

Articles are selected for CME credit designation on the basis of our assessment of the needs of readers of *The Primary Care Companion*, with the purpose of providing readers with a curriculum of CME articles on a variety of topics throughout each volume. There are no prerequisites for participation in this CME activity.

To obtain credit, please study the designated article and complete the Posttest.

Accreditation Statement

Physicians Postgraduate Press, Inc. is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

Credit Designation

Physicians Postgraduate Press, Inc. designates this educational activity for up to 1 Category 1 credit toward the American Medical Association Physician's Recognition Award. Each participant should claim only those credits that he/she actually spent in the educational activity.

Date of Original Release/Review

This educational activity is eligible for CME credit through December 31, 2005. The latest review of this material was November 2003.

Educational Objective

After studying the article by Rushing et al., the participant will be able to:

· Recognize signs of bulimia nervosa in primary care settings and initiate treatment

This pretest is designed to facilitate your study of the material.

1. Which of the following patients is most likely suffering from bulimia?

- a. A slightly overweight college woman with enamel erosion
- b. A very thin unemployed woman complaining of lack of appetite
- c. An obese woman who admits to binge eating with no compensatory action
- d. An extremely thin high school girl who only allows herself 2 crackers and water each day

Pretest answer and Posttest on page 225.

Disclosure of Off-Label Usage

The authors of this article have determined that, to the best of their knowledge, citalopram, fluvoxamine, sertraline, and milnacipran are not approved by the U.S. Food and Drug Administration for the treatment of bulimia nervosa.



Bulimia Nervosa: A Primary Care Review

Jona M. Rushing, B.S.; Laura E. Jones, M.S.; and Caroline P. Carney, M.D., M.Sc.

Bulimia nervosa is a psychiatric condition that affects many adolescent and young adult women. The disorder is characterized by bingeing and purging behavior and can lead to medical complications. Thus, patients with bulimia nervosa commonly present in the primary care setting. Physical and laboratory examinations reveal markers of bulimia nervosa that are useful in making the diagnosis. Treatment is beneficial, and outcomes of early intervention are good. This article discusses the history, presentation, and tools needed for recognizing and treating bulimia nervosa in primary care.

(Primary Care Companion J Clin Psychiatry 2003;5:217-224)

Received July 25, 2003; accepted Oct. 28, 2003. From the Department of Psychiatry (Mss. Jones and Rushing and Dr. Carney), and the Department of Internal Medicine (Dr. Carney), University of Iowa Carver College of Medicine, Iowa City; and the Department of Epidemiology, University of Iowa College of Public Health, Iowa City (Ms. Jones and Dr. Carney).

This work was supported by the University of Iowa Roy J. and Lucille A. Carver College of Medicine Summer Research Fellowship (Ms. Rushing) and grant K08 MH01932-01A1 from the National Institute of Mental Health, Bethesda, Md. (Dr. Carney).

In the spirit of full disclosure and in compliance with all ACCME Essential Areas and Policies, the faculty for this CME activity were asked to complete a full disclosure statement. The information received is as follows: Mss. Jones and Rushing and Dr. Carney have no significant commercial relationships to disclose relative to this article.

Corresponding author and reprints: Caroline P. Carney, M.D., M.Sc., Regenstrief Institute, Indiana University School of Medicine, 1050 Wishard Blvd., RG6, Indianapolis, IN 46202-2872 (e-mail: cdoebbeling@regenstrief.org).

n the United States, eating disorders affect 5 to 10 million people, primarily young women between the ages of 14 and 40 years.¹ Much attention has been paid to the identification and treatment of anorexia nervosa in this age group.^{2,3} However, bulimia nervosa is a more prevalent disorder that is more difficult to identify in the primary care setting. It is imperative that primary care physicians are aware of the presentation and subsequent interventions available to treat patients with bulimia nervosa, considering the relatively silent presentation of the disease. In this article, we provide an overview of bulimia nervosa, associated physical and laboratory examination findings, and diagnostic strategies pertinent to primary care practice.

Bulimia nervosa was described as an ominous variant of anorexia nervosa by Russell in 1979.⁴ However, as more research has been conducted and more patients suffering from bulimia nervosa have been identified, bulimia nervosa and anorexia nervosa are now recognized as 2 distinct syndromes. According to the *Diagnostic and Statistical Manual for Mental Disorders*, Fourth Edition (DSM-IV), bulimia nervosa is characterized by recurrent episodes of binge eating followed by 1 or more compensatory behaviors to eliminate the calories (vomiting, laxatives, fasting, etc.) that take place on average a minimum of twice weekly for 3 or more months.⁵ Patients who do not meet the frequency or length criteria may be diagnosed with DSM-IV eating disorder not otherwise specified.⁵

Bulimia nervosa is also delineated into 2 distinct subtypes: purging and nonpurging. With the purging subtype, patients engage in some method to remove the binged food from their bodies. This is most often accomplished by self-induced vomiting but can include the misuse of laxatives, enemas, or diuretics. Nonpurging bulimics use fasting or excessive exercise as the primary compensation for binges but do not regularly purge. Regardless of subtype, bulimic patients have negative self-evaluations, placing inappropriate importance on weight and body image.

EPIDEMIOLOGY

Whereas the prevalence of anorexia nervosa for women in the United States is 0.5% to 1%, the prevalence of bulimia nervosa is 2% to 3% and can be as high as 10% in vulnerable populations, such as college-aged women.⁶ Males are affected by eating disorders at a rate one tenth of that of females. Demographically, most patients with bulimia nervosa are single, college educated, and in their mid-20s. However, most patients begin experiencing bulimic symptoms during adolescence.⁷ Bulimia nervosa occurs in 2.3% of white women but in only 0.40% of black women.⁸

Risk factors for bulimia nervosa include, but are not limited to, childhood sexual abuse, male homosexuality, eating alone, living in a sorority house, diabetic poor glycemic control, low self-esteem, dieting, involvement in athletics, and occupations that focus on weight.⁹⁻¹⁴ Patients with these risk factors or in high-risk populations and those who the practitioner suspects may have an eating disorder should undergo screening.

SCREENING FOR BULIMIA NERVOSA

Many tools are available for screening in the primary care setting, yet few tools focus on the diagnosis of bulimia nervosa. In the spirit of the CAGE assessment for alcohol abuse,¹⁵ the SCOFF questionnaire screens for eating disorders using 5 simple, easy-to-remember questions.¹⁶ An eating disorder can be suspected with 84.6% sensitivity and 89.6% specificity if a patient responds positively to 2 or more questions.¹⁷ The negative predictive value is 99.3% for the SCOFF questionnaire, which makes this tool useful for screening in clinical practice.

The SCOFF was recently tested against a new instrument, the Eating Disorder Screen for Primary Care.¹⁸ The latter instrument performed slightly better than the SCOFF, with 1 or no abnormal responses ruling out an eating disorder (likelihood ratio [LR] = 0.0) compared with the SCOFF (LR = 0.25). Both were similarly effective in ruling positive for an eating disorder (LR = 11).¹⁸

A 2-item assessment specific to bulimia nervosa may also be an effective screening tool. For responders who answered positively to 1 of the 2 screening questions (Do you ever eat in secret? and Are you satisfied with your eating patterns?), the positive and negative predictive values were 22% and 91% when based upon the gold-standard of a clinical assessment that found a 16% prevalence of bulimia nervosa in the studied population.¹⁹ These 2 screening items had been previously shown to have a sensitivity of 1.00 and specificity of 0.90 for bulimia nervosa.²⁰

The Bulimic Investigatory Test, Edinburgh (BITE) questionnaire is a brief test for the detection and description of bulimia nervosa.²¹ The BITE consists of a set of 33 questions (30 yes/no type and 3 graded-response) that simultaneously assess the presence and relative severity of a binge-eating disorder. The BITE is divided into 2 sections: the symptom scale and the severity scale. The symptom scale consists of the 30 yes/no questions; 1 point is assigned for every "yes" answer, and a score of 20 or greater indicates a binge-eating disorder. The 3 gradedresponse questions make up the severity scale and ask the patient to score the frequency of their actions. A score of 5 or more on this section is considered clinically significant, and a score of 10 or more is considered severe. The BITE takes an average of 10 minutes to complete and can be immediately scored by the practitioner. Although not intended for screening in primary care, this instrument could be used to track disease severity in patients with known disease.

The Eating Attitudes Test has been widely used in epidemiologic studies, particularly to assess for symptoms of anorexia nervosa.^{22–25} The instrument has been validated for bulimia nervosa and modified for use in patients with diabetes mellitus.^{26–28} We recommend starting with the Eating Disorder Screen for Primary Care or the SCOFF for screening purposes in primary care.

CLINICAL PRESENTATION

The diagnosis and long-term treatment of bulimia nervosa require the practitioner to have insight into the patient's thoughts regarding weight, food, and eating rituals. The cardinal symptoms of bulimia nervosa include only inappropriate bingeing and compensatory behavior, not low body weight. It is possible for a patient to suffer from bulimia nervosa and be at or above normal weight. For this reason, normal and overweight patients should not be overlooked if other symptoms or signs of bulimia nervosa are present.

It is necessary to understand the patient's eating behaviors, especially those involving binge eating. The bulimic's idea of a binge may not be, and most likely is not, the same as the physician's idea of a binge. Although a binge is loosely defined as a large consumption of calories in a brief period of time, bulimics tend to define binges by the type of food consumed and their mood state while consuming, not necessarily by the actual caloric intake.^{29,30} For instance, snack foods and desserts are viewed as binge foods more often than are other foods. Some bulimics may have an internal list of forbidden foods that, when consumed, will constitute a binge to that patient, regardless of caloric content. A bulimic could consume a large quantity of fruits and vegetables of the same caloric amount as a candy bar but not view it as a binge because fruits and vegetables are "good" or "safe" foods.

In any case, a sense of loss of control over eating is an important feature of bulimia nervosa. Subjective binges are defined not by the amount of food consumed, but by an appraisal that more food was eaten than should have been consumed and a concomitant sense of lack of control.^{31,32} Further, a typical behavioral pattern may be noted around the time of the binge episode. The bulimic may experience pre-episode boredom, cravings, and depression followed by post-episode depression and lack of self-control.³³ Bulimics commonly binge in private, and some may plan binges and purges according to time of day and privacy issues. They may eat normally around friends and family but then binge at other times when alone. In more severe cases, bulimics may alter daily schedules to be assured of time for bingeing and purging. They may also deprive themselves of food for hours before the binge, and it is thought that this deprivation plays into the ritualistic pattern of bulimic eating.³⁴ Because regular binges may be costly, food may be stolen from grocery and convenience stores.35 The severity of weight and eating attitudes may fuel the frequency of binge and purge behaviors. It is these behaviors that may lead to serious medical and psychiatric complications.

MEDICAL COMPLICATIONS

Whereas amenorrhea is required for a DSM-IV classification of anorexia nervosa in women, only one half of patients with bulimia nervosa experience menstrual disturbances, including amenorrhea or oligomenorrhea.³⁶ Menstrual disturbances occurring in women with eating disorders may be secondary to a decrease in the pulsatility of gonadotropin-releasing hormone and possibly to the hormonal mechanisms of leptin, which decreases with decreasing weight.^{37,38} Since a threshold level of weight or body fat is believed to be necessary for normal reproductive functioning, normal or overweight bulimics are less likely to experience amenorrhea.³⁹

Gastrointestinal (GI) tract abnormalities may occur in bulimics and typically are secondary to purging behaviors. The most commonly reported GI symptoms are bloating, flatulence, and constipation.⁴⁰ Gastric emptying and motility may be delayed.⁴¹ The patient also may develop gastroesophageal reflux disease and Mallory-Weiss tears from excessive vomiting. Rectal prolapse has been described, and when it occurs in a young woman, the clinician should include bulimia nervosa in the differential diagnosis.⁴²

Ipecac, an over-the-counter emetic agent, is often used by bulimics to induce vomiting. Nearly 8% of women with eating disorders may experiment with ipecac, while 1% to 2% regularly abuse the medication.⁴³ Ipecac has been associated with serious cardiac toxicity, including cardiomyopathy with left ventricular dysfunction. Whether or not this dysfunction is reversible remains controversial.⁴⁴ Patients with a history of ipecac abuse may be candidates for echocardiography, especially if symptoms of heart failure are present.

Due to excessive vomiting, patients may exhibit enamel erosion, especially on the lingual surface of the teeth; calluses on middle phalanges from using fingers to induce vomiting (Russell's sign); and sialadenosis, a noninflammatory enlargement of the salivary glands.45,46 Parotid gland swelling is commonly found. Sialadenosis is almost always associated with an underlying systemic disorder, including diabetes, alcoholism, malnutrition, anorexia nervosa, and bulimia nervosa. In bulimics, the incidence of sialadenosis ranges from 10% to 66%. The clinician should suspect bulimia nervosa in a young woman with sialadenosis who has no underlying medical conditions.^{47,48} It is thought that the various causes of sialadenosis all result in a common pathogenic effect in that they produce a peripheral autonomic neuropathy responsible for disordered metabolism and secretion, resulting in acinar enlargement.^{46,49} Although the condition may reverse once vomiting ceases, in cases of refractory swelling, parotidectomy may be considered as a last resort to improve facial aesthetics.46

Unlike patients with anorexia nervosa, bulimic patients generally do not have disturbances in bone mineral density, especially when weight-bearing exercise is used as a compensatory mechanism.⁵⁰ Risk factors for decreased bone mineral density include total duration of amenorrhea,

age at first menarche, time since last menstrual period, and low body weight.⁵¹ Therefore, early-onset osteopenia is not a major concern for normal or overweight bulimic patients, unless a history of anorexia nervosa with a period of greater than 5 months spent below normal weight is also present.

PSYCHIATRIC COMORBIDITY

The psychiatric comorbidity associated with bulimia nervosa is striking. Bulimic patients are characterized as extroverted perfectionists who are self-critical, impulsive, and emotionally undercontrolled.⁵² High prevalence rates of any affective disorder (75%), major depressive disorder (63%), and anxiety disorders (36%) have been reported.⁵³ The majority of patients report that the initial presentation of the depression or anxiety disorder occurred prior to the presentation of bulimic symptoms.⁵³ Thus, positive early identification of an affective or anxiety disorder may provide an opportunity to prevent development of eating symptoms and disorders, especially in high-risk populations. Studies describing the incidence of comorbidities in bulimia nervosa may suffer from sampling bias, referral bias, and lack of appropriate control groups.

Substance abuse is an additional common comorbidity. The National Center on Addiction and Substance Abuse at Columbia University reported that 30% to 70% of bulimics have a substance abuse problem.⁵⁴ Substances of abuse include tobacco, alcohol, and prescription and over-the-counter medications, such as diet pills and stimulants. Alcoholism has been reported to affect 31% of bulimics and is often found with major depressive disorder and post-traumatic stress disorder.⁵⁵ Strong familial relationships have also been observed between bulimia nervosa and alcoholism.⁵⁶

A controlled community sample study compared women with bulimia nervosa with normal controls and controls with other psychiatric disorders. Although current alcoholism was similar among the groups, bulimics had higher rates of deliberate self-harm than both control groups and more illicit drug use than the normal controls.⁵⁷

Self-injury is a concern for patients with bulimia nervosa. In one study, 34% of bulimic patients reported having injured themselves at sometime in their lives, and 21.3% reported having injured themselves in the last 5 months.⁵⁸ Patients most often injure themselves by cutting or scratching their arms, hands, legs, or face, and many of these injuries result in bleeding and scarring. Patients with borderline personality disorder who injure themselves are more likely to also suffer from bulimia nervosa than are those who do not injure themselves. Comorbid diagnoses of bulimia nervosa and borderline personality disorder have been shown to increase risk of frequent selfmutilation, which may influence rates of attempted and completed suicide in these patients.⁵⁹ Bulimic patients are more likely to have experienced parental alcoholism, low parental contact, and high parental expectations.⁶⁰ Although the core symptoms of this disorder are disturbed eating habits and self-perception, the significant comorbidities complicate the identification and treatment of bulimia nervosa.

LABORATORY ABNORMALITIES

The normal-weight or overweight bulimic may or may not have significant laboratory abnormalities. Laboratory abnormalities become more common with decreasing weight and increasing severity of purging behaviors. The electrolyte levels are most likely to be affected.

Hypokalemia, hypochloremia, hyperphosphatemia, and metabolic alkalosis are common, especially in lowerweight bulimics. The severity of hypokalemia and hypochloremia is directly related to the number of purging episodes the patient experiences, especially those involving diuretics, laxatives, and repetitive vomiting.⁶¹ A recent case-control study⁶² suggested that the ratio of urine sodium to urine chloride is the best predictor of bulimic behavior. A ratio of > 1.16 identified nearly 52% of cases, and the false-positive rate was 5%.62 Additionally, the presence of metabolic alkalosis and elevated phosphorus levels should also raise clinical suspicion for surreptitious vomiting.63-65 Although serum potassium levels have been considered a good marker for bulimic behavior, the relative infrequency (4.1% to 13.7%) of clinically significant hypokalemia among bulimics lowers its sensitivity as a screening test.61,62,64,66

Importantly, the patient's overall laboratory picture is dependent on the compensatory mechanisms. Patients who purge by vomiting may present with metabolic alkalosis (elevated serum bicarbonate levels) due to volume contraction. However, patients who primarily abuse laxatives may present with metabolic acidosis (decreased serum bicarbonate levels) due to loss of alkaline fluid from the bowel.⁶³ Patients using more than one purging mechanism may display mixed acid-base findings. These electrolyte imbalances contribute to weakness, fatigue, and, in severe cases, may predispose the patient to cardiac arrhythmias and sudden death.

Serum amylase determination may help to diagnose and monitor bulimia nervosa. An elevated amylase level may suggest that a patient has been vomiting. In some cases, it will be necessary to rule out an organic cause of elevated amylase levels or vomiting, such as pancreatitis. When fractionated into serum and salivary components, elevations are usually disproportionate, with elevated salivary amylase exceeding pancreatic amylase in patients who have been vomiting. The fractionated test, therefore, may be helpful to use as a diagnostic aid in cases where vomiting is denied and to monitor continued vomiting in patients undergoing treatment.⁶⁷ Unlike anorexia nervosa, which is easily discernible given low body weight, the variance in weight presentation among bulimics makes this condition more difficult to diagnose. A baseline assessment for patients suspected of eating disorders includes several elements. Historical information, including prior episodes of electrolyte abnormalities, menstrual irregularities, or GI symptoms such as constipation, provide important clues if these are otherwise unexplained.

Given time constraints in the primary care clinic, the clinician may choose to use either the 2-question assessment discussed previously or the Eating Disorder Screen for Primary Care.¹⁸ Negative scores on these instruments do not rule out the possibility of an eating disorder, as patients who wish to keep the condition secret may not answer the questions in a positive fashion. The physical examination may provide important clues suggesting the presence of bulimia nervosa, particularly the purging subtype of the disorder. On examination, the clinician may look for signs of the medical complications mentioned previously, including dental erosion, scarring or abrasion on the knuckles, and swollen parotid glands.

Primary care providers should consider the use of laboratory tests in both the diagnostic and follow-up evaluation. For underweight patients, patients in whom bulimia nervosa is suspected but denied, and patients in whom physical symptoms and signs are present, laboratory tests may be useful to rule out other disorders or to positively diagnose bulimia nervosa. Although no standard panel of tests is described, serum and urine electrolyte counts, acid-base assessments, and phosphorus levels should be obtained from the underweight patient at both diagnosis and follow-up. Fractionated amylase testing may be beneficial in evaluating for vomiting in patients suspected of having bulimia nervosa and in patients undergoing treatment for the disorder. Electrocardiogram monitoring should be performed in bulimic patients with electrolyte abnormalities, palpitations, chest pain, or low body weight. The bulimic patient with at least a 5-month history of low body weight or anorexia should have a bone density assessment. Other testing, such as upper or lower GI endoscopy, should be considered, depending on the constellation of symptoms and signs. For instance, other conditions that can manifest with GI symptoms include inflammatory bowel diseases, celiac sprue, and irritable bowel syndrome.

TREATMENT

Most patients with bulimia nervosa do not require hospitalization, and the preferred method of treatment is outpatient-based.⁶⁸ Cognitive-behavioral therapy (CBT) is the standard and preferred psychological method of treatment for these patients. In multiple studies, CBT has been demonstrated superior to other forms of psychological treatment, such as interpersonal psychotherapy.^{59,69} This method of treatment for bulimia nervosa was first described by Fairburn in 1981 and has 2 goals: (1) to stop the vicious circle of overeating and vomiting and (2) to modify the abnormal attitudes toward food.⁷⁰ The CBT method has 3 phases that overlap in a 20-week course of therapy.⁷¹ During the first phase, patients are taught about bulimia nervosa and actions that perpetuate the disease. The patient keeps detailed food records, including the frequency of bingeing or purging, which are used in therapy sessions. The second phase consists of teaching the patient to broaden food choices, and additional time is spent on correcting dysfunctional food and body thoughts. The final 3 therapy sessions comprise the third stage and focus on maintenance and relapse prevention. Upon completion of CBT, 45% of patients stopped bingeing and purging and 35% no longer met diagnostic criteria for bulimia nervosa.71

Relapse primarily occurs during the first 4 months after CBT and affects 31% to 44% of patients.⁷² Relapse has been attributed to low motivation during treatment and overly ritualized eating and high frequency of vomiting before treatment.^{72,73} Rapid and sustained response to CBT has been found to be dependent upon frequency of bingeing before treatment and a self-directed personality.⁷⁴

Pharmacologic treatment may be a primary or adjunct therapy for bulimia nervosa. Fluoxetine has been shown to be effective in the treatment of bulimia nervosa. One 16-week and 2 double-blind 8-week clinical trials conducted in the early to mid-1990s all found fluoxetine, 60 mg/day, to significantly decrease overall binge eating and vomiting.41-43,75-77 Fluoxetine also has been reported to significantly decrease vomiting and binge eating in as little as 4 weeks of therapy.78 Bulimic patients followed for up to 1 year on fluoxetine therapy were found to have a significantly decreased likelihood of relapse and improved overall outcome when compared with placebo.79 A case series of 5 underweight patients with binge-eating and purging reported sertraline to be effective in weight restoration and reduction of core eating disorder behaviors.⁸⁰ Although citalopram⁸¹ was effective in treating binge-eating disorder, no differences were noted between cases and controls for fluvoxamine.82 Milnacipran, an antidepressant with both serotonergic and noradrenergic function, was effective for the reduction of bulimic symptoms in an uncontrolled case series.⁸³ To date, fluoxetine remains the only medication approved by the U.S. Food and Drug Administration for the treatment of bulimia nervosa.

The combination of CBT with fluoxetine therapy has been shown to be superior to medication or CBT alone.⁸⁴ Frequency and severity of vomiting and binge eating were decreased in patients on both therapies. Also, in a recent study, patients reported better social adjustment up to 10 years after receiving CBT, fluoxetine, or both therapies for treatment of bulimia nervosa compared with that of women who had been randomized to placebo.⁸⁵ For bulimic patients who do not respond to CBT, fluoxetine has been shown to be effective in decreasing bulimic features.⁸⁶ In light of this research, the current treatment for bulimia nervosa consists of outpatient-based CBT and fluoxetine therapy.

Generally, treatment of physical symptoms is dependent upon severity of the problem (e.g., hypokalemia) and whether or not reversal is expected at cessation of purging behaviors (e.g., dysphagia).

Primary care physicians should consider referring patients for specialty care in situations where eating disorder symptoms are persistent, comorbid psychopathology is present, or self-injurious behaviors or suicidal ideation is encountered. Specialty clinics directed at the care of patients with eating disorders may be most beneficial.^{87,88}

Primary care physicians, patients, and families may benefit from reading about eating disorders or researching community or college campus resources. The American Psychiatric Association issued practice guidelines for the assessment and treatment of anorexia nervosa and bulimia nervosa. These guidelines include an overview of the clinical characteristics and evidence-based treatments.¹ Online resources are plentiful, but among the most comprehensive Web sites are http://www.mentalhealth.com and http://www.besttreatments.org/bulimianervosa.89,90 The former provides links to the American Psychiatric Association and National Institute of Mental Health guidelines, as well as recent articles and research advances. The latter source was produced in affiliation with the Clinical Evidence Program of the British Medical Journal. This site is designed for the lay public and provides descriptions of the clinical features of and treatment options for bulimia nervosa.

OUTCOMES

Although bulimia nervosa is more common than anorexia nervosa, the mortality rate is lower and the recovery rate higher than that of anorexia nervosa.⁹¹ Mortality from bulimia nervosa is approximated at 0% to 3% but may be underestimated due to few long-term follow-up studies involving bulimic patients. Approximately 50% of patients are free from all bulimic symptoms 5 years after treatment.⁹¹ Although outcomes research on bulimia nervosa is sparse, with limited statistical estimates, it has been shown that mortality and recovery are directly related to early intervention and treatment.⁸⁸

Patients who suffer from anorexia nervosa and exhibit bulimic features have more difficulty reaching normal body weight and tend to be at lower body weights, even after treatment.⁹² Anorexics are also prone to developing binge eating after treatment for anorexia nervosa. A 1997 study reported that 30% of treated anorexics developed binge-eating behavior up to 5 years posthospitalization.⁸⁷ When assessing a normal or overweight patient with bulimia nervosa, it is important to gather historical information about the presence and duration of past anorexia nervosa. Anorexia nervosa complicated by bulimic symptoms is associated with higher mortality rates than those from bulimia nervosa alone.93 However, mortality rates and comorbidity rates for all eating disorders may be overestimated due to the fact that most studies take place in academic or specialty research settings. These patients are often more severely ill than patients in outpatient, community-based settings. The actual recovery rates for eating disorders are probably greater, and the overall outcome picture not so bleak.94 Yet, it is important for primary care physicians to be familiar with the presenting symptoms of bulimia nervosa or anorexia nervosa complicated by bulimia and to intervene early in the disease course. Unfortunately, in a study conducted nearly 10 years ago, approximately 1 in 10 patients with bulimia nervosa were in treatment.95

CONCLUSIONS

Bulimia nervosa is a common and treatable condition that often presents in the primary care setting. The hallmark sign for bulimia nervosa is a distorted view of food and eating. Patients with this condition often maintain a normal or above-normal weight. Purging can present outwardly via sialadenosis, dental enamel erosion, and calloused middle phalanges. Patients with bulimia nervosa may have electrolyte and acid-base abnormalities. Bulimia nervosa is also often associated with depression, borderline personality disorder, substance abuse, selfinjury, and a troubled family life.

Bulimia nervosa is a treatable disease, especially when diagnosed early. The majority of patients are successfully treated with fluoxetine and CBT. However, better outcomes are associated with prevention and early detection of abnormal eating patterns. It is important that primary care physicians consider this disease in patients with eating or weight symptoms, especially in the most at-risk population: single, white, educated, college-aged women. Early treatment and specialty referral for these patients is indicated.

Drug names: citalopram (Celexa), fluoxetine (Prozac and others), sertraline (Zoloft).

REFERENCES

- American Psychiatric Association. Practice Guideline for the Treatment of Patients With Eating Disorders [Revision]. Am J Psychiatry 2000;157 (suppl 1):1–39
- Mehler PS. Diagnosis and care of patients with anorexia nervosa in primary care settings. Ann Intern Med 2001;134:1048–1059

- Becker AE, Grinspoon SK, Klibanski A, et al. Eating disorders. N Engl J Med 1999;340:1092–1098
- Russell G. Bulimia nervosa: an ominous variant of anorexia nervosa. Psychol Med 1979;9:429–448
- American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Washington, DC: American Psychiatric Association; 1994
- Hsu LK. Epidemiology of the eating disorders. Psychiatr Clin North Am 1996;19:681–700
- Lee NF, Rush AJ, Mitchell JE. Bulimia and depression. J Affect Disord 1985;9:231–238
- Striegel-Moore RH, Dohm FA, Kraemer HC, et al. Eating disorders in white and black women. Am J Psychiatry 2003;160:1326–1331
- Carlat DJ, Camargo CA Jr, Herzog DB. Eating disorders in males: a report on 135 patients. Am J Psychiatry 1997;154:1127–1132
- Russell CJ, Keel PK. Homosexuality as a specific risk factor for eating disorders in men. Int J Eat Disord 2002;31:300–306
- Schulken ED, Pinciaro PJ, Sawyer RG, et al. Sorority women's body size perceptions and their weight-related attitudes and behaviors. J Am Coll Health 1997;46:69–74
- Steiner H, Kwan W, Shaffer TG, et al. Risk and protective factors for juvenile eating disorders. Eur Child Adolesc Psychiatry 2003;12(suppl 1):38–46
- Stice E, Presnell K, Spangler D. Risk factors for binge eating onset in adolescent girls: a 2-year prospective investigation. Health Psychol 2002;21:131–138
- Rodin GM, Johnson LE, Garfinkel PE, et al. Eating disorders in female adolescents with insulin dependent diabetes mellitus. Int Psychiatry Med 1986;16:49–57
- Ewing JA. Detecting alcoholism: the CAGE questionnaire. JAMA 1984;252:1905–1907
- Morgan JF, Reid F, Lacey JH. The SCOFF questionnaire: assessment of a new screening tool for eating disorders. BMJ 1999;319:1467–1468
- Luck AJ, Morgan JF, Reid F, et al. The SCOFF questionnaire and clinical interview for eating disorders in general practice: comparative study. BMJ 2002;325:755–756
- Cotton MA, Ball C, Robinson P. Four simple questions can help screen for eating disorders. J Gen Intern Med 2003;18:53–56
- Freund KM, Boss RD, Handleman EK, et al. Secret patterns: validation of a screening tool to detect bulimia. J Womens Health Gend Based Med 1999;8:1281–1284
- Freund KM, Graham SM, Lesky LG, et al. Detection of bulimia in a primary care setting. J Gen Intern Med 1993;8:236–242
- Henderson M, Freeman CP. A self-rating scale for bulimia: the "BITE." Br J Psychiatry 1987;150:18–24
- Mann AH, Wakeling A, Wood K, et al. Screening for abnormal eating attitudes and psychiatric morbidity in an unselected population of 15-year-old schoolgirls. Psychol Med 1983;13:573–580
- Garner DM, Olmsted MP, Bohr Y, et al. The eating attitudes test: psychometric features and clinical correlates. Psychol Med 1982; 12:871–878
- Garner DM, Garfinkel PE. Socio-cultural factors in the development of anorexia nervosa. Psychol Med 1980;10:647–656
- Garner DM, Garfinkel PE. The eating attitudes test: an index of the symptoms of anorexia nervosa. Psychol Med 1979;9:273–279
- Gross J, Rosen JC, Leitenberg H, et al. Validity of the eating attitudes test and the eating disorders inventory in bulimia nervosa. J Consult Clin Psychol 1986;54:875–876
- Cantwell R, Steel JM. Screening for eating disorders in diabetes mellitus. J Psychosom Res 1996;40:15–20
- Robertson P, Rosenvinge JH. Insulin-dependent diabetes mellitus: a risk factor in anorexia nervosa or bulimia nervosa? an empirical study of 116 women. J Psychosom Res 1990;34:535–541
- 29. Rosen JC, Leitenberg H, Fisher C, et al. Binge-eating episodes in bulimia nervosa: the amount and type of food consumed. Int J Eat Disord 1986;5: 255–267
- Gleaves DH, Williamson DA, Barker SE. Additive effects of mood and eating forbidden foods upon the perceptions of overeating and binging in bulimia nervosa. Addict Behav 1993;18:299–309
- 31. Fairburn CG, Cooper Z, Cooper PJ. The clinical features and maintenance of bulimia nervosa. In: Brownell KD, Foreyt JP, eds. Handbook of Eating Disorders: Physiology, Psychology and Treatment of Obesity, Anorexia and Bulimia. New York, NY: Basic Books; 1986:389–404

- Kerzhnerman I, Lowe MR. Correlates of subjective and objective binge eating in binge-purge syndromes. Int J Eat Disord 2002;31:220–228
- Jansen A, van den Hout M, Griez E. Clinical and non-clinical binges. Behav Res Ther 1990;28:439–444
- Hetherington MM, Altemus M, Nelson ML, et al. Eating behavior in bulimia nervosa: multiple meal analyses. Am J Clin Nutr 1994;60: 864–873
- Mitchell JE, Gibeau L, Pyle RL, et al. Shoplifting in bulimia nervosa. Compr Psychiatry 1992;33:342–345
- Seidenfeld ME, Rickert VI. Impact of anorexia, bulimia and obesity on the gynecologic health of adolescents. Am Fam Physician 2001;64: 445–450
- Pirke KM, Dogs M, Fichter MM, et al. Gonadotrophins, oestradiol and progesterone during the menstrual cycle in bulimia nervosa. Clin Endocrinol 1988;29:265–270
- Grinspoon S, Gulick T, Askari H, et al. Serum leptin levels in women with anorexia nervosa. J Clin Endocrinol Metab 1996;81:3861–3863
- 39. Frisch RE, Revelle R, Cook S. Components of weight at menarche and the initiation of the adolescent growth spurt in girls: estimated total water, lean body weight and fat. Hum Biol 1973;45:469–483
- Chami TN, Andersen AE, Crowell MD, et al. Gastrointestinal symptoms in bulimia nervosa: effects of treatment. Am J Gastroenterol 1995;90: 88–92
- Kamal N, Chami T, Andersen A, et al. Delayed gastrointestinal transit times in anorexia nervosa and bulimia nervosa. Gastroenterology 1991;101:1320–1324
- Malik M, Stratton J, Sweeney WB. Rectal prolapse associated with bulimia nervosa: report of seven cases. Dis Colon Rectum 1997;40: 1382–1385
- Greenfeld D, Mickley D, Quinlan DM, et al. Ipecac abuse in a sample of eating disordered outpatients. Int J Eat Disord 1993;13:411–414
- Ho PC, Dweik R, Cohen MC. Rapidly reversible cardiomyopathy associated with chronic ipecac ingestion. Clin Cardiol 1998;21: 780–783
- Simmons MS, Grayden SK, Mitchell JE. The need for psychiatric-dental liaison in the treatment of bulimia. Am J Psychiatry 1986;143:783–784
- Coleman H, Altini M, Nayler S, et al. Sialadenosis: a presenting sign in bulimia. Head Neck 1998;20:758–762
- Vavrina J, Muller W, Gebbers JO. Enlargement of salivary glands in bulimia. J Laryngol Otol 1994;108:516–518
- Kinzl J, Biebl W, Herold M. Significance of vomiting for hyperamylasemia and sialadenosis in patients with eating disorders. Int J Eat Disord 1993;13:117–124
- Riad M, Barton JR, Wilson JA, et al. Parotid salivary secretory pattern in bulimia nervosa. Acta Otolaryngol 1991;111:392–395
- Sundgot-Borgen J, Bahr R, Falch JA, et al. Normal bone mass in bulimic women. J Clin Endocrinol Metab 1998;83:3144–3149
- Grinspoon S, Thomas E, Pitts S, et al. Prevalence and predictive factors for regional osteopenia in women with anorexia nervosa. Ann Intern Med 2000;133:790–794
- Westen D, Harnden-Fischer J. Personality profiles in eating disorders: rethinking the distinction between axis I and axis II. Am J Psychiatry 2001;158:547–562
- Brewerton TD, Lydiard RB, Herzog DB, et al. Comorbidity of axis I psychiatric disorders in bulimia nervosa. J Clin Psychiatry 1995;56: 77–80
- Vastag B. What's the connection? no easy answers for people with eating disorders and drug abuse. JAMA 2001;285:1006–1007
- Dansky BS, Brewerton TD, Kilpatrick DG. Comorbidity of bulimia nervosa and alcohol use disorders: results from the National Women's Study. Int J Eat Disord 2000;27:180–190
- Kaye WH, Lilenfeld LR, Plotnicov K, et al. Bulimia nervosa and substance dependence: association and family transmission. Alcohol Clin Exp Res 1996;20:878–881
- Welch SL, Fairburn CG. Impulsivity or comorbidity in bulimia nervosa: a controlled study of deliberate self-harm and alcohol and drug misuse in a community sample. Br J Psychiatry 1996;169:451–458
- Paul T, Schroeter K, Dahme B, et al. Self-injurious behavior in women with eating disorders. Am J Psychiatry 2002;159:408–411
- Dulit RA, Fyer MR, Leon AC, et al. Clinical correlates of self-mutilation in borderline personality disorder. Am J Psychiatry 1994;151:1305–1311
- 60. Fairburn CG, Welch SL, Doll HA, et al. Risk factors for bulimia nervosa: a community-based case-control study. Arch Gen Psychiatry

1997;54:509-517

- Wolfe BE, Metzger ED, Levine JM, et al. Laboratory screening for electrolyte abnormalities and anemia in bulimia nervosa: a controlled study. Int J Eat Disord 2001;30:288–293
- Crow SJ, Rosenberg ME, Mitchell JE, et al. Urine electrolytes as markers of bulimia nervosa. Int J Eat Disord 2001;30:279–287
- Mitchell JE, Hatsukami D, Pyle RL. Metabolic acidosis as a marker for laxative abuse in patients with bulimia. Int J Eat Disord 1987;6:557–560
- Mitchell JE, Pyle RL, Eckert ED, et al. Electrolyte and other physiological abnormalities in patients with bulimia. Psychol Med 1983;13: 273–278
- Bonne OB, Gur E, Berry EM. Hyperphosphatemia: an objective marker for bulimia nervosa? Compr Psychiatry 1995;36:236–240
- Greenfeld D, Mickley D, Quinlan DM, et al. Hypokalemia in outpatients with eating disorders. Am J Psychiatry 1995;152:60–63
- 67. Robertson C, Millar H. Hyperamylasemia in bulimia nervosa and hyperemesis gravidarum. Int J Eat Disord 1999;26:223–227
- Powers PS. Initial assessment and early treatment options for anorexia nervosa and bulimia nervosa. Psychiatr Clin North Am 1996;19:639–655
- Fairburn CG, Jones R, Peveler RC, et al. Three psychological treatments for bulimia nervosa: a comparative trial. Arch Gen Psychiatry 1991;48: 463–469
- 70. Fairburn C. A cognitive behavioural approach to the treatment of bulimia. Psychol Med 1981;11:707–711
- Agras WS, Walsh T, Fairburn CT, et al. A multicenter comparison of cognitive-behavioral therapy and interpersonal psychotherapy for bulimia nervosa. Arch Gen Psychiatry 2000;57:459–466
- Halmi KA, Agras WS, Mitchell J, et al. Relapse predictors of patients with bulimia nervosa who achieved abstinence through cognitive behavioral therapy. Arch Gen Psychiatry 2002;59:1105–1109
- Olmsted MP, Kaplan AS, Rockert W. Rate and prediction of relapse in bulimia nervosa. Am J Psychiatry 1994;151:738–743
- Bulik CM, Sullivan PF, Carter FA, et al. Predictors of rapid and sustained response to cognitive-behavioral therapy for bulimia nervosa. Int J Eat Disord 1999;26:137–144
- Fichter MM, Leibl K, Rief W, et al. Fluoxetine versus placebo: a doubleblind study with bulimic inpatients undergoing intensive psychotherapy. Pharmacopsychiatry 1991;24:1–7
- Goldstein DJ, Wilson MG, Thompson VI, et al. Fluoxetine Bulimia Nervosa Research Group. Long-term fluoxetine treatment of bulimia nervosa. Br J Psychiatry 1995;166:660–666
- Fluoxetine Bulimia Nervosa Collaborative Study Group. Fluoxetine in the treatment of bulimia nervosa: a multicenter, placebo-controlled, double-blind trial. Arch Gen Psychiatry 1992;49:139–147
- Mitchell JE, Fletcher I, Hanson K, et al. The relative efficacy of fluoxetine and manual-based self-help in the treatment of outpatients with bulimia nervosa. J Clin Psychopharmacol 2001;21:298–304
- Romano SJ, Halmi KA, Sarkar NP, et al. A placebo-controlled study of fluoxetine in continued treatment of bulimia nervosa after successful acute fluoxetine treatment. Am J Psychiatry 2002;159:96–102
- Frank GK, Kaye WH, Marcus MD. Sertraline in underweight binge eating/purging-type eating disorders: five case reports. Int J Eat Disord 2001;29:495–498
- McElroy SL, Hudson JI, Malhotra S, et al. Citalopram in the treatment of binge-eating disorder: a placebo-controlled trial. J Clin Psychiatry 2003; 64:807–813
- Pearlstein T, Spurell E, Holstein LA, et al. A double-blind, placebo-controlled trial of fluvoxamine in binge eating disorder: a high placebo response. Arch Women Ment Health 2003;6:147–151
- El-Giamal N, de Zwaan M, Bailer U, et al. Milnacipran in the treatment of bulimia nervosa: a report of 16 cases. Eur Neuropsychopharmacol 2003;13:73–79
- Walsh BT, Wilson GT, Loeb KI, et al. Medication and psychotherapy in the treatment of bulimia nervosa. Am J Psychiatry 1997;154:523–531
- Keel PK, Mitchell JE, Davis TL, et al. Long-term impact of treatment in women diagnosed with bulimia nervosa. Int J Eat Disord 2002;31: 151–158
- Walsh BT, Agras WS, Devlin MJ, et al. Fluoxetine for bulimia nervosa following poor response to psychotherapy. Am J Psychiatry 2000;157: 1332–1334
- 87. Strober MR, Freeman R, Morrell W. The long-term course of severe anorexia nervosa in adolescents: survival analysis of recovery, relapse, and outcome predictors over 10–15 years in a prospective study. Int J Eat

Disord 1997;22:339-360

- Herzog DB, Nussbaum KM, Marmor AK. Comorbidity and outcome in eating disorders. Psychiatr Clin North Am 1996;19:843–859
- Best Treatments Clinical Advance for Patients and Doctors. Available at: http://www.besttreatments.org/bulimianervosa. Accessed Nov 3, 2003
- 90. Internet Mental Health. Available at: http://www.mentalhealth.com. Accessed Nov 3, 2003
- Keel PK, Mitchell JE. Outcome in bulimia nervosa. Am J Psychiatry 1997;154:313–321
- Vaz FJ, Guisado JA, Penas-Lledo EM. History of anorexia nervosa in bulimic patients: its influence on body composition. Int J Eat Disord 2003;34:148–155
- Herzog DB, Greenwood DN, Dorer DJ, et al. Mortality in eating disorders: a descriptive study. Int J Eat Disord 2000;28:20–26
- Johnson CL, Lund BC, Yates WR. Recovery rates for anorexia nervosa [letter]. Am J Psychiatry 2003;160:798; author reply 798
- Welch SL, Fairburn CG. Sexual abuse and bulimia nervosa: three integrated case control comparisons. Am J Psychiatry 1994;151:402–407

For the CME Posttest for this article, see pages 225–226.

For more CME activities, visit —

www.psychiatrist.com/pcc/cme



Participants may receive up to 1 Category 1 credit toward the American Medical Association Physician's Recognition Award by reading the CME article and correctly answering at least 70% of the questions in the Posttest that follows:

Go to **www.psychiatrist.com/cmehome** to take this Posttest online and earn credit immediately. Or

- 1. Read each question carefully and circle the answer on the Registration Form.
- 2. Type or print the registration information in the spaces provided and complete the evaluation.
- 3. Send the Registration Form to the address or fax number listed on the Registration Form.

All replies and results are confidential. Answer sheets, once graded, will not be returned. Unanswered questions will be considered incorrect and so scored. The Physicians Postgraduate Press, Inc. Office of Continuing Medical Education will keep only a record of participation, which indicates the completion of the activity and the designated number of Category 1 credits that have been awarded. Correct answers to the Posttest will be made available to the participants of this activity upon request after the submission deadline.

Accreditation Statement

Physicians Postgraduate Press, Inc. is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

1. The hallmark symptoms for bulimia include all of the following *except*:

- a. Binge eating
- b. Low body weight
- c. Compensatory mechanisms such as vomiting or laxatives
- d. Distorted body image
- 2. Which of the following is associated with vomiting as a compensatory mechanism?
 - a. Calluses on middle phalanges
 - b. Enlarged tongue
 - c. Acidosis
 - d. Decreased serum amylase

3. Electrolyte laboratory values for bulimic patients would show:

- a. Hypokalemia, hypochloremia, and hypophosphatemia
- b. Hypokalemia, hypochloremia, and hyperphosphatemia
- c. Hyperkalemia, hypochloremia, and acidosis
- d. Hypokalemia, hyperchloremia, and hyperphosphatemia

4. When evaluating a suspected case of bulimia, clinicians should also screen for all *except*:

- a. Depressive disorders
- b. Alcohol dependence
- c. Self-injury
- d. Schizophrenia

5. The most effective treatment for bulimia is:

- a. Cognitive-behavioral therapy (CBT) alone
- b. Antidepressants alone
- c. CBT and lithium
- d. CBT and an antidepressant combined

Answer to Pretest: 1. a



Registration and Evaluation

Circle the one correct answer for each question.

1.	а	b	с	d	4.	a	b	с	d
2.	а	b	с	d	5.	а	b	с	d
3.	а	b	с	d					

Print or type

Name						
Social Security number						
Degree	Specialty					
Affiliation						
Address						
City, State, Zij	0					
Phone ()					
Fax ()_						
E-mail						
🖵 Hospital	Private Practice	Resident	🗅 Intern			

Deadline for submission

For a credit certificate to be issued, please complete this Registration Form no later than December 31, 2005. Online submissions will receive credit certificates immediately. Faxed or mailed submissions will receive credit certificates within 6 to 8 weeks.

Keep a copy for your files

Retain a copy of your answers and compare them with the correct answers, which will be published after the submission deadline.

Payment

If you complete the test online, no payment is necessary. A \$10 payment must accompany this form. You may pay by check, money order, or credit card (Visa or MasterCard). Make check or money order payable to Physicians Postgraduate Press, Inc. If paying by credit card, please provide the information below.

Check one:	🖵 Visa	MasterCard			
Card number	r				
Expiration date					
Your signature					

Please evaluate the effectiveness of this CME activity by answering the following questions.

- 1. Was the educational content relevant to the stated educational objectives? Use Ves
- 2. Did this activity provide information that is useful in your clinical practice? □ Yes □ No
- 3. Was the format of this activity appropriate for the content being presented? □ Yes □ No
- 4. Did the method of presentation hold your interest and make the material easy to understand?
 □ Yes □ No
- 5. Achievement of educational objective:
 - A. Enabled me to recognize signs of bulimia nervosa in primary care settings and initiate treatment. □ Yes □ No
- 6. Did this CME activity provide a balanced, scientifically rigorous presentation of therapeutic options related to the topic, without commercial bias? □ Yes □ No
- 7. Does the information you received from this CME activity confirm the way you presently manage your patients? □ Yes □ No
- 8. Does the information you received from this CME activity change the way you will manage your patients in the future? □ Yes □ No
- 9. Please offer comments and/or suggested topics for future CME activities.
- 10. How much time did you spend completing this CME activity?
- 11. Do you have convenient access to the Internet?□ Yes □ No

TEAR OUT AND SEND THIS PAGE, ALONG WITH YOUR PAYMENT, TO:

PHYSICIANS POSTGRADUATE PRESS, INC. • OFFICE OF CONTINUING MEDICAL EDUCATION • P.O. BOX 752870 • MEMPHIS, TN 38175-2870

IF YOU ARE PAYING BY CREDIT CARD, YOU MAY FAX THIS PAGE TO: OFFICE OF CONTINUING MEDICAL EDUCATION AT 901-751-3444

QUESTIONS? CALL 1-800-489-1001 EXT. 8 WWW.PSYCHIATRIST.COM