain the development of weight and body image concerns, specific food avoidance, ritualistic eating behaviors, trauma, and feelings of being out of control. Patients with eating disorders typically struggle with cognitive distortions (dichotomous thinking, overgeneralization, personalization, etc.) that develop in response to low self-esteem, anxiety about one's physical appearance, and other negative core beliefs.

Because of the great gender disparity in the incidence of eating disorders, several feminist explanations for the etiology of the eating disorders have been developed that emphasize the role of various social and cultural influences (e.g., diet, fitness, food, fashion, entertainment, cosmetic, and advertising industries). These influences exert strong pressure on women, especially at the onset of puberty, to be thin at all cost. Other common transitions, such as a new school or job, moves, going off to college, death, marriage, or divorce may trigger eating disorders. Concern about body weight and dieting is mani-

fested among girls as early as the preschool years. Women are uniquely vulnerable to our culture's youth and thinness obsession because they are, more than men, judged and valued based largely on appearance. Bulimic women reveal a greater acceptance of attitudes and beliefs about the relationships between thinness, attractiveness, and success than non-bulimic women.²⁰ The Westernization of many countries, including Japan, China, and Fiji is believed to have contributed to the increasing prevalence of eating disorders. Western society's similar preoccupation with dieting, exercise, cosmetics, and cosmetic surgery also appears to contribute to the maladaptive behaviors exhibited by patients with eating disorders. When normal individuals are subjected to semistarvation, behavior becomes increasingly and obsessively focused on food.²¹

Another sociocultural variable is the societal expectation that women should be caretakers and nurturers while being self-sacrificing and other-oriented.²² Schwartz and Barrett²³ suggested that the processes of starvation and purging behavior cause a "numbing effect" that helps the person to deny their feelings, needs, desires, and hunger so that others can be served and satisfied. They also hypothesized that eating disorders have given women a sense of power and control in their lives while they struggle to satisfy society's recommendation to be passive and dependent.

It is also helpful to consider the family system as a whole rather than focusing on the individual patient.²⁴⁻²⁵ Whether the dysfunctional family patterns have caused the eating disorders or the individuals with the eating disorders have contributed to the pathology seen in the family remains unclear. Minuchin, Rosman, and Baker²⁴ reported a number of characteristics of eating disorder families that contribute to the onset and maintenance of the disorder, including enmeshment, overprotectiveness, rigidity, lack of conflict resolution/conflict avoidance, and a pattern by which the symptomatic child diverts marital conflict. Selvini-Palazzoli and Viaro²⁵ reported that families of AN patients follow a six-stage process by which the patient, usually a daughter, plays out a covert game of switching coalitions between herself and her parents throughout the developmental process, which ultimately results in the daughter perceiving power through her illness and a return to the privileged and overindulged status of her childhood. Other researchers²⁶⁻²⁷ have reported unhealthy coalitions formed between the AN patient and a parent, with the eating disorder patient being placed in the role of a parentified child. Root, Fallon, and Friedrich²⁸ have theorized that the normal adolescent processes of separation and individuation and the establishment of autonomy are adversely affected in bulimic families. They suggested that three different kinds of bulimic families (perfect, overprotective, and chaotic) exist that are different across a number of dimensions, including boundary problems, difficulties with affective expression, parental control or lack thereof, trust issues, enmeshment or isolation, and the function of the eating disorder symptoms within the family system. There is a strong emphasis on weight and appearance in these families.

Comorbid Psychiatric Conditions

Eating disorders are commonly associated with many other psychiatric conditions, with mood, anxiety, and personality disorders appearing to be the most common forms of comorbidity (Table 68.6). AN patients tend to develop eating disorders first and then develop comorbidity, whereas patients with BN tend to manifest mood disorders or anxiety disorders prior to the onset of the eating disorder. It is unclear whether psychiatric comorbidity precedes or follows the onset of BED.

TABLE 68.6**Common Comorbid Psychiatric Conditions Found in Patients with Eating Disorders**

Mood Disorders
Major depressive disorder
Dysthymic disorder
Mood disorder due to general medical condition
Anxiety Disorders
Posttraumatic stress disorder
Obsessive-compulsive disorder
Generalized anxiety disorder
Social phobia
Panic disorder
Personality Disorders
Cluster B (borderline, histrionic, narcissistic)
Cluster C (dependent, passive-aggressive, avoidant, obsessive-compulsive)
Substance Abuse and Dependence
Adjustment Disorders

Other Identified Problems in Patients with Eating Disorders

Other common problems typically found among patients with eating disorders are listed in Table 68.7. They should be evaluated and targeted during treatment, as they may contribute to the development and maintenance of the eating disorder. Many patients experience the struggle between being the model child or conforming to others' rules and expectations and wanting to act out in an irresponsible or oppositional manner. AN patients may strive for and attain high academic achievement, while others, particularly BN patients, may experience academic difficulties. Patients may have a wide range of feelings and attitudes about their sexuality and sexual behavior, including anxiety (AN) or sexual promiscuity (BN).

TABLE 68.7**Other Identified Problems Commonly Found among Patients with Eating Disorders**

Perfectionism	Maturity fears
Family dysfunction	Low self-esteem
Low self-efficacy	Control issues
Alexithymia	Poor interoceptive awareness
Cognitive distortions	Self-harming behavior
Relationship problems	Feelings of detachment
Social avoidance and distress	Fear of negative evaluation
Interpersonal distrust and conflict	Mood swings/irritability
Self-destructive anger	Shame and guilt
Emotional, physical, or sexual abuse	Unwanted sexual experience

Bio-Psychosocial Assessment (Table 68.8)**Interview with Referral Source**

Most patients are identified by concerned parents, a spouse, relatives, friends, or a primary care physician. Interviewing a referral source is a first step. The practitioner may be better

able to communicate understanding, concern, and empathy, and develop better trust with a new or prospective patient if this information is used appropriately. This process will be discussed later in this section.

TABLE 68.8

Bio-Psychosocial Assessment of the Eating Disorders

Interview with referral source
Assessment of the patient's motivation for treatment
Medical evaluation
Nutritional assessment
Clinical interview
Psychological testing
Behavioral assessment
Body image assessment
Interview with family members/significant others

Assessment of the Patient's Motivation for Treatment

The patient's motivation for seeking treatment and her probable stage of readiness for change²⁹⁻³⁰ should be considered. A patient may be in the precontemplation, contemplation, preparation, or action stage of change. It is important for the patient, especially the AN patient, to feel that she has some control over the treatment process, although compulsory treatment for AN appears equally effective (in terms of amount of weight gain) when compared to voluntary treatment. The weight gain may take longer in patients receiving compulsory treatment, and their mortality rate is higher.³¹

Medical Evaluation

Nonpsychiatric physicians, especially pediatricians, family physicians, gynecologists, internists, and gastroenterologists play a valuable role in the prevention, early detection, and management of patients suspected of eating disorders.² The patient should receive a comprehensive medical evaluation. This evaluation should include a complete history and physical exam, laboratory tests (electrolytes, complete blood count with differential, urinalysis, BUN, creatinine, glucose, albumin, prealbumin, and thyroid function tests), and an electrocardiogram. Consideration also should be given to determining cholesterol, magnesium, calcium, amylase, liver enzymes, muscle enzymes, and performing bone densitometry and a drug screen.

Symptoms and Signs of Anorexia Nervosa

Most of the physical signs and symptoms of AN reflect adaptation to semistarvation. Growth arrest may occur if starvation precedes epiphyseal closure. Decreases in catecholamine, thyroid, and insulin levels are responsible for the reductions in metabolic rate, pulse rate, blood pressure, respiratory rate, oxygen consumption, carbon dioxide production, cardiac output, hypothermia, cold intolerance, dry skin, dry hair, hypercarotenemia, hypercholesterolemia, prolongation of ankle reflexes, slowed gastric motility, and constipation. Along with emaciation, brain and myocardial atrophy may be present. The latter may produce mitral valve prolapse, which is reversible with weight gain. The QT interval on the electrocardiogram may be prolonged. Cardiac arrest may occur in 5 to 15% of cases. Neurologic signs include peripheral neuropathy, apathy, withdrawal, irritability, impaired

cognition, and obsessive thinking about food. Volume deficits in gray matter may persist in spite of weight restoration. The hypothalamic-gonadal axis response to energy deprivation decreases gonadotropin hormones (LH, FSH), testosterone, and estrogen resulting in lanugo hair, anovulation, amenorrhea, infertility, reduced libido, and decreased bone density. Other signs of diffuse hypothalamic dysfunction are altered fluid balance (edema, dehydration, dizziness, syncope) and abnormal thermoregulation (hypothermia, defective shivering, inadequate response to heat and cold exposure). Nonspecific findings include abdominal distress, bloating, delayed gastric emptying, slowed gastrointestinal transit time, anemia, and kidney dysfunction. Osteoporosis and stress fractures may result from both malnutrition and estrogen deficiency. Signs of micronutrient deficiencies such as Wernicke's syndrome, night blindness, scurvy, etc., may develop in occasional patients. Laboratory abnormalities include anemia, leukopenia, thrombocytopenia, hypo- or hypernatremia, hypo- or hyperkalemia, ketoacidosis, abnormal liver functions, and elevated amylase. If the patient participates in purging behavior, signs and symptoms similar to those of bulimia may also be present. Even in the absence of vomiting there may be significant dental disease (decalcification, enamel erosion, tooth decay, gum disease) because the saliva is deficient in buffers and many of the low-calorie foods and soft drinks that AN patients favor provide an acid load.

Symptoms and Signs of Bulimia Nervosa

Body weight may range from underweight to any degree of obesity; significant weight fluctuations are common. Menses may become irregular. Binge eating may induce pancreatitis, disruption of the myenteric plexus, and rupture of the stomach. Subjects who are prone to reactive hypoglycemia may experience symptoms following binges.

The method of purging determines many of the signs. Fluid and electrolyte disturbances may result from any purging method, whereas signs and symptoms of vomiting include swelling of salivary glands, sore throat, dental erosions and caries, calluses of knuckles, tearing of the esophagus, esophagitis, hematemesis, muscle weakness (including cardiac) secondary to ipecac toxicity, alkalosis, and hypokalemia.

Laxative abuse may produce disruption of bowel function leading to laxative dependence, cathartic colon, hyperchloremic metabolic acidosis, hypokalemia, and loss of protective mucus that may increase vulnerability to infection. Diuretic abuse may cause symptomatic hypokalemia with cardiac arrhythmias, palpitations, muscle spasms, myalgia, and weakness.

Laboratory signs include electrolyte imbalance (hypo- or hypernatremia, hypo- or hyperkalemia, hyperchloremia, acidosis, alkalosis), elevated amylase (salivary or pancreatic origin), and elevated creatine phosphokinase.

To the extent that semistarvation is a feature in bulimia, signs and symptoms are similar to those seen in AN.

Symptoms and Signs of Binge Eating Disorder (BED)

The major complications associated with BED are those associated with obesity, including hypertension, diabetes, and dyslipidemia.

Nutritional Assessment

The nutritional assessment (Table 68.9) should include weight and diet history, nutritional knowledge, feelings and thoughts after consuming various foods, medication regimen,

and the frequency and severity of various potential purging strategies. Signs and symptoms of macro- and micronutrient deficiencies should be sought (see above). The patient's level of nutritional knowledge should be assessed; while many patients will be able to recite exact fat and calorie contents of many foods, they may not have knowledge of what constitutes a healthy diet or understand nutritional requirements. A food diary (time, place, type and amount of food eaten, mood, level of hunger) will provide additional useful information.

TABLE 68.9

Comprehensive Nutritional Assessment

Growth and weight history (current, maximum, minimum, premorbid, and ideal weights)
Dieting history
24-hr recall of foods consumed (typical vs. atypical)
Binge eating behavior (frequency, types and amounts of food, precipitating factors)
Other behaviors (harsh/restrictive dieting, fasting, tasting, diet pills, overeating, snacking)
Method, frequency, and severity of purging behavior (including ipecac use)
Physical activity patterns and exercise
Review of laboratory results (serum electrolytes, serum glucose, CBC, albumin, prealbumin)
Review of medications and supplements
Completion of baseline food diary
Food surveys
Nutritional knowledge
Food portion size estimation
Physical findings
Interview with family members/significant others

Clinical Interview

The clinical interview (Table 68.10) should include a detailed assessment of the disordered eating behavior as well as comorbid conditions, family dysfunction, and other pathology. In addition to the nutrition assessment, the patient's body image is extremely important (image dissatisfaction, image distortion, image goals, ratings of satisfaction with various body parts, comparisons between current self and ideal self, and thoughts and feelings about weight, shape, and size).

TABLE 68.10

Comprehensive Clinical Interview of Patient with an Eating Disorder

Nutritional assessment (see above)
Body image (overall, specific body parts and areas, dissatisfaction, distortion)
Substance use and abuse
Sleep patterns
Sexual maturation/menstrual history
Mental status exam
Presence of comorbid diagnoses
Other identified problems commonly found among patients with eating disorders
Suicidal ideation/self-harming behavior
History of unwanted/traumatic sexual experiences
Complete developmental history
Medical complications associated with eating disorders
Medical history and family medical history
Psychiatric history (include details about past treatment) and family psychiatric history
Academic and occupational history
Assessment of relationships with family members, peers, significant others

Psychological Testing

A number of psychological tests are designed specifically for the assessment of eating disorders and related problems, and have been validated. A battery of psychological tests designed to assess the presence of other psychiatric diagnoses is also desirable. This includes objective measurements of depression, personality, and other diagnostic categories.

Behavioral Assessment

Completion of a food diary is important not only to assess the patient's baseline level of food intake, but also to determine various factors that may influence the restrictive dieting, binge eating, and purging processes. Direct observation of the consumption of a meal helps the assessment process. Attention should be paid to the types of food selections, the presence of finicky eating behaviors, bite sizes, rate of chewing and swallowing, the duration of the meal, the amount of food and liquid consumed, and the patient's thoughts and feelings present during the meal. Ratings before, during and after the meal concerning hunger level, fear of weight gain, anxiety level, fullness/bloatedness, urge to binge, and urge to purge are helpful. Finally, pulse and blood pressure readings before, during, and after the meal might provide valuable information, given their correlation with one's anxiety level.

Interview with Family Members/Significant Others

Patients with eating disorders are not likely to seek treatment on their own and may falsify and conceal information about their food intake. Therefore, talking to family members or a significant other with permission from the patient can provide valuable information about the extent of disordered eating and purging patterns, the amount of weight loss, the patient's premorbid level of functioning, comorbid symptoms, the patient's family environment, and other potential contributing factors. It also is important to assess the family as a system. [Table 68.11](#) provides a brief outline of family factors to consider.

Treatment

There is no single treatment regimen; optimal treatment must be tailored for the individual. Hospitalization is indicated in the presence of life-threatening malnutrition, severe psychiatric impairment, and overwhelming comorbidity. Less severe illness may be treated with partial hospitalization, outpatient therapy, or in rare cases, with a healthy peer or self-help program. A multidisciplinary approach involving a treatment team with a minimum of a psychologist, psychiatrist, and nutritionist is highly recommended.

A number of factors should be considered in determining the most appropriate setting ([Table 68.12](#)) and form of treatment.¹ Inpatient treatment in a medical/psychiatric setting or an eating disorders specialty unit will increase the chances of recovery when compared to general inpatient psychiatric settings whose staffs lack the training and experience typically necessary to treat eating disorder patients.³²

Refeeding is an absolute requirement of the recovery process. Garner and Needleman³³ and LaVia et al.³⁴ have provided excellent guides for the development of a treatment plan, within various settings, that is determined by a number of patient characteristics and the

TABLE 68.11**Assessment of the Family of Eating Disorder Patients**

General functioning
Emphasis on appearance and thinness
Influence of social and cultural factors
History of eating disorders or obesity among other family members
History of psychopathology among other family members
Physical illness among other family members
Poor relations between parents; divorce, separation
Impact of the eating disorder on the family system and its members
Impaired interactions/communication patterns between family members
Enmeshment, triangulation, and unhealthy alliances or coalitions
Overindulgence of the patient
Overprotectiveness of the patient
Separation/individuation and autonomy issues
Independence/dependence conflict
Poor affective expression
Diffusion of boundaries
Parentification of the patient
Rigidity
Chaotic environment
Conflict avoidance or poor conflict resolution

TABLE 68.12**Determining the Most Appropriate Eating Disorder Treatment Setting^a***Medical Factors*

Orthostatic hypotension
 Bradycardia
 Tachycardia
 Inability to sustain core body temperature
 Prior experience with patient at weight required medical intervention

Behavioral/Nutritional Factors

Rapid or persistent decrease in oral food intake and inability to consume adequate diet
 Continued decline in weight despite outpatient and partial hospitalization intervention
 Failure to abide by reasonable minimum weight contract

Psychiatric Factors

Presence of additional stressors that contribute to patient's inability to consume adequate diet
 Significant comorbid diagnoses that warrant intervention on inpatient basis (suicidality)

^a Adapted from American Psychiatric Association, *Am J Psych* 157:1; 2000.

severity of the illness. Hospitalization alone will probably not be enough for most patients, but is an important and often necessary first step in the treatment process which will need to include outpatient followup and possibly day treatment.

All treatment plans should include the goal of reestablishing a healthy weight (i.e., return of menses and ovulation in females, healthy physical growth, sexual maturation and development in children and adolescents). Efforts should be made to increase patient motivation and commitment to the therapeutic process and facilitate ownership of a goal of "wellness." Nutritional education and counseling also is a necessary component of the treatment regimen. Efforts should be made to modify core thoughts, feelings, and attitudes related to the eating disorder symptomatology, and associated comorbidity and other maladaptive

behaviors should be targeted for change. Inclusion of the patient's family or a significant other in the therapeutic process is strongly recommended and is necessary when working with patients who remain in the family home. Treatment should only be considered complete after a period of extensive followup that is designed to enhance relapse prevention. For a thorough review of treatment methods, see Garner and Garfinkel.³⁵

Medical Stabilization and Nutritional Rehabilitation, Education, and Counseling

The primary goal of inpatient treatment is medical stabilization with followup care provided within a partial hospitalization or outpatient setting. Fluid and electrolyte abnormalities should be corrected before implementation of refeeding, since these abnormalities will be exacerbated by the refeeding. Dehydration, hypo- or hypernatremia, and hypokalemia are common with any purging method, whereas alkalosis and acidosis accompany vomiting and laxative abuse, respectively. The refeeding syndrome is characterized by glucose and fluid intolerance, hypokalemia, hypophosphatemia, hypomagnesemia, thiamin deficiency, and cardiac insufficiency.³⁶ These electrolyte abnormalities may not be present prior to the initiation of feeding and may develop precipitously. In the face of malnutrition, cells are depleted in potassium, phosphate, and magnesium, and overloaded with sodium. As energy substrate becomes more available, the sodium pump rapidly restores the intracellular electrolyte levels at the expense of extracellular pools.

Standard medical therapy should be provided for pancreatitis and gastrointestinal bleeding. Symptoms related to gastrointestinal dysmotility improve significantly with refeeding.³⁷ Prokinetic agents improve gastric emptying,³⁸ but it remains to be determined if they affect outcome of the eating disorder. Osteoporosis is not rapidly reversed by weight gain or resumption of menses; the efficacy of estrogen and/or calcium supplementation remains to be determined. Patients should be referred to a dentist to repair damage and minimize future problems. Parotid swelling may persist for years after recovery³⁹ but is benign and does not require treatment.

The general principles of nutritional treatment include:

- Education about the physical and psychological consequences of starvation and binge eating
- Encouragement to begin eating a healthy diet
- Interruption of fasting, binge eating, and purging behaviors
- Initial weight stabilization with gradual restitution of a healthy body weight
- Being comfortable with and eventually including all foods in the patient's diet
- Recognition of appropriate weight and body fat proportions of a healthy body

Education also should include information concerning the body's needs for various macronutrients, sources of nutrients, RDAs, and other pertinent information. Patients should learn about the food pyramid, food portion size estimation, food labeling, grocery shopping, and cooking techniques.

Eating disorders are heterogeneous; different approaches are needed with different patients. The clinician must be open to whatever treatment approach works best with the particular patient. Regular contact with a nutritionist is warranted, especially in the early stages of treatment.

For AN, encouraging large portions and high-energy snacks is often counterproductive. Due to the low body weight and hypometabolic state, "average" portions of food will begin the weight restoration process. A minimum of 1200 kcal per day is suggested.

Opinions differ regarding the rate of weight gain and appropriate techniques. Increases in energy intake should be gradual to prevent refeeding syndrome (see above). Energy requirements during the weight gain phase may vary widely. In forced feeding (parenteral nutrition) studies, the excess calories required per kilogram of weight gain ranged between 5569 and 15619 kcal, with a mean of 9768 kcal.⁴⁰ Subjects who were normal weight prior to the onset of anorexia gain less rapidly and increase their metabolic rate after a glucose challenge more dramatically than subjects who were obese prior to the onset of anorexia.⁴¹ The thermic effect of glucose is greater in AN than in controls.⁴¹ In subjects who have experienced growth arrest, energy intake should be sufficient to support catch-up growth. Exercise should not be severely restricted, so that muscle mass can be restored.

In the inpatient setting, meals and subsequent bathroom access are usually supervised initially. Programs vary in their flexibility regarding refusal to eat certain foods and the use of liquid supplements to replace food not eaten at regular meals. Ultimately, the meal plan should be well-balanced, consist of conventional foods, and be individualized to patient needs. Multiple small meals may be better tolerated than three main meals. Efforts should be made to help patients to eat during three designated meal times, as this meal pattern will probably allow the patient to generalize her behavior to the home environment more easily. "Fattening" and "forbidden foods" should be gradually introduced into the diet. Aversion to fat persists in the recovered state.⁴² Educating the patient on a meal plan based on the exchange system rather than counting kcalories is preferred to prevent obsession with the energy content of food. Weight should be tracked, but not overemphasized. Enteral or parenteral nutritional support may be required for life-threatening protein energy malnutrition (i.e., body weight <70% of ideal weight). Therapists disagree about the appropriateness of forced feeding, as control is such an important issue in most AN patients. It should be borne in mind, however, that severe malnutrition may impair cognition. If possible, patients should be allowed approximately 24 h to do as they please with their meals, after which time a supplemental regimen might be considered to ensure adequate energy intake. This will promote autonomy over the eating process for the patient. Refeeding should be done with extreme caution to avoid electrolyte abnormalities (see above), edema, and fatal cardiac arrhythmias.

Since the initial energy intake recommendations in early phases of treatment are likely to be low, a multiple vitamin and mineral supplement is appropriate. If signs and symptoms of micronutrient deficiency syndromes are present, the specific micronutrient should be prescribed.

For BN and BED, initial therapy focuses on regularity in eating habits and stabilization in weight. Bulimic subjects tend to eat more fat and simple carbohydrate and less protein and complex carbohydrate than subjects without eating disorders.⁴³⁻⁴⁴ Normalized eating with adequate protein and complex carbohydrate intake will reduce the risk of a binge being induced by excessive hunger. The energy content of a patient's meal plan should be determined by the Harris-Benedict equation. Patients have been known to respond better to meals if they are allowed to exclude certain high-risk binge foods from their diet early in the course of treatment. Following achievement of appropriate eating behavior, a healthy diet to achieve gradual weight loss can be implemented if appropriate. However, setting strict limitations on fat consumption only perpetuates the notion of "bad" foods, and may trigger bingeing.

Pharmacotherapy

Pharmacologic treatment should be considered as an adjunct, especially in the management of comorbid behavior, e.g., depression, obsessive-compulsive behavior, and anxiety. For

BN and BED patients, a selective serotonin reuptake inhibitor (SSRI, e.g., fluoxetine) is usually the drug of choice. SSRIs suppress symptoms of disordered eating independent of their antidepressant effects. Tricyclic antidepressants, mirtazapine and olanzapine, often cause weight gain as a side effect, and thus would be more suitable for AN patients. For overweight BN and BED patients, sibutramine (inhibits reuptake of serotonin and norepinephrine) might be helpful.

Individual Therapy

It is important to establish trust and communicate empathy, support, encouragement, and understanding while also setting specific behavioral limitations. Once a therapeutic alliance has been established, the therapist must work toward moving the focus of therapy sessions away from discussions about the amount of food to be consumed, weight, and other specific symptoms, and toward underlying issues related to family dysfunction, relationship problems, low self-esteem, body image concerns, the patient's struggle for autonomy, identity issues, and other identified problems previously outlined. Therapists also should consider exploration of the developmental and cultural factors as well as the family dynamics that may have contributed to the development and maintenance of the eating disorder symptoms. Many forms of individual therapy have been employed in the treatment of the eating disorders, including cognitive-behavioral therapy (CBT), behavioral therapy (BT), interpersonal therapy (IPT), feminist treatment, and various psychodynamically-oriented therapies. Although each of these approaches may prove successful with eating disorder patients, a combination of therapies may prove most helpful, especially as one considers the comorbid disorders that accompany the eating disorders.

CBT, designed to challenge the patient's irrational, distorted, and negative automatic thinking patterns and the negative core belief system, has become the standard of treatment for the eating disorders.³³ CBT is at least comparable, and often superior to, all other types of therapy for eating disorders, especially BN and BED.⁴⁵ CBT emphasizes the self-monitoring of food intake and the identification of antecedent stimuli that elicit periods of restrictive dieting and/or binge eating and purging.^{46,47} Patients are taught to identify stressful situations and the accompanying aversive thoughts and emotions. Ways to cope (problem-solving skills, cognitive restructuring, and other coping strategies) are emphasized. CBT also targets normalized eating patterns, meal planning, goal setting, cognitive restructuring, education about the eating disorders and related medical complications, and prevention of relapse. CBT appears to be superior to the use of medication alone in reducing bulimic symptoms. The combination of CBT and medication may prove even more beneficial, especially if the medication is indicated after consideration of any comorbidity.⁴⁵

BT utilizes combinations of reinforcers (empathic praise and encouragement, access to exercise, visitation, social activities, and other privileges) contingent upon weight gain, appropriate food consumption, decreased purging behavior, and a general movement toward the display of "well" behavior. BT may prove helpful during the initial stages of inpatient treatment for AN. BT also may include meal monitoring, post-meal observations, exposure with response prevention, and temptation exposure with response prevention procedures. Specifically, patients are exposed to meals or binge foods and guided in their efforts to refrain from vomiting after food consumption. Results are equivocal.¹ The addition of behavior therapy to aid in the weight loss process in BED patients has been shown to contribute to decreases in binge eating and weight loss.⁴⁵

In the last decade, increasing attention has been paid to the use of IPT in the treatment of eating disorders. IPT⁴⁸ does not focus directly on the eating disorder symptoms, but

rather on the interpersonal difficulties the patient is experiencing. It is believed that problems in relationships with family members, friends, and significant others contribute to the onset and maintenance of eating disorders, and that resolving these interpersonal difficulties will help to eliminate the eating disorder symptoms. Working through issues of grief, interpersonal role disputes, role transitions, and interpersonal deficits are important aspects of this treatment. IPT may be less effective than CBT at the end of treatment for BN. After a one-year follow-up period, CBT and IPT appeared equally effective.^{47,49} Similarly, CBT and IPT have proved equally effective in the treatment of BED.⁵⁰

Psychodynamic therapies have been employed in the treatment of eating disorders since Bruch¹⁹ first introduced her etiological theories for the eating disorders. Although numerous case studies have suggested that these approaches may prove helpful, no controlled studies of the effectiveness of these therapies compared to other forms of treatment have been published.³³ Psychodynamic therapies incorporated into the types of therapy described above may be warranted in the treatment of patients who fail to respond.³³

Scant data exist regarding the efficacy of utilizing a feminist treatment paradigm in the treatment of the eating disorders. Many therapists who specialize in the treatment of eating disorders believe emphasis should be placed on helping the female patient to identify the sociocultural factors that may have contributed to her struggle with body image concerns and maladaptive dieting patterns. It is important to consider the distribution of power in the therapeutic relationship and to encourage the empowerment of the patient through cooperative treatment planning.⁵¹ Russo⁵² has provided a detailed outline of principles to consider in treating patients from a feminist perspective. Feminist treatment emphasizes the importance of considering issues such as various forms of victimization, role conflicts and confusion, sexual abuse, the struggle for power and control, and general interpersonal relationships.³³

Family Therapy

Family therapy is a necessary component of the treatment regimen when working with a child or adolescent or an adult who is still living with his/her family of origin. The primary goal of family therapy is to facilitate the remission of the eating disorder symptomatology and begin a therapeutic process of change within the family. Other goals include changing the status or role of the identified patient within the family, attempting to translate the eating disorder symptoms into a problem of interpersonal communication and family relationships, expanding the problem and taking emphasis off of the eating disorder, and identifying other maladaptive communication patterns. Attempts are made to disengage the patient's parents from using the eating disorder symptoms in a way that leads to further conflict avoidance, overprotectiveness, overindulgence, patient dependence, enmeshment, diffusion of boundaries, and unhealthy coalitions. Family members are taught how to more effectively express and tolerate strong emotions, and the patient's struggle for independence or need for increased dependence is acknowledged and addressed in family therapy. A comprehensive review of family therapy for eating disorders has recently been published by Lemmon and Josephson.⁵³

Group Therapy

Oesterheld and colleagues⁵⁴ conducted a meta-analysis of 40 group treatment studies, and concluded that group therapy is moderately effective in the treatment of eating disorders. Group therapies prove most helpful when utilized in combination with individual ther-

apies and nutritional education and counseling. Group therapies have merit, since the group experience helps patients reduce the tremendous shame, guilt, and isolation often seen among these patients. Similarly, patients may benefit from the feedback and support provided to them by peers who will typically present with varying degrees of progress in treatment.¹ Group therapy may take the form of a process-oriented group that emphasizes working through difficulties in relationships through the interactions patients experience with their peers, or a more psychoeducational approach that emphasizes the acquisition and practice of new skills. Groups also may be designed to include therapies that closely resemble the types of individual treatment outlined above. Others may emphasize specific problems encountered by patients, such as body image problems, stress management deficits, and other specific skill deficits. A combination of these approaches will probably prove most helpful.

Although little research has investigated the efficacy of treatment programs that follow an addiction model, participation in Overeater's Anonymous has clearly helped some patients. Overeaters Anonymous is a 12-step self-help program, adapted from Alcoholics Anonymous, for people trying to overcome compulsive eating. Groups offer unconditional acceptance and support based on principles grounded in spirituality. Major drawbacks include lack of nutrition education, sometimes the restriction of specific foods (especially sugar and white flour), and lack of data supporting effectiveness.

Participation in group occupational therapy also may prove beneficial, as this type of treatment may help to reduce perfectionism and enhance a patient's self-esteem and self-efficacy.¹

Physical Therapy

The basic goal of physical therapy is to develop regular moderate physical activity as part of a new lifestyle for the purposes of improving health, stress management, and weight management. Sedentary patients should be counseled about starting an exercise regimen, determining physical activity interests, picking an exercise regimen that will fit into one's daily schedule, setting realistic goals, addressing safety issues, methods of self-reinforcement, and anticipating and refraining from noncompliance. Other important topics include emphasizing improved health rather than weight loss, determining one's resting heart rate, maximum heart rate, and a training-sensitive zone, and the importance of finding an exercise partner.

For BN patients who use excessive exercise as a purging method and for most AN patients, emphasis should be placed on decreasing (but not eliminating) physical activity.

Environmental Exposure

A treatment method often not considered involves exposing patients in graduated steps to environmental situations that they are likely to encounter outside of treatment. This would include activities such as taking more responsibility for refraining from purging behavior by operating without a post-meal observation period, and accepting more responsibility for adequate nutrition by going to the cafeteria and making one's own food selections prior to discharge from the hospital. Other activities might include grocery shopping, eating at a fast-food restaurant or the food court at the mall, eating with one's family, trying on clothes in a store, and any other behaviors the patient has avoided because of his eating disorder. Patients are guided through this process with the help of a therapist,

and taught how to use cognitive therapy techniques, self-reinforcement, self-soothing statements, relaxation responses, etc.

Prognosis

Accurate statistics are difficult to determine, but the mortality rate for AN is estimated to be 4 to 6%. Long-term followup shows persistent psychiatric and weight disturbances in the majority of patients. The prognosis is worse for males than females. It is estimated that 50% of BN patients fully recover. The outcome for BED patients is unknown.

Additional Sources of Information

Organizations

AABA — American Anorexia/Bulimia Assoc., 165 West 46th St. #1108, New York, NY 10036, (212) 575-6200, www.aabainc.org

AED — Academy for Eating Disorders, Degnon Associates, Inc., 6728 Old McLean Village Dr., McLean, VA 22101-3906, (703) 556-8729, www.acadeatdis.org

ANAD — Anorexia Nervosa and Associated Disorders, Box 7, Highland Park, IL 60035, (708) 831-3438, www.anad.org

ANRED — Anorexia Nervosa and Related Eating Disorders, P.O. Box 5102, Eugene, OR, 97405, (541) 344-1144, www.anred.com

EDAP — Eating Disorders Awareness and Prevention, 603 Stewart St., Suite 803, Seattle, WA 98101, (206) 382-3587, www.edap.org

IAEDP — International Assoc. of Eating Disorders Professionals, 427 CenterPointe Circle #1819, Altamonte Springs, FL 32701, (800) 800-8126, www.iaedp.com

Overeaters Anonymous Headquarters, World Services Office, 6075 Zenith Ct. NE, Rio Rancho, NM 87124, (505) 891-2664, www.overeatersanonymous.org

Bibliographies

Professional Resources about Eating Disorders (compiled by USDA Dec. 1995).

www.nal.usda.gov/fnic/pubs/bibs/gen/anorhpbr.htm

EDAP Reading List Resources for the Prevention of Eating Disorders (prepared winter 1999).

www.edap.org/reading.html

Online Resources

www.mentalhelp.net

www.something-fishy.com

www.closetoyou.org/eatingdisorders

www.caringonline.com

www.gurze.com

References

1. American Psychiatric Association, *Am J Psych* 157:1; 2000.
2. Yager J. In: *Clinical Psychiatry for Medical Students*, Stoudemire A, Ed, J. B. Lippincott, Philadelphia, PA, 1994, pg 355.

3. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed, American Psychiatric Association, Washington, DC, 1994.
4. Stunkard, AJ. *Psychiatr Q* 33:284; 1959.
5. Andersen AE. *Eating Disorders Rev* 4(5):1; 1993.
6. Olivardia R, Pope HG, Mangweth B, Hudson JI. *Am J Psych* 152:1279; 1995.
7. Yanovski SZ, Nelson JE, Dubbert BK, Spitzer RL. *Am J Psych* 150:1472; 1993.
8. National Institutes of Health. *Binge eating disorder*, U.S. Government Printing Office, Washington, DC, 1993, pg 1.
9. Yanovski SZ. *Obesity Res* 1:306; 1993.
10. Powers PS. In *Handbook of Treatment for Eating Disorders*, 2nd ed, Garner DM, Garfinkel PE, Eds, The Guilford Press, New York, 1997, pg 424.
11. Fava M, Copeland P, Schweiger U, Herzog D. *Am J Psych* 146:963; 1989.
12. Herholz K. *Psych Res* 62:105; 1996.
13. Lucas A. *Mayo Clin Proc* 56:254; 1981.
14. Casper RC. *Psych Clin N Am* 7:201; 1984.
15. Goldbloom DS, Kennedy SH. In: *Medical Issues and the Eating Disorders*, Kaplan AS, Garfinkel PE, Eds, Brunner/Mazel, New York, 1993, pg 123.
16. Walters EE, Kendler KS. *Am J Psych* 152:64; 1995.
17. Woodside DB. In: *Medical Issues and the Eating Disorders*, Kaplan AS, Garfinkel PE, Eds, Brunner/Mazel, New York, 1993, pg 193.
18. Lilienfeld LR, Kaye WH. In: *Neurobiology in the Treatment of Eating Disorders* Hoek HW, Treasure JL, Katzman MA, Eds, Wiley, New York, 1998, pg 169.
19. Bruch H. *Eating Disorders: Obesity, Anorexia Nervosa, and the Person Within*, New York, Basic Books, 1973, pg 1.
20. Striegel-Moore RH, Silberstein LR, Rodin J. *Am Psychol* 41:246; 1986.
21. Keys A, Brozek J, Henschel A, et al. *The Biology of Human Starvation*, University of Minnesota Press, Minneapolis, 1950, pg 1.
22. Killian K. *Fam Relations* 43:311; 1994.
23. Schwartz RC, Barrett MJ. *Psychother Fam* 3:131; 1988.
24. Minuchin S, Rosman BL, Baker L. *Psychosomatic Families: Anorexia Nervosa in Context*, Harvard University Press, Cambridge, MA, 1978, pg 1.
25. Selvini-Palazzoli M, Viaro M. *Fam Proc* 27:129; 1988.
26. Yager J. In: *Family Therapy and Major Psychopathology*, Lansky MR, Ed, Grune and Stratton, New York, NY, 1981, pg 249.
27. Stierlin H, Weber G. *Unlocking the Family Door: A Systemic Approach to the Understanding and Treatment of Anorexia Nervosa*, Brunner/Mazel, New York, 1989, pg 1.
28. Root MPP, Fallon P, Friedrich WN. *Bulimia: A Systems Approach to Treatment*, Norton, NY, 1986, pg 1.
29. Prochaska JO, DiClemente CC, Norcross JC. *Am Psychol* 47:1102; 1992.
30. Vitousek K, Watson S, Wilson GT. *Clin Psych Rev* 18:391; 1998.
31. Ramsay R, Ward A, Treasure J, Russell GFM. *Brit J Psych* 175:147; 1999.
32. Palmer RL, Treasure J. *Brit J Psych* 175:306; 1999.
33. Garner DM, Needleman LD. In: *Handbook of Treatment for Eating Disorders*, 2nd ed, Garner DM, Garfinkel PE, Ed, The Guilford Press, New York, 1997, pg 50.
34. LaVia M, Kaye WH, Andersen A, et al. *Am J Psych* 157:1; 2000.
35. Garner DM, Garfinkel PE, Eds. *Handbook of Treatment for Eating Disorders*, 2nd ed, The Guilford Press, New York, 1997.
36. Solomon SM, Kirby DF. *JPEN* 14:90; 1990.
37. Waldholtz BD, Andersen AE. *Gastroenterology* 98:1415; 1990.
38. Stacher G, Bergmann H, Wiesnagrotzki S, et al. *Gastroenterology* 92:1000; 1987.
39. Hasler JF. *Oral Surg* 52:567; 1982.
40. Dempsey DT, Crosby LO, Pertschuck MJ, et al. *Am J Clin Nutr* 39:236; 1984.
41. Stordy BJ, Marks V, Kalucy RS, Crisp AH, *Am J Clin Nutr* 30:138; 1977.
42. Sunday SR, Einhorn A, Halmi KA. *Am J Clin Nutr* 55:362; 1992.
43. Van der Ster Wallin G, Norring C, Lennernas MAC, Holmgren S. *J Am Coll Nutr* 14:271; 1995.

44. Hetherington MH, Altemus M, Nelson ML, et al. *Am J Clin Nutr* 60:864; 1994.
45. Peterson CB, Mitchell JE. *JCLP/In Session: Psychotherapy in Practice* 55:685; 1999.
46. Fairburn CG. *Psychol Med* 141:631; 1981.
47. Fairburn CG, Marcus MD, Wilson GW. In: *Binge Eating: Nature, Assessment and Treatment* Fairburn CG, Wilson GW, Eds, The Guilford Press, New York, 1993, pg 361.
48. Fairburn CG. In: *Handbook of Treatment for Eating Disorders*, 2nd ed, Garner DM, Garfinkel PE. Eds, The Guilford Press, New York, 1997, pg 278.
49. Fairburn CG, Jones R, Peveler RC, et al. *Arch Gen Psych* 48:463; 1991.
50. Wilfley DE, Agras WS, Telch CF, et al. *J Consulting Clin Psychol* 61:296; 1993.
51. Sesan R. In: *Feminist Perspectives on Eating Disorders* Fallon P, Katzman M, Wooley S, Eds, Guilford Press, New York, NY, 1994, pg 1.
52. Russo D. *Newsletter of the American Psychological Association of Graduate Students* 9:3; 1997.
53. Lemmon CR, Josephson AM. In: *Child and Adolescent Psychiatric Clinics of North America: Current Perspectives on Family Therapy*, Josephson, A, Ed, WB Saunders, Philadelphia, 2001, pg. 519.
54. Oesterheld JR, McKenna MS, Gould NB. *Int J Group Psychotherapy* 37:163; 1987.