

# Chapter 28

## EATING DISORDERS

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**INTRODUCTION**

**OVERVIEW OF EATING DISORDERS**

**ETIOLOGY**

**CLINICAL FEATURES**

**DIFFERENTIAL DIAGNOSIS**

**COURSE AND PROGNOSIS**

**ABNORMAL EATING IN MILITARY POPULATIONS**

**MEDICAL AND PSYCHIATRIC TREATMENT OF EATING DISORDERS**

**TREATMENT OF EATING DISORDER ISSUES IN THE COMBAT ENVIRONMENT**

**AREAS FOR FURTHER RESEARCH**

**SUMMARY**

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## INTRODUCTION

Over the past decade, the growing obesity epidemic in the United States has received increased attention. Like the general population, the US military is experiencing an increase in those classified as overweight (54%) despite high physical activity levels.<sup>1</sup> Although the majority of Americans are overweight or even obese by Centers for Disease Control and Prevention standards,<sup>2</sup> the ideal body image as portrayed by television, movie, and fashion mediums appears to be underweight. This ideal may drive some individuals to abnormal eating behaviors, such as food restriction or bingeing with compensatory behaviors. Military service members may be particularly at risk because of the expectation that they conform to specific weight standards, with adverse career consequences for those who fail to live up to those standards. Under the stress of deployment to a combat zone, service members with an eating disorder may experience an exacerbation of their illness. Even those without a history of an eating disorder may develop poor eating habits as a reaction to stress, or, ironically, due to the ready availability of fast food in some locations. On the other hand, for some individuals, eating behaviors and overall fitness may improve in the military, with nutritional counseling and time for physical exercise more available.

The rates of eating disorders in the military parallel those reported in high-risk groups such as athletes and dancers, who place an emphasis on thinness.<sup>3,4</sup> Abnormal eating and dieting behaviors are reported in 25% to 76% of female service members, with a significant

increase around the time of personal fitness assessments (PFAs).<sup>5</sup> Because deployed service members are not subject to PFAs, they may feel less pressured to engage in the abnormal eating and dieting behaviors. Also, with meals eaten community style and public bathrooms, binge and purge behaviors may be more difficult to enact.

The diagnosis of an established eating disorder does not necessarily preclude deployment if the service member's condition is in remission. For example, a physician deployed several years ago soon became overwhelmed with the stress of her duties and separation from family, leading to repetitive bingeing and intentional vomiting several times a day. She had previously avoided mental health treatment due to concerns about an adverse impact on her career. She was subsequently medically evacuated and treated for her bulimic behaviors and depression by one of the authors. Three years later, both her depressive symptoms and bulimia were in remission, allowing her to successfully deploy to a combat zone. Unlike the outcome in this case, other service members with eating disorders have experienced recurrence of their symptoms under the stress of deployment, resulting in early returns.

This chapter presents information on the identification and recognition of eating disorders, medical complications, treatment, and prognoses. The authors also review available literature on eating disorder behaviors in military populations and potential risks of deploying individuals with an eating disorder.

## OVERVIEW OF EATING DISORDERS

Eating disorders consist of a group of increasingly common psychiatric and medical conditions that have been studied extensively in women and in certain groups of men. The risks of both bulimia nervosa (BN) and binge eating disorder (BED) in the general population have increased with successive birth cohorts.<sup>6</sup> Anorexia nervosa (AN) was described in the scientific literature by Sir William Gull (in 1873) as a "mental state [that] destroys the appetite."<sup>7</sup> BN, marked by episodes of bingeing and purging, was first described in 1979.<sup>8</sup> The lifetime prevalence for women in the general population is estimated as 0.5% to 1% for AN and 1% to 3% for BN. Rates for men were previously estimated to be about one tenth as high as those for women.<sup>9</sup> However, in 2007, the National Comorbidity Survey Replication<sup>6</sup> found that the estimated point prevalence of BN in men is significantly higher than previously thought,

approximately one fifth that in women. Eating disorder not otherwise specified (EDNOS) is a residual category for conditions that do not meet the full criteria for AN or BN. It is difficult to determine the prevalence of EDNOS, but estimates range from 1% to 30% in men and women.<sup>10</sup> As many as 60% of treated eating disorder cases fall into the EDNOS category.<sup>11</sup> It is estimated that only about one third of individuals with AN and 6% of those with BN receive mental healthcare,<sup>12</sup> although the majority of persons with eating disorders receive treatment for another mental health complaint.<sup>6</sup> Because physicians infrequently assess for eating disorders, and patients rarely spontaneously disclose them, these disorders may be underdiagnosed.<sup>6</sup> Healthcare providers should routinely ask patients about eating disorder symptoms even when these symptoms are not the presenting complaint.

## Anorexia Nervosa

AN is a psychiatric disorder characterized by extreme weight loss in the absence of a medical cause, refusal to regain weight, and intense determination to continue or maintain weight loss. Patients with AN may deny that they are underweight and take measures to conceal their emaciation with bulky clothes to avoid being “ordered” to gain weight by family members or doctors. Typically, onset is between the ages of 13 and 20, peaking at 17 to 18 years of age.<sup>10</sup> AN is defined by the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV),<sup>9</sup> as including the following conditions:

1. the refusal to maintain body weight at or above a minimally normal weight (approximately 85% of those with the AN are below ideal body weight);
2. an intense fear of gaining weight or becoming fat, even though underweight;
3. a disturbance in the way in which one’s body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of current low body weight; and
4. in postmenarcheal females, amenorrhea (ie, the absence of at least three consecutive menstrual cycles).

AN can be divided into (a) the restricting type, in which the individual does not engage regularly in bingeing or purging behaviors, but severely restricts calories intake, and (b) the bingeing/purging type, in which the individual meets criteria for AN and engages regularly in binge/purge behaviors such as self-induced vomiting and laxative or diuretic misuse. Exhibit 28-1 lists the warning signs and symptoms of AN.

## Bulimia Nervosa

BN is defined according to the DSM-IV<sup>9</sup> as consisting of the following conditions:

1. recurrent episodes of binge eating, with the binge episode characterized by eating within a certain time period more food than most people would eat in the same time period and under the same circumstances, including a sense of lack of control over eating during the episode;
2. recurrent inappropriate compensatory behavior to prevent weight gain;

### EXHIBIT 28-1

#### WARNING SIGNS OF ANOREXIA NERVOSA

- Abnormal weight loss without a medical cause
- Severe restriction of food intake
- Denial of hunger and/or a problem with low weight
- Strict food preferences, such as complete avoidance of foods containing fat or strict vegetarianism
- Intense fear of gaining weight and determination to continue weight loss
- Abnormal reproductive functioning (loss of interest in sex, amenorrhea in females, low testosterone in males)
- Excessive exercise despite fatigue and weakness
- Unusual behaviors or rituals with meal preparation and eating
- Distorted perception of weight or body shape
- Inability to stop losing weight or decrease exercise
- Unrealistically high self-expectations; perfectionism with a sense of ineffectiveness
- Use of dangerous methods to lose weight
- Belief that body weight and shape are extremely important to self-esteem and self-definition
- Psychological symptoms of starvation (eg, depression, difficulty concentrating, social withdrawal)
- Physical signs of starvation (eg, sensitivity to cold, fine hair on the face or body, emaciation)

3. binge eating and inappropriate behaviors occurring at least twice weekly for 3 months;
4. self-evaluation unduly influenced by body shape and weight; and
5. episodes not occurring exclusively during episodes of AN.

BN can be divided into purging and nonpurging types; during the latter, individuals use inappropriate compensatory behaviors such as fasting or excessive exercise rather than engaging regularly in vomiting, laxative use, diuretics, or enemas. Purging-type BN differs from purging-type AN in that patients with the latter are significantly underweight. The onset of BN is usually late adolescence to early adulthood.

Warning signs and symptoms of BN are shown in Exhibit 28-2.

### Eating Disorder Not Otherwise Specified

The EDNOS category is considered by some to be overly broad, whereas the criteria for BN or AN may be too rigid. Examples of EDNOS include meeting all the criteria of AN except for amenorrhea or being underweight, meeting criteria for BN except for frequency of episodes, or engaging in purging behaviors without binge episodes. BED, bingeing without compensatory behaviors, is a category proposed in the DSM-IV for further study. Although only 1% of the general population is felt to meet strict DSM-IV diagnostic criteria for BED,<sup>11</sup> the prevalence of binge behaviors is thought to be significantly higher. Night-eating syndrome (NES) is characterized by continual eating during evening hours between dinner and breakfast, accompanied by negative feelings about the eating behavior, insomnia, and nocturnal awakenings followed by food intake. Prevalence for this condition is estimated at 1.5% of the general population.<sup>13</sup> Like BED, the prevalence of NES is significantly higher in overweight and obese individuals. NES is also a category proposed in the DSM-IV for further study.

### Obesity

Increasing media attention has focused on overeating and obesity. According to the World Health Organization, a body mass index (BMI) of 18.5 to 24.9 is normal, a BMI of 25 to 29.9 is overweight, and a BMI greater than 30 is obese. The Centers for Disease Control and Prevention report that 51% of American women are overweight and 34% are obese.<sup>2</sup> Although obesity is not itself considered an eating disorder, up

**EXHIBIT 28-2**

**WARNING SIGNS OF BULIMIA NERVOSA**

- Fluctuations in weight, usually within about 15 lb of normal weight
- Fear of gaining weight
- Uncontrollable, secretive episodes of binge eating followed by attempts to purge by vomiting, laxatives, etc
- Excessive exercise for weight control
- Physical problems with the throat, stomach, and colon; swelling of the parotid glands
- Dental problems, tooth decay
- Psychological problems such as depression, mood swings, impulsivity
- Dissatisfaction with body shape and preoccupation with weight loss
- Belief that body weight and shape are extremely important to self-esteem and self-definition
- Unrealistically high self-expectations; perfectionism with a sense of ineffectiveness

to 50% of obese individuals, particularly those seeking bariatric surgery, have BED or NES.<sup>11,13</sup> A cross-sectional analysis from the US Department of Defense Survey of Health-Related Behaviors found that 10% of active duty men and 4% of active duty women were obese.<sup>14</sup> Based on data from the general population, it is likely that many of these individuals have an eating disorder contributing to their obesity; thus, recognition of eating disorders directly impacts fitness for duty and deployability.

### ETIOLOGY

The etiology of eating disorders includes genetic, biological, sociocultural, psychological, familial, developmental, and comorbid factors.<sup>15</sup> Although all eating disorders are characterized by an abnormal relationship with food, and usually a disturbance in body image, the underlying factors vary with the type of eating disorder. Although it is important to understand etiological factors commonly found in the different eating disorders, it is also necessary to remember that each person is an individual with a complex history and a unique set of characteristics.

### Biological Factors

Brain serotonin, norepinephrine, dopamine, en-

dogenous opioids, and a variety of neuropeptides (eg, leptin, neuropeptide Y) have been implicated in appetite, food intake, satiety, and the development of eating disorders.<sup>16</sup> Traditionally, the catecholamines, particularly norepinephrine and dopamine, have been associated with appetite or onset of eating. Serotonin has been associated with satiety or cessation of an eating episode as well as specific carbohydrate cravings sometimes associated with binges. Disorders of appetite, satiety, or both, underlie abnormal eating behaviors. This may be relevant to the mechanism of action for medications, particularly antidepressants that modulate brain serotonin and catecholamine activity in the treatment of eating disorders.

Eating disorders co-occur more frequently in

monozygotic twins (50% concordance rate) than in dizygotic twins (10% concordance) and are more likely to occur in first-degree relatives of patients with eating disorders than in the general population. Genetic factors account for 58% to 76% of the variance in AN and for 54% to 83% of the variance in BN, with a 7- to 12-fold increase in prevalence among relatives.<sup>17</sup> Some studies have found a link between the serotonin receptor 5-HT<sub>2A</sub> gene polymorphism in the promoter region and eating disorders.<sup>18</sup> Enhanced 5-HT<sub>2A</sub> receptor binding suggestive of serotonergic dysfunction has been found in AN and BN patients.<sup>19</sup> Polymorphism of the 5-HT<sub>1B</sub> receptor gene has also been linked to minimum lifetime BMI in women with BN.<sup>20</sup> These findings suggest that some individuals are more at risk genetically than others to developing an eating disorder. The increasing incidence of these disorders in society may be the result of the sociocultural importance placed on body image and thinness that has allowed more people with a genetic predisposition for AN or BN to develop an eating disorder.

Although all the complex peripheral and central mechanisms that regulate appetite, food intake, and body weight are not yet understood, most people maintain a body weight around a relatively stable “set point” or “settling point,” which changes across the age span in a predictable manner. In individuals with an eating disorder, regulation of appetite—and in many cases body weight—is thrown out of balance. People who engage in periods of excessive food restriction that may be followed by binges ignore the biological signals of hunger and satiation. Binge behaviors may be a natural holdover from a time when humans foraged or hunted for food and needed to eat as much as possible before food was lost to competitors or spoilage.

### Sociocultural Factors

Social and cultural factors are important in the development of both AN and BN. Although the stereotype persists that these disorders, particularly AN, are more common in higher socioeconomic groups, most of this evidence is based on small, uncontrolled case series.<sup>7</sup> These eating disorders are most common in Western cultures, where tasty, high-calorie food is abundant. Evidence suggests that, as other cultures have become richer and more Westernized, body dissatisfaction has grown and eating disorders have increased.<sup>21</sup>

Obesity is considered a socioculturally driven eating disorder. Individuals and families dine more often in restaurants, where the food served is often high in fat and in large portions. The media present conflicting signals: on one hand, images show thin, happy fami-

lies eating at fast food restaurants; on the other hand, magazines present not only the latest fad diet, but also recipes for beautiful, high-calorie dishes. Eating is a social or family event. From an early age, many people learn to see tasty foods as a reward for good behavior or accomplishment. As individuals struggle to lose or maintain weight by severely restricting intake or skipping meals, they may trigger the urge to binge or impulsively overeat, followed by guilt, and in the case of bulimics, purging or using other compensatory behaviors. In an effort to demonstrate control, patients with AN may simply refuse to indulge.

### Psychological and Family Factors

Families or first-degree relatives of individuals with eating disorders have a higher rate of eating disorders (approximately 10% vs 1%–3% in the general population). Although these rates may indicate a genetic component, there is also evidence for learning and modeling within the family and from peers.<sup>22</sup> Patients with eating disorders frequently report that their parents or siblings were overly concerned with body weight and external appearances. Often, their first diet was started in response to criticism from a family member or friend.

Families of patients with AN are often described as perfectionistic, with one or both parents described as authoritarian and having high expectations for their children. Psychological factors in the development of AN include a drive to perfection, unrealistic self-expectations, and perhaps a misdirected search for autonomy and self-control through control of food intake and weight. The primary comorbid psychiatric condition associated with AN is depression, although it is not clear if this is a preceding condition or a result of AN.

Families of patients with BN are more often described as chaotic, with a higher rate of mood disorders, substance abuse, and eating disorders. A history of sexual abuse during childhood has been reported in one third to nearly one half of women with BN.<sup>23,24</sup> A review of 53 controlled studies that examined the link between sexual abuse and eating disorders found that childhood sexual abuse was a risk factor for eating disorders, particularly BN, with psychiatric comorbidity.<sup>25</sup> Furthermore, women who had experienced both childhood sexual abuse and rape in adulthood had even higher rates of eating disorder behavior and marked impulsivity.<sup>25</sup> Conditions associated comorbidly with BN—including borderline personality disorder, substance abuse, and mood instability—have also been found to be more prevalent in people with a history of childhood abuse.<sup>26</sup> Childhood sexual abuse may lead to diminished self-esteem, development of maladaptive behaviors (including eating disorders), and place-

ment of individuals at risk for further trauma.

A study of 1,887 female Navy recruits found that 57% had a history of childhood physical or sexual abuse or both, and 35% had been sexually assaulted as adults.<sup>27</sup> In addition, women who were raped as adults were 4.8-fold more likely to have experienced

childhood sexual abuse. Trauma may also result in psychobiological changes that increase vulnerability to developing an eating disorder. These findings suggest that healthcare providers should ask about eating patterns, purging behaviors, and body image in female service members with a history of abuse.

## CLINICAL FEATURES

Eating disorders are often hidden.<sup>28</sup> The individual may consider these behaviors to be shameful or may lack insight into their pathological nature. Patients with restricting-type AN may move food around on their plates or otherwise disguise their lack of food consumption. Individuals with BN may eat normally but subsequently purge in secret. Those with AN may be easier to identify based on height and weight measurements and their emaciated appearance. A BMI of less than 17.5 in an individual from Western cultures, where food is abundant, should raise suspicion among healthcare workers and prompt further evaluation. It may be more difficult to spot BN or EDNOS among normal-weight individuals.

In an operational environment, service members with BN may find it more difficult to binge and purge because of lack of privacy for these behaviors. One of the authors treated a female service member who carried a plastic bag into which she vomited in secret due to lack of privacy in the latrine area. She would then wait to dispose of the bag into a trash receptacle when no one was looking. On the other hand, excessive exercise as a compensatory behavior may easily be overlooked as adaptive rather than disguising pathological behavior.

### Medical Findings

#### *Anorexia Nervosa*

In addition to an emaciated appearance with sunken cheeks, prominent bone structure, low body fat, and muscle wasting, patients with AN may have dry skin, hypercarotenemia (manifested by a yellow-orange discoloration of the skin); lanugo (fine, downy hair covering the body to compensate for lower body temperature resulting from loss of body fat); acrocyanosis (digits of the hands and feet become blue and sweaty from decreased circulation); and atrophy of the breasts.<sup>28-30</sup> Symptoms of concomitant hypothyroidism include hair loss, peripheral edema, and sensitivity to cold. Tachypnea and shortness of breath may result from metabolic alkalosis caused by vomiting. Conversely, metabolic acidosis may occur from laxative abuse.<sup>31</sup> Gastrointestinal symptoms

include pain, bloating, and severe constipation (from starvation, chronic laxative abuse, or both) that may result in obstruction and megacolon. Exercise-induced disorders (eg, hernias, shin splints, and other injuries) are also common.

Cardiac problems include mitral valve prolapse, prolongation of the corrected QT interval, sinus bradycardia, and arrhythmias from electrolyte imbalance (particularly hypokalemia).<sup>27</sup> Pneumomediastinum induced by vomiting<sup>32</sup> or cardiomyopathy (from ipsecac poisoning)<sup>28</sup> may be seen on a radiograph. The heart is often strophic due to chronic hypovolemia.<sup>33</sup> The second leading cause of death in AN is cardiac arrhythmia. Central nervous system changes include nonspecific electroencephalogram changes and generalized reversible atrophy associated with starvation and dehydration.

Osteoporosis occurs in half of women with AN and can lead to compression fractures and kyphosis.<sup>34,35</sup> Patients with AN have a 3-fold higher risk of fracture than those who do not. Bone loss may develop in as short a time as 6 months after onset of the illness and persist even after recovery, leading to a long-term risk of fractures. Fractures were found in 57% of women with AN in the ensuing 20-year period after onset.<sup>36</sup> Although most studies of osteoporosis in AN have focused on women, one study<sup>37</sup> found that 50% of men with eating disorders had lumbar spine and femoral neck bone densities more than two standard deviations below those of age-matched controls. Compared with women with eating disorders, this group of men had more severe bone loss. Andersen, Watson, and Schlechte<sup>37</sup> suggested that the correlation between reduced body weight in men and lowered testosterone led to more bone loss. Given the increased physical demands in an operational environment, those with AN may be at even higher risk for fractures.

#### *Bulimia Nervosa*

Patients with BN and other binge/purge conditions usually have normal weight without the concomitant features of starvation. Even patients with bingeing/purging-type AN do not achieve as low a body weight as restrictive-type AN patients. External examination

may reveal damage to teeth and gums from acidic vomit. Russell sign is the calloused posterior surface of one or more fingers used to induce vomiting. Physical findings include gastrointestinal disorders (eg, sequelae of laxative abuse, esophageal tearing from excessive vomiting, and complications of electrolyte imbalance, including metabolic alkalosis and cardiac arrhythmias).<sup>28,31</sup>

### Laboratory Studies

Serum chemistry may show electrolyte disturbances. Patients who purge may develop hypokalemia, hypochloremia, and elevated serum bicarbonate.<sup>38</sup> Hypokalemia appears to be particularly common, occurring in up to one half of those who purge; however, the ratio of urinary sodium to urinary chloride may be

a better predictor of purging behavior than serum hypokalemia.<sup>39</sup> Metabolic acidosis with low serum bicarbonate may occur in laxative abusers.<sup>38</sup> Hyponatremia may result from water intoxication or a syndrome of inappropriate antidiuretic hormone.<sup>28</sup>

Other laboratory abnormalities include anemia; leucopenia, neutropenia, and thrombocytopenia<sup>40</sup>; hypercholesterolemia<sup>41</sup>; and euthyroid sick syndrome with normal thyroid-stimulating hormone and low triiodothyronine and thyroxine.<sup>28,42</sup> Hypercarotenemia has been proposed as a laboratory marker for restricting-type AN, with a sensitivity of 62% and a specificity of 83% when a cutoff marker of 200  $\mu\text{g}/\text{mL}$  is used.<sup>30</sup> In one study of patients with AN, high serum creatinine and uric acid levels were associated with a chronic disease course, whereas low serum albumin and low body weight predicted lethality.<sup>38</sup>

## DIFFERENTIAL DIAGNOSIS

Medical conditions that must be considered in the differential diagnosis include inflammatory bowel disease, thyroid disease, abdominal malignancy, central nervous system disease or tumor, and new-onset diabetes mellitus. The psychiatric differential includes

depression, substance abuse, psychosis, and obsessive-compulsive disorder. Comorbidity with psychiatric conditions (including depression, psychosis, anxiety, personality disorder, and substance abuse) must also be addressed.

## COURSE AND PROGNOSIS

### Anorexia Nervosa

AN is associated with significant morbidity and mortality. It is usually a chronic, sometimes life-long disease with low full recovery rates. In a review of studies<sup>43</sup> conducted with patients at least 4 years after onset of illness, approximately 24% had what was considered a poor outcome (eg, never reached target weight gain within 15% of normal, had not established regular menses). Another 44% were considered to have a good outcome (eg, achieved and maintained weight within 15% of normal, had regular menses). About 28% had outcomes that fell between poor and good. The mortality rate was 5%. Even among those judged to be recovered based on body weight and menses, two thirds continued to struggle with body image, obsessive preoccupation with weight and appearance, and disordered eating habits.

Crude 10-year mortality rates for patients receiving treatment for AN has been cited at 3.3% to 5.6%.<sup>44,45</sup> Twenty-year mortality rates are 15% to 20%, with suicide and cardiac arrest the leading causes of death.<sup>46-49</sup> The annual mortality rate associated with AN is 12-fold higher than the annual death rate as a result of all causes of death for women in the general population 15 to 24 years of age.<sup>46</sup> AN is associated with social and

functional impairments,<sup>41</sup> as well as with medical and psychiatric morbidities.<sup>47</sup>

### Bulimia Nervosa

There is limited evidence that some untreated patients with BN have modest rates of improvement or recovery. For patients treated with either psychotherapy or medication, the short-term improvement rates are 50% to 70%; however, relapse rates are high (30%–50% in 6 months to 6 years follow-up).<sup>50,51</sup> Longer-term prognosis may be somewhat better. Patients with milder symptoms and fewer medical and psychiatric comorbidities who do not require hospitalization have a better course and prognosis. The mortality rate for BN has been cited at 0.3% per year.<sup>52</sup> A metaanalysis of standardized mortality rates in BN 5 to 11 years after diagnosis found a 7-fold greater mortality rate than expected.<sup>51</sup>

### Eating Disorder Not Otherwise Specified

The only study to date that reported mortality information on EDNOS found that 4 of 28 subjects had died in an 11-year follow-up.<sup>53</sup> The risk of dying may be greater in the first few years after diagnosis of an

eating disorder. A review of 10 eating disorder populations found a 2% risk of dying for women in the first

year after presentation and a 5% risk of dying for men in the first and second years.<sup>53</sup>

### ABNORMAL EATING IN MILITARY POPULATIONS

Research on eating disorders in military populations primarily consists of case reports<sup>54-56</sup> and surveys of military populations within gender or service. These surveys generally rely on self-reporting, which in civilian populations has been shown to underestimate pathological weight-control behaviors.<sup>57</sup> A summary of the published literature assessing eating disorders in military populations is provided in Table 28-1. There are no studies of eating disorders in military populations in combat operational environments.

McNulty<sup>5</sup> surveyed prevalence and contributing factors of abnormal eating behaviors in 3,000 active duty women in the US Army, Navy, Air Force, and Marine Corps. The rate of AN was highest in the Marine Corps, at 4.9%. Rates across the other services were Army, 1.3%; Navy, 1.1%; and Air Force, 0.8%. The rate of BN was again highest among marines at 15.9%. Rates in other services were Army, 4.3%; Navy, 5.2%; and Air Force, 9.3%. Rates of EDNOS were far higher: 62.8% of the total population met the criteria for this diagnosis. Again, the Marine Corps reported a significantly higher rate: 76.7%. The rates for the other services were Army, 57.4%; Navy, 61.2%; and Air Force, 58.6%. Notably, more than 60% of respondents had some type of eating disorder, and nearly every Marine Corps respondent (97.5%) met criteria for an eating disorder. Of those with an eating disorder at the time of the survey, the overwhelming majority had no history of previous eating disorder and negative family histories. Fasting or purging increased during PFA periods, suggesting that the military environment may put women at risk for eating disorders and increased use of unhealthy strategies of weight reduction to meet standards.

In a survey of 423 active duty Army women (officers and enlisted personnel) from medical and field commands, Lauder and colleagues<sup>58</sup> found that 142 women (33.6%) were at risk for abnormal eating behaviors. These at-risk women admitted to abnormal eating or purging behaviors more than once a month for 3 months, or had high scores of body dissatisfaction and a drive for thinness associated with a BMI of less than 21. Of the 142 women deemed at risk by the survey, 108 completed a structured interview with a board-certified psychiatrist. Of this number, 33 were diagnosed with an eating disorder. The other women had specific stressors, such as PFA periods, that prompted their abnormal eating behaviors. Therefore, 8% of the total sample was diagnosed with an eating

disorder, and 3.1% met criteria for an eating disorder situational to the military environment. In a follow-up study of this same population, Lauder et al<sup>59</sup> evaluated their subjects for the prevalence of the female athlete triad, defined by the presence of an eating disorder, amenorrhea, and osteoporosis. They found no subjects who met the full triad, although, as they pointed out, the military physical activities that the subjects participated in may have had a protective effect on bone mass density.

McNulty<sup>60</sup> surveyed 1,425 Navy men from medical and line communities. She reported prevalence for AN (2.5%), BN (6.8%), and EDNOS (40.8%) in this population. Purging behaviors increased dramatically (up to 15%) during PFA periods. Fasting during these times occurred in nearly one third of respondents. Stressors of military life significant for AN, BN, or EDNOS included failing to be selected for advanced training schools, fear of being involuntarily separated, mandatory physical fitness, nonsupport of a supervisor, height/weight requirements, and rotating shifts. McNulty further reported that some sailors spoke of anger and discouragement over the PFA and their personal struggles with weight. One soldier even reported past suicidal ideation associated with the issue.

In a similar survey of 1,323 female Navy nurses, McNulty<sup>61</sup> reported the prevalence of AN (1.1%), BN (12.5%), and EDNOS (36%). To lose weight rapidly, these respondents reported skipping meals (44.4%), binge eating (19.2%), exercising excessively (16.9%), using diet pills (8.5%), using laxatives (7.1%), and vomiting (3%). Poor body image and satisfaction were predictors of eating disorders, as were height (in AN) and weight (in BN). Work-related stressors that adversely impacted eating disorder behaviors included working in an undesired area or in the intensive care unit, rotating shifts, and being a staff nurse. It is unclear if the high rates of BN and EDNOS in the survey personnel, compared with the general population, were related to their status as military officers or their occupation as nurses. Other studies of eating behaviors in nursing students have found conflicting results. A study of female medical students and nursing students found abnormal eating behaviors in one fifth of the respondents, with a higher rate in the nursing students.<sup>62</sup> Another study found a similar rate of overall abnormal eating behaviors in nursing, medical, and art students (~20%), but no difference among the three groups.<sup>63</sup>



**TABLE 28-1**  
**SUMMARY OF ABNORMAL EATING BEHAVIOR STUDIES IN MILITARY POPULATIONS**

Subjects	N	Findings	Study			
Army ROTC	310	20% subjects with increased bulimic behaviors, body dissatisfaction, drive for thinness	Lauder TD. Abnormal eating behaviors in female Reserve Officer Training Corps cadets. <i>Mil Med.</i> 2001;166:264–268.			
Women in the Army, Navy, Air Force, and Marines	3,000					
			<u>AN (%)</u> <u>BN (%)</u> <u>EDNOS (%)</u>	McNulty P. Prevalence and contributing factors of eating disorder behaviors in active duty service women in the Army, Navy, Air Force, and Marines. <i>Mil Med.</i> 2001;166:53–58.		
		Marines	4.9		15.9	76.7
		Navy	1.1		5.2	61.2
Air Force	0.8	9.3	58.6			
Army women	423	33.6% abnormal eating or purging behaviors and increased body dissatisfaction; 8% diagnosed with eating disorder; 3.1% situational eating disorder	Lauder T, Williams MV, Campbell CS, Davis GD, Sherman RA. Abnormal eating behaviors in military women. <i>Med Sci Sports Exerc.</i> 1999;31:1265–1271.			
Army women with “at-risk” eating behaviors	108	None with female athlete triad (osteoporosis, amenorrhea, eating disorder)	Lauder T, Williams M, Campbell C, Davis G, Sherman R. The female athlete triad: prevalence in military women. <i>Mil Med.</i> 1999;164:630–635.			
Navy men	1,425					
			<u>AN (%)</u> <u>BN (%)</u> <u>EDNOS (%)</u>	McNulty P. Prevalence and contributing factors of eating disorder behaviors in active duty Navy men. <i>Mil Med.</i> 1997;162:753–758.		
Men	2.5	6.8	40.8			
Navy women (nurses)	1,323	Women	1.1 12.5 36.0	McNulty P. Prevalence and contributing factors of eating disorder behaviors in a population of female Navy nurses. <i>Mil Med.</i> 1997;162:703–706.		
Air Force weight-management patients + Civilian weight-management patients + Military control population	155	Air Force group: 4 times more purging behaviors than civilian group, 2–5 times more purging behaviors than military control group; great fluctuations in weight	Peterson AL, Talcott GW. Bulimic weight-loss behaviors in military versus civilian weight-management programs. <i>Mil Med.</i> 1995;160:616–620.			
Israeli Army women diagnosed with anorexia	16	6/1,000 had AN; 16 treated with CBT and clomipramine; 12 returned to full duty; 3 returned to limited duty; 1 discharged	Mark M, Rabinowitz J, Rabinowitz S, Gaoni B, Babur I, Danon Y. Brief treatment of anorexia nervosa in military personnel. <i>Hosp Community Psychiatry.</i> 1993;44:69–71.			
Male Hungarian military college students	480	No anorexic or bulimic subjects but general college students more likely to have behavioral and psychological characteristics of eating disorders. Body builders in both groups more likely to be perfectionistic	Lukacs L, Muranyi I, Tury F. Eating and body attitudes related to noncompetitive bodybuilding in military and general Hungarian male student populations. <i>Mil Med.</i> 2007;172:152–156.			
Male Hungarian general college students	752					
Army women and men in advanced individual training	1,090	40% overweight; 10.8% with prior psychiatric history; 25.4% with history of verbal abuse; 9.8% reported disordered eating. Females, overweight, and those with previous psychiatric treatment and history of verbal abuse most at risk	Warner C, Warner C, Matuszak T, Rachal J, Flynn J, Grieger TA. Disordered eating in entry-level military personnel. <i>Mil Med.</i> 2007;172:147–151.			

AN: anorexia nervosa  
 BN: bulimia nervosa

CBT: cognitive-behavioral therapy  
 EDNOS: eating disorder not otherwise specified

ROTC: Reserve Officer Training Corps

Peterson and colleagues<sup>64</sup> compared the prevalence of bulimic weight-loss behaviors in patients enrolled in an Air Force weight-management program with civilians enrolled in a weight-management program and with normal military controls. The Air Force members in the weight-management program vomited, engaged in strenuous exercise, or used the sauna/steam room four times more often than the civilian group. They were 2- to 5-fold more likely than the military comparison group to engage in bulimic weight-loss behaviors. They also lost more weight than the other two groups: 53% of the Air Force members in the weight-management program, but only 10% of the other two groups, reported a weight loss of more than 10 pounds in 1 month. The Air Force members in the weight-management group also showed more variability, with 41% gaining more than 5 pounds in 1 week, compared with 27% in the civilian group and 14% in the military control group. Fixed-interval reinforcement operant conditioning may have affected the fluctuations in weight and heightened weight-loss behaviors seen in the Air Force weight-management group, because participants were required to weigh in every month on a specified date. Thus, they may have continued with their routine eating habits until just a few days before the weigh-in and made drastic, last-minute attempts to drop weight, similar to the behaviors seen in the 6-month PFA cycles.

Warner et al<sup>65</sup> assessed prevalence and risks factors for disordered eating in a cross-sectional survey of over 1,000 advanced individual training soldiers. Even in this entry-level population, 40% were overweight and 9.8% endorsed disordered eating (7.0% of the men, 29.6% of the women). Risk factors for abnormal eating behaviors included being a woman, being overweight, having a history of previous psychiatric treatment, and having a history of verbal abuse.

Carlton et al<sup>66</sup> surveyed eating disorders in a mixed military population at a large Navy medical center. A relatively high percentage of respondents were men, officers, or both, which reflected the general makeup of the population studied. The average BMI reported by the respondents—both men and women—would

put them in the overweight category. More than 50% of respondents reported a BMI of 25 or greater.

Overall, the findings were consistent with those of other studies on eating and dieting behavior in the military, with high rates of body image dissatisfaction, abnormal patterns of eating and dieting, and a high correlation between these behaviors and the PFA cycle. Nearly 40% of respondents reported bingeing or binge-like behaviors, 18% or more reported some type of purging behavior, and 25% reported fasting. These behaviors were associated with worrying about the PFA and were more likely to occur in those with higher BMI, poor body image, or both. A high percentage of the study population reported dissatisfaction with body appearance and self-esteem dependent on body image. Women scored higher; however, data indicated that a significant proportion of men in the Navy are also dissatisfied with their bodies and engage in abnormal eating behaviors. These unhealthy attitudes and behaviors were common even though the sample was from a medical command; however, other studies in Navy personnel have found lower rates of abnormal eating behaviors in healthcare workers (30%) and medical doctors (6%), compared with the rate in shipboard service members (65%).<sup>60</sup>

Although the data indicate that one third or more of the population sample exhibited abnormal eating and weight-loss behaviors, only 2% had actually been clinically diagnosed.<sup>66</sup> This finding is consistent with other indications that eating disorders are underreported in the military, even at a medical command. It is not surprising that service members are reluctant to come forward. In addition to the stigma, the diagnosis may be grounds for disqualification from many assignments, mandatory enrollment in weight-loss programs, denial of promotion, and involuntary separation from service. McNulty<sup>5</sup> reported that in fiscal year 1995 approximately 5,000 people were discharged from military service for failure to meet weight standards. Military providers may be reluctant to diagnose eating disorders because they are traditionally considered difficult to treat and may end a service member's career.

## MEDICAL AND PSYCHIATRIC TREATMENT OF EATING DISORDERS

### Anorexia Nervosa

#### *Medical Assessment and Treatment*

Patients with AN have poor insight into their conditions. They may perceive their low body weight as an accomplishment and have limited motivation to change their behaviors.<sup>67</sup> The American Psychiatric As-

sociation guidelines recommend that patients weighing less than 75% of ideal body weight be treated on an inpatient basis.<sup>68</sup> Other indications for hospitalization are shown in Exhibit 28-3. The hospital utilization rate for individuals with AN is higher than that for any other psychiatric disorder except schizophrenia and organic mental disorders.<sup>69</sup> The cost of treatment is substantial and is estimated to be even higher than

**EXHIBIT 28-3****INDICATIONS FOR HOSPITALIZATION IN ANOREXIA NERVOSA \***

- Limited motivation to change abnormal eating behaviors
- Intractable (or rapid) weight loss despite treatment
- Refusal to eat
- Prolonged QT interval
- Bradycardia < 40 beats per minute
- Arrhythmia
- Hypothermia
- Symptomatic hypotension
- Less than 75% ideal body weight
- Persistent suicidal ideation
- Need for withdrawal/detoxification from laxatives, diet pills, or diuretics

\*This list is not all-inclusive, and a decision to admit a patient to a hospital should always be based on a clinical assessment of the patient's psychiatric and general medical conditions.

that for schizophrenia.<sup>70</sup> Some patients may refuse treatment out of dread of weight gain and limited insight. In these cases, involuntary hospitalization may be necessary. Patients involuntarily committed for AN may show short-term benefit as manifested by weight gain, but have a higher mortality rate than those who undergo treatment voluntarily.<sup>71</sup> With changes in managed care, treatment for AN is increasingly moving toward partial-day programs or outpatient treatment. Hospitalization is often reserved for patients with serious life-threatening medical complications or those who can afford to pay privately.

Once diagnosed with AN, an individual's general medical condition should be assessed. Medical comorbidities, if they exist, must be addressed, although many (such as electrolyte imbalance) may resolve or improve once malnutrition and purging behaviors are rectified. Patients who require hospitalization need careful management because rapid refeeding can lead to gastric bloating, edema, arrhythmia, tachycardia, congestive heart failure, and sudden cardiac death.<sup>33,72</sup> Vitamin supplementation, with calcium at doses of 1,000 to 1,500 mg in addition to a multivitamin, is recommended.<sup>73</sup> Alendronate<sup>74</sup> and etidronate<sup>75</sup> have been found helpful in promoting bone formation in patients with anorexia; however, bone restoration appears to be most determined by weight restoration.

**Psychotherapy**

There are fewer controlled trials in the psychotherapeutic management of AN compared to BN.<sup>76</sup> Family therapy for adolescent patients may be one of the more effective treatments<sup>77,78</sup>; however, this approach is impractical in the military setting. Although better studied in BN, cognitive-behavioral therapy (CBT)—in which cognitive distortions of body image and feelings of self-worth are addressed—has been applied with some success to patients with AN.<sup>79</sup>

Mark et al<sup>80</sup> reported on a treatment protocol implemented by the Israeli Defence Forces to treat AN in their armed forces. They surveyed, weighed, and measured all female soldiers over a 6-month period. In this sample, 6 of every 1,000 (0.6%) female soldiers were anorexic. Requirements for enrollment in the treatment program were an identifiable trigger for the disorder, motivation for treatment and military service, a social support system, and self-acknowledgment of the eating disorder. As part of the treatment program, the soldiers were educated on the serious medical nature of AN and instructed that they could be discharged if they failed or refused treatment. Service members were hospitalized for 4 to 6 weeks. During this time, each underwent a thorough medical workup, was placed on a high-calorie diet, and set goals for weekly weight gain. Therapy was based on a CBT model. All patients were initially given clomipramine to decrease obsessional ruminations; this drug was tapered off over several months. Of the 16 patients followed in the study for 1 year after discharge from the program, 12 were returned to full duty, three were returned to limited duty, and one was discharged.

In one study,<sup>81</sup> interpersonal therapy (IPT), CBT, and “nonspecific supportive clinical management”—defined as supportive therapy techniques, education, and nutritional advice—were compared in a randomized trial. Surprisingly, nonspecific supportive clinical management was found superior to both CBT and IPT, with CBT yielding superior results to IPT. Therapists administering the treatments investigated in the study were not eating-disorder specialists. Results of this study have yet to be replicated, but may have implications for the feasibility of treating AN in the military healthcare setting.

In general, the treatment plan for AN patients should involve a multidisciplinary team, including experts in mental health, nutrition, and internal medicine or primary care. The therapeutic approach should be to treat the whole patient. It is often better to focus away from food and toward resolving underlying issues of self-esteem and perfectionism.

## Medications

Controlled trials of medications in the treatment of AN are summarized in Exhibit 28-4. Restoring weight and subsequent metabolic stabilization are treatment priorities for patients with AN. Cyproheptadine (32 mg/day) was found to improve weight gain in patients with restricting-type AN but not those with bingeing/purging-type AN.<sup>78</sup> Because zinc deficiency has been linked to AN through inhibition of release of neuropeptide Y,<sup>82</sup> supplementation with this mineral may be beneficial in promoting recovery from AN and improving these patients' levels of anxiety and depression.<sup>83</sup> In a controlled trial of 35 female inpatients with AN, supplementation with zinc promoted a rate of increase of BMI twice that of placebo.<sup>84</sup> Birmingham and Gritzner<sup>85</sup> recommend oral administration of 14 mg daily of elemental zinc for 2 months. A double-blind trial<sup>86</sup> of cisapride (10 mg tid) found reduction

in subjective distress during meals in a small group of AN patients, but did not find a difference in gastric emptying or weight gain.

To date, no antidepressant or antipsychotic has been demonstrated to improve the long-term recovery rate from anorexia.<sup>87,88</sup> Tricyclic antidepressants (TCAs) have not only been shown ineffective, but also given their potential lethality, may be risky in this patient population.<sup>89,90</sup> Selective serotonin reuptake inhibitors (SSRIs) have not been found useful in low-weight patients.<sup>91</sup> This may be because of the general state of malnutrition of low-weight patients with AN, resulting in deficiency of tryptophan, the amino acid required for serotonin synthesis.<sup>92</sup> One SSRI, fluoxetine, was shown to be useful at higher doses in preventing relapse in those who have regained weight<sup>93</sup>; however, a larger study reported negative results.<sup>94</sup>

Antipsychotic drugs may have an augmenting role, particularly in patients with AN who have poor insight into their conditions.<sup>95-98</sup> However, not all studies have demonstrated the efficacy of antipsychotics,<sup>99,100</sup> and their use may have serious adverse effects in this population.<sup>73</sup> In a small, randomized trial, olanzapine was superior in reducing ego-syntonic anorexic ruminations compared to chlorpromazine, although there was no difference in weight gain.<sup>101</sup> Theoretically opiate antagonists may be helpful for subgroups of eating-disorder patients who fit an addiction model. Marrazzi et al<sup>102</sup> reported reduction in binge/purge

### EXHIBIT 28-4

#### MEDICATIONS EFFECTIVE\* AS MONOTHERAPY IN PLACEBO-CONTROLLED TRIALS FOR ANOREXIA NERVOSA

- Zinc<sup>1</sup>
- Cyproheptadine<sup>2</sup>
- Fluoxetine (after weight restoration, bingeing/purging subtype)<sup>3,4</sup>
- Naltrexone (bulimic subtype)<sup>5</sup>
- Olanzapine<sup>6</sup>

\*Defined as fewer days to reach healthy BMI or reduction in binge/purge behavior for bulimia nervosa subtype of anorexia nervosa.

BMI: body mass index

(1) Birmingham CL, Goldner FM, Bakan R. Controlled trial of zinc supplementation in anorexia nervosa. *Int J Eat Disord.* 1994;15:251-255. (2) Halmi KA, Eckert E, LaDu TJ, Cohen J. Anorexia nervosa. Treatment efficacy of cyproheptadine and amitriptyline. *Arch Gen Psychiatry.* 1986;43:177-181. (3) Kaye WH, Nagata T, Weltzin TE, et al. Double-blind placebo-controlled administration of fluoxetine in restricting- and restricting-purging-type anorexia nervosa. *Biol Psychiatry.* 2001;49:644-652. (4) Walsh BT, Kaplan AS, Attia E, et al. Fluoxetine after weight restoration in anorexia nervosa: a randomized controlled trial. *JAMA.* 2006;295:2605-2612. (5) Marrazzi MA, Bacon JP, Kinzie J, Luby ED. Naltrexone use in the treatment of anorexia nervosa and bulimia nervosa. *Int Clin Psychopharmacol.* 1995;10:163-172. (6) Bissada H, Tasca GA, Barber AM, Bradwejn J. Olanzapine in the treatment of low body weight and obsessive thinking in women with anorexia nervosa: a randomized, double-blind, placebo-controlled trial. *Am J Psychiatry.* 2008;165:1281-1288.

### EXHIBIT 28-5

#### INDICATIONS FOR HOSPITALIZATION IN BULIMIA NERVOSA\*

- Changes in vital signs (pulse, blood pressure)
- Syncope
- Hypothermia
- Suicide risk
- Alcohol or drug abuse
- Uncontrolled vomiting
- Hematemesis (vomiting of blood)
- Arrhythmia
- Electrolyte imbalance
- Need for withdrawal from laxatives, diet pills, and diuretics

\*This list is not all-inclusive, and a decision to admit a patient to the hospital should always be based on a clinical assessment of the patient's psychiatric and general medical conditions.

behaviors in a mixed population of BN and AN (bulimic subtype) patients treated with naltrexone in a randomized trial of 37 patients.

### Bulimia Nervosa

Literature to guide treatment of BN is fairly extensive; hospitalization is seldom necessary unless there are medical complications (Exhibit 28-5). Compared to AN, evidence shows that BN can be treated more effectively with medication, although psychotherapy remains the cornerstone of treatment. The most established treatment for BN is CBT; in one study,<sup>103</sup> however, fewer than 10% of patients with bulimia who

received psychotherapy were treated with this type of therapy. Guided self-help manuals that use CBT principles have also been found effective.<sup>92,104</sup> Even with CBT, it is estimated that only 50% of patients with BN recover.<sup>105</sup>

Patients with BN who fail to respond to psychotherapeutic techniques may benefit from pharmacotherapy. Medications that have been shown effective in randomized, placebo-controlled trials in the treatment of BN are listed in Exhibit 28-6. Controlled trial data have shown efficacy for TCAs<sup>106-110</sup> and monoamine oxidase inhibitors,<sup>111,112</sup> with the latter demonstrating some superiority.<sup>113</sup> Both TCAs and monoamine oxidase inhibitors may have lethal adverse effects in

#### EXHIBIT 28-6

#### MEDICATIONS EFFECTIVE IN PLACEBO-CONTROLLED TRIALS FOR BULIMIA NERVOSA

- Tricyclic antidepressants (desipramine, imipramine)<sup>1-4</sup>
- Monoamine oxidase inhibitors (phenelzine, isocarboxazid)<sup>5,6</sup>
- Fenfluramine<sup>7</sup>
- Fluoxetine<sup>8-11</sup>
- Bupropion<sup>\*,12</sup>
- Trazodone<sup>13</sup>
- Naltrexone (high dose)<sup>14</sup>
- Ondansetron<sup>15</sup>
- Topiramate<sup>16,17</sup>
- Flutamide<sup>18</sup>
- Citalopram<sup>19</sup>
- Sertraline<sup>20</sup>
- Fluvoxamine<sup>11</sup>

\*Contraindicated in treatment of bulimia because of increased risk of seizure.

(1) Pope HG Jr, Hudson JL, Jonas JM, Yurgelun-Todd D. Bulimia treated with imipramine: a placebo-controlled, double-blind study. *Am J Psychiatry*. 1983;140:554-558. (2) Agras WS, Dorian B, Kirkley BG, Arnow B, Bachman J. Imipramine in the treatment of bulimia: a double-blind controlled study. *Int J Eat Disord*. 1987;6:29-38. (3) Barlow J, Blouin J, Blouin A, Perez E. Treatment of bulimia with desipramine: a double-blind crossover study. *Can J Psychiatry*. 1988;33:129-133. (4) Hughes PL, Wells LA, Cunningham CJ, Ilstrup DM. Treating bulimia with desipramine. A double-blind, placebo-controlled study. *Arch Gen Psychiatry*. 1986;43:182-186. (5) Walsh BT, Gladis M, Roose SP, Stewart JW, Stetner F, Glassman AH. Phenelzine vs placebo in 50 patients with bulimia. *Arch Gen Psychiatry*. 1988;45:471-475. (6) Kennedy SH, Piran N, Warsh JJ, et al. A trial of isocarboxazid in the treatment of bulimia nervosa. *J Clin Psychopharmacol*. 1988;8:391-396. (7) Blouin AG, Blouin JH, Perez EL, Bushnik T, Zuro C, Mulder E. Treatment of bulimia with fenfluramine and desipramine. *J Clin Psychopharmacol*. 1988;8:261-269. (8) Walsh BT, Agras WS, Devlin MJ, et al. Fluoxetine for bulimia nervosa following poor response to psychotherapy. *Am J Psychiatry*. 2000;157:1332-1334. (9) 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. (10) Goldstein DJ, Wilson MG, Thompson VL, Potvin JH, Rampey AH Jr. Fluoxetine Bulimia Nervosa Research Group. Long-term fluoxetine treatment of bulimia nervosa. *Br J Psychiatry*. 1995;166:660-666. (11) Romano SJ, Halmi KA, Sarkar NP, Koke SC, Lee JS. A placebo-controlled study of fluoxetine in continued treatment of bulimia nervosa after successful acute fluoxetine treatment. *Am J Psychiatry*. 2002;159:96-102. (12) Horne RL, Ferguson JM, Pope HG Jr, et al. Treatment of bulimia with bupropion: a multicenter controlled trial. *J Clin Psychiatry*. 1988;49:262-266. (13) Pope HG Jr, Keck PE Jr, McElroy SL, Hudson JL. A placebo-controlled study of trazodone in bulimia nervosa. *J Clin Psychopharmacol*. 1989;9:254-259. (14) Marrazzi MA, Bacon JP, Kinzie J, Luby ED. Naltrexone use in the treatment of anorexia nervosa and bulimia nervosa. *Int Clin Psychopharmacol*. 1995;10:163-172. (15) Faris PL, Kim SW, Meller WH, et al. Effect of decreasing afferent vagal activity with ondansetron on symptoms of bulimia nervosa: a randomized, double-blind trial. *Lancet*. 2000;355:792-797. (16) Hoopes SP, Reimherr FW, Hedges DW, et al. Treatment of bulimia nervosa with topiramate in a randomized, double-blind, placebo-controlled trial, part 1: improvement in binge and purge measures. *J Clin Psychiatry*. 2003;64:1335-1341. (17) Nickel C, Tritt K, Muehlbacher M, et al. Topiramate treatment in bulimia nervosa patients: a randomized, double-blind, placebo-controlled trial. *Int J Eat Disord*. 2005;38:295-300. (18) Sundblad C, Landén M, Eriksson T, Bergman L, Eriksson E. Effects of the androgen antagonist flutamide and the serotonin reuptake inhibitor citalopram in bulimia nervosa: a placebo-controlled pilot study. *J Clin Psychopharmacol*. 2005;25:85-88. (19) Leombruni P, Amianto F, Delsedime N, Gramaglia C, Abbate-Daga G, Fassino S. Citalopram versus fluoxetine for the treatment of patients with bulimia nervosa: a single-blind randomized controlled trial. *Adv Ther*. 2006;23:481-494. (20) Milano W, Petrella C, Sabatino C, Capasso A. Treatment of bulimia nervosa with sertraline: a randomized controlled trial. *Adv Ther*. 2004;21:232-237.

this patient population (potentially made worse by underlying electrolyte imbalance) and are not currently recommended as first-line treatment. Fenfluramine, a serotonergic agonist withdrawn from the market, was shown to be superior to both placebo and desipramine (a TCA).<sup>110</sup> The SSRI antidepressants are, similarly, serotonergic agonists and theoretically may work through a similar mechanism. Fluoxetine at high doses (60 mg/day) was superior to placebo in two 8-week trials<sup>114,115</sup> and one 16-week trial.<sup>116</sup> Fluoxetine's efficacy in BN, as with the TCAs,<sup>117</sup> is not a secondary effect of its antidepressant properties.<sup>118</sup> As many as one third of initial responders may relapse by the end of 1 year despite continued treatment,<sup>119</sup> suggesting that, whereas continued treatment may afford some protective effect, additional treatments may be needed for sustained effectiveness.

Although fluoxetine is the only antidepressant currently approved for BN, other SSRIs may also be effective. In a randomized, placebo-controlled trial, sertraline 100 (mg/d for 12 weeks) was found to be significantly more effective than placebo in reducing binge/purge behaviors in 20 female outpatients.<sup>120</sup> Fluvoxamine has been found effective in preventing relapse in patients with BN who had responded to inpatient behavioral psychotherapy.<sup>121</sup> In a single-blind trial comparing citalopram to fluoxetine, both agents resulted in significant improvement in eating psychopathology. Patients on fluoxetine displayed greater reduction in introjected anger, whereas those on citalopram demonstrated greater improvement in depressive feelings.<sup>122</sup> In comparison, another study found that use of the androgen receptor antagonist flutamide reduced craving and binge behaviors but not purging, whereas citalopram did not separate from placebo on these measures.<sup>123</sup> Among the newer, non-SSRI antidepressants, only bupropion has been studied in a controlled trial. Although bupropion was highly effective in reducing bingeing, there was a 5.8% incidence of seizure among study participants.<sup>124</sup> As a result, this agent is contraindicated in BN. As a class, antidepressants reduce binge eating by 61.4% (remission rate: 22%) and reduce purging by 58.9% (remission rate: 34%).<sup>125</sup> Unfortunately, up to 45% of patients who respond to pharmacotherapy may relapse in the first 6 months.<sup>126</sup>

Topiramate is becoming increasingly popular for

treating BN after two randomized, double-blind, placebo-controlled trials established efficacy.<sup>127,128</sup> Other agents that have been found effective in at least one double-blind trial include the 5-HT<sub>3</sub> antagonist ondansetron,<sup>129</sup> the opioid antagonist naltrexone,<sup>102</sup> and trazodone.<sup>130</sup>

Several studies have compared psychotherapy to pharmacotherapy and failed to find any advantage of combining medication with therapy versus therapy alone.<sup>131-133</sup> Results from two metaanalyses found that combination approaches were associated with higher remission rates, although adding medication to therapy increased the dropout rate.<sup>134,135</sup> In summary, the preference is to treat BN with psychotherapy, primarily CBT, or a combination of psychotherapy and medication.

### Eating Disorder Not Otherwise Specified

Because EDNOS is a nonspecific diagnostic category, treatment approaches depend on symptoms. The one category of EDNOS for which there appears to be a growing body of literature is BED. In general, the treatment resembles that for BN, with outcome measures defined by reduction in bingeing. The best-studied psychotropic agents used to treat BED and obesity are antidepressants and anticonvulsants. SSRIs have been found effective in double-blind trials for treatment of BED.<sup>136-138</sup> The anticonvulsant topiramate has also been found effective in double-blind trials for BED associated with obesity,<sup>139</sup> and a similar agent, zonisamide, was also associated with significant weight loss in a double-blind trial of obese adults.<sup>140</sup> Alternatively, agents specifically marketed for weight loss (including phentermine, sibutramine, and orlistat)<sup>141,142</sup> have been used to treat weight gain in overweight or obese patients with BED. Although several agents have been found effective for short-term weight loss, there are relatively few data on long-term efficacy with these agents. A review of the Cochrane database system found that, compared with placebo, the number of patients achieving 10% or more weight loss was 12% higher with orlistat and 15% higher with sibutramine in double-blind trials that lasted more than 1 year.<sup>143</sup> However, there was significant attrition in these studies, with an average of 33% of those on orlistat and 43% of those on sibutramine dropping out.

## TREATMENT OF EATING DISORDER ISSUES IN THE COMBAT ENVIRONMENT

No formal studies have been conducted of disorders of eating behavior among military personnel in a combat environment. Operation Enduring Freedom and Operation Iraqi Freedom are the first US military

actions in which women play a major part in combat; given their higher incidence in this group, eating disorders may become an increasingly important issue. Although significant evidence shows that abnormal

eating and weight-loss behaviors are more prevalent in the military than in the general population, the actual rates of eating-disorder diagnoses in the military are at or below the civilian rates. This discrepancy may be due to a variety of reasons, including the pronounced situational component in the military (such as the PFA) producing abnormal eating and dieting patterns, the fear of adverse career actions probably leading to underreporting, and the fact that those with severe eating disorders are screened out prior to enlistment or are discharged when their eating disorders become evident. Additionally, service members with an eating disorder may try to conceal it in order to deploy, because operational and combat experience can be an important step toward promotion.

It is likely that few service members with a recognized eating disorder will be deployed to a combat zone. Individuals who meet criteria for AN clearly should not deploy, and probably should not be on active duty, due to the high rates of morbidity and mortality in AN as well as specialized treatment requirements. Those with a history of BN or EDNOS whose symptoms are well controlled should be considered on a case-by-case basis.

A careful history and evaluation should be conducted prior to the decision allowing affected service members to deploy. Given the chronicity of eating, only those who display good insight, treatment compliance, adequate symptom control, and a general high function should be considered for deployment. Potential side effects and availability of the prescribed medication should be considered. If multiple psychotropic agents, antipsychotics, or anticonvulsants are required for symptom stabilization, the service member should not be deployed. In particular, it should be noted that topiramate is becoming increasingly popular for bulimia but carries a warning for anhidrosis and hyperthermia,<sup>144</sup> which would be particularly problematic in a desert environment. Additional factors to consider include assessment of strengths and weaknesses, support systems, past aggravating factors, comorbidity

with substance abuse or depression, motivation for deployment, anticipated job while deployed, the deployment site, and anticipated access to mental healthcare.

It is more likely that a service member with a previously concealed or controlled eating disorder will present in theater when symptoms become obvious to coworkers or impact the individual's ability to perform. Once again a careful assessment is needed to determine if the service member can remain in theater or should be returned. Factors to consider are similar to those above and include the severity of current symptoms, prior history, comorbidity, the individual's job, the location, access to care, and impact on the mission. Metabolic abnormalities and dehydration are of particular concern in strenuous or hot climates; thus, bulimic patients with such abnormalities should be medically evacuated.

Both positive and negative situational factors are associated with deployment, and their effects may vary on an individual basis. During deployment PFAs are suspended, so the pressure of the weigh-in and measurements are removed. In most deployment situations personnel have more time to exercise. Also, a hot climate affects appetite and food intake. Most dining facilities offer a variety of foods so that an individual can make healthy choices; however, some may find that the buffet style (usually including a dessert and ice cream bar) leads to overeating. The communal eating and living environment may make bingeing and purging less likely (although not impossible). Laxatives and diuretics are more difficult to obtain in the combat environment, thus further reducing purging options. Structured meal times can also either decrease the likelihood of overeating or increase the urge to binge. There are clearly fewer environmental cues such as food commercials and restaurants, although many areas have fast food restaurants that are open for extended hours. The separation from home environment and family may have either a positive or negative impact and should be individually assessed for each patient.

#### AREAS FOR FURTHER RESEARCH

Further research is necessary to improve the understanding of eating disorders in several areas. It is likely that the global war on terror will continue for some time, either as continued combat or with a US operational presence at widespread locations around the world. Given the known high incidence of abnormal eating behaviors in military personnel across services, military medical providers must understand how or if these conditions impact operational readiness and

performance. Anecdotally, it appears that most service members who deploy lose weight during the deployment, most likely in healthy ways from increased exercise and decreased food intake. Deployment may somehow be protective for eating disorders due to decreases in certain types of stress. Additionally, a primary symptom in eating disorders is an overconcern about physical appearance and self-esteem tied to appearance. It may be that in a combat zone these issues

are less important, and that personal satisfaction and feelings of reward derive from sources other than food or physical appearance. Other valuable research would be measuring the actual amount of weight loss during deployment, identifying any difference in weight loss among those who are and are not overweight prior to deployment, and determining whether the incidence of eating disorder behaviors is increased or decreased with deployment.

Another area of research interest is the potential

for comorbid development of posttraumatic stress disorder (PTSD) in patients with eating disorders. Both eating disorders<sup>23-27</sup> and adult PTSD<sup>145,146</sup> have been linked independently to a history of childhood trauma and sexual abuse. It is not known if there is an independent link between eating disorders and the development of PTSD. An additional area for further research is the effect, currently unknown, of combat trauma on the risk of developing an eating disorder.

## SUMMARY

Eating disorders are common among service members, and the military environment includes stressors that may contribute to unhealthy behaviors. Healthcare providers must be aware of the signs and symptoms of each disorder, capable of accurate diagnoses, and proactive in offering the available treatments, both medical and psychiatric. Vigilance is especially important with troops preparing for deployment, for these disorders may be exacerbated in theater, with

negative consequences for individuals, their units, and their missions. Despite significant long-term morbidity and mortality associated with these conditions, eating disorders are treatable. The military may be an advantageous environment to provide deterrents to unhealthy behaviors, structured support and treatment for affected individuals, and opportunities for further research to increase the understanding of these disorders.

## REFERENCES

1. Lindquist CH, Bray RM. Trends in overweight and physical activity among US military personnel, 1995–1998. *Prev Med.* 2001;32:57–65.
2. Centers for Disease Control and Prevention. *Overweight and Obesity: Selected US National Research Findings.* Available at: <http://www.cdc.gov/women/natstat/overwght.htm>. Accessed March 2, 2010.
3. Byrne S, McLean N. Eating disorders in athletes: a review of the literature. *J Sci Med Sport.* 2001;4:145–159.
4. Marshall JD, Harber VJ. Body dissatisfaction and drive for thinness in high performance field hockey athletes. *Int J Sports Med.* 1996;17:541–544.
5. McNulty PA. Prevalence and contributing factors of eating disorder behaviors in active duty service women in the Army, Navy, Air Force, and Marines. *Mil Med.* 2001;166:53–58.
6. Hudson JL, Hiripi E, Pope HG Jr, Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biol Psychiatry.* 2007;61:348–358.
7. Silverman JA. Sir William Gull (1819–1890). Limner of anorexia nervosa and myxoedema. An historical essay and encomium. *Eat Weight Disord.* 1997;2:111–116.
8. Gard MCE, Freeman CP. The dismantling of a myth: a review of eating disorders and socioeconomic status. *Int J Eat Disord.* 1996;20:1–12.
9. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 4th ed. Washington, DC: APA; 1994.
10. Dorian BJ, Garfinkel PE. The contributions of epidemiologic studies to the etiology and treatment of the eating disorders. *Psychiatric Ann.* 1999;29:187–196.
11. Andersen AE, Bowers WA, Watson T. A slimming program for eating disorders not otherwise specified. Reconceptualizing a confusing, residual diagnostic category. *Psychiatr Clin North Am.* 2001;24:271–280.



12. Hoek HW, van Hoeken D. Review of the prevalence and incidence of eating disorders. *Int J Eat Disord*. 2003;34:383–396.
13. O'Reardon JP, Peshek A, Allison KC. Night eating syndrome: diagnosis, epidemiology and management. *CNS Drugs*. 2005;19:997–1008.
14. Kress AM, Peterson MR, Hartzell MD. Association between obesity and depressive symptoms among US military active duty service personnel, 2002. *J Psychosom Res*. 2006;60(3):263–271.
15. Rodin G. The etiology of eating disorders: lessons from high-risk groups. *Psychiatr Ann*. 1999;29:181–182.
16. Bailer UF, Kaye WH. A review of neuropeptide and neuroendocrine dysregulation in anorexia and bulimia nervosa. *Curr Drug Targets CNS Neurol Disord*. 2003;2:53–59.
17. Klump KL, Kaye WH, Strober M. The evolving genetic foundations of eating disorders. *Psychiatr Clin North Am*. 2001;24:215–225.
18. Nacmias B, Ricca V, Tedde A, Mezzani B, Rotella CM, Sorbi S. 5-HT<sub>A</sub> receptor gene polymorphisms in anorexia nervosa and bulimia nervosa. *Neurosci Lett*. 1999;277:134–136.
19. Spigset O, Andersen T, Hägg S, Mjøndal T. Enhanced platelet serotonin 5-HT<sub>2A</sub> receptor binding in anorexia nervosa and bulimia nervosa. *Eur Neuropsychopharmacol*. 1999;9:469–473.
20. Levitan RD, Kaplan AS, Masellis M, et al. Polymorphism of the serotonin 5-HT<sub>1B</sub> receptor gene (HTR<sub>1B</sub>) associated with minimum lifetime body mass index in women with bulimia nervosa. *Biol Psychiatry*. 2001;50:640–643.
21. King M. The epidemiology of eating disorders. *Epidemiol Psychiatr Soc*. 1998;7:32–41.
22. Levine MP, Smolak L, Moodey AF, Shuman MD, Hessen LD. Normative developmental challenges and dieting and eating disturbances in middle school girls. *Int J Eat Disord*. 1994;15:11–20.
23. Connors ME, Morse W. Sexual abuse and eating disorders: a review. *Int J Eat Disord*. 1993;13:1–11.
24. Léonard S, Steiger H, Kao A. Childhood and adulthood abuse in bulimic and nonbulimic women: prevalences and psychological correlates. *Int J Eat Disord*. 2003;33:397–405.
25. Wonderlich SA, Brewerton TD, Jocic Z, Dansky BS, Abbott DW. Relationship of childhood sexual abuse and eating disorders. *J Am Acad Child Adolesc Psychiatry*. 1997;36:1107–1115.
26. Wonderlich SA, Crosby RD, Mitchell JE, et al. Eating disturbance and sexual trauma in childhood and adulthood. *Int J Eat Disord*. 2001;30:401–412.
27. Merrill LL, Newell CE, Thomsen CJ, et al. Childhood abuse and sexual revictimization in a female Navy recruit sample. *J Trauma Stress*. 1999;12:211–225.
28. Becker AE, Grinspoon SK, Klibanski A, Herzog DB. Eating disorders. *N Engl J Med*. 1999;340:1092–1098.
29. Birmingham CL. Hypercarotenemia [letter]. *N Engl J Med*. 2002;347:222–223.
30. Boland B, Beguin C, Zech F, Desager JP, Lambert M. Serum beta-carotene in anorexia nervosa patients: a case-control study. *Int J Eat Disord*. 2001;30:299–305.
31. Mitchell JE, Seim HC, Colon E, Pomeroy C. Medical complications and medical management of bulimia. *Ann Intern Med*. 1987;107:71–77.
32. Karim A, Ahmed S, Rossoff L. Pneumomediastinum simulating a panic attack in a patient with anorexia nervosa. *Int J Eat Disord*. 2003;33:104–107.

33. Casiero D, Frishman WH. Cardiovascular complications of eating disorders. *Cardio Rev*. 2006;14:227–231.
34. Mehler PS. Osteoporosis in anorexia nervosa: prevention and treatment. *Int J Eat Disord*. 2003;33:113–126.
35. Powers PS. Osteoporosis and eating disorders. *J Pediatr Adolesc Gynecol*. 1999;12:51–57.
36. Lucas AR, Melton LJ 3rd, Crowson CS, O'Fallon WM. Long-term fracture risk among women with anorexia nervosa: a population-based cohort study. *Mayo Clin Proc*. 1999;74:972–977.
37. Andersen AE, Watson T, Schlechte J. Osteoporosis and osteopenia in men with eating disorders. *Lancet*. 2000;355:1967–1968.
38. Wolfe BE, Metzger ED, Levine JM, Jimerson DC. Laboratory screening for electrolyte abnormalities and anemia in bulimia nervosa: a controlled study. *Int J Eat Disord*. 2001;30:288–293.
39. Crow SJ, Rosenberg ME, Mitchell JE, Thuras P. Urine electrolytes as markers of bulimia nervosa. *Int J Eat Disord*. 2001;30:279–287.
40. Devuyst O, Lambert M, Rodhain J, Lefebvre C, Coche E. Haematological changes and infectious complications in anorexia nervosa: a case-control study. *Q J Med*. 1993;86:791–799.
41. Pauporte J, Walsh BT. Serum cholesterol in bulimia nervosa. *Int J Eat Disord*. 2001;30:294–298.
42. Pritts SD, Susman J. Diagnosis of eating disorders in primary care. *Am Fam Physician*. 2003;67:297–304.
43. American Psychiatric Association. *Practice Guideline for the Treatment of Patients With Eating Disorders*. 2nd ed. Washington, DC: APA; 2000.
44. Patton GC. Mortality in eating disorders. *Psychol Med*. 1988;18:947–951.
45. Sullivan PF. Mortality in anorexia nervosa. *Am J Psychiatry*. 1995;152:1073–1074.
46. Eckert ED, Halmi KA, Marchi P, Grove W, Crosby R. Ten-year follow-up of anorexia nervosa: clinical course and outcome. *Psychol Med*. 1995;25:143–156.
47. Herzog W, Deter HC, Fiehn W, Petzold E. Medical findings and predictors of long-term physical outcome in anorexia nervosa: a prospective 12-year follow-up study. *Psychol Med*. 1997;27:269–279.
48. Ratnasuriya RH, Eisler I, Szmukler GI, Russell GF. Anorexia nervosa: outcome and prognostic factors after 20 years. *Br J Psychiatry*. 1991;158:495–502.
49. Zipfel S, Löwe B, Reas DL, Deter HC, Herzog W. Long-term prognosis in anorexia nervosa: lessons from a 21-year follow-up study. *Lancet*. 2000;355:721–722.
50. Herzog DB, Nussbaum KM, Marmor AK. Comorbidity and outcome in eating disorders. *Psychiatr Clin North Am*. 1996;19:843–859.
51. Keel PK, Mitchell JE. Outcome in bulimia nervosa. *Am J Psychiatry*. 1997;154:313–321.
52. Nielsen S. Epidemiology and mortality of eating disorders. *Psychiatr Clin North Am*. 2001;24:201–214, vii–viii.
53. Nielsen S, Moller-Madsen S, Isager T, Jorgensen J, Pagsberg K, Theander S. Standardized mortality in eating disorders—a quantitative summary of previously published and new evidence. *J Psychosom Res*. 1998;44:413–434.
54. Davidson DM. Anorexia nervosa in a serviceman: case report. *Mil Med*. 1976;141:617–619.
55. Voge VM, Yacavone D. Bulimia: an uncommon problem in aircrewmen—a case report. *Aviat Space Environ Med*. 1987;58:347–349.

56. Berg JS, Moore JL. A case of "eating disorder NOS": aeromedical implications of DSM-IV diagnostic criteria. *Aviat Space Environ Med.* 1996;67:157–160.
57. Sundgot-Borgen J. Risk and trigger factors for the development of eating disorders in female elite athletes. *Med Sci Sports Exerc.* 1994;26:414–419.
58. Lauder TD, Williams MV, Campbell CS, Davis GD, Sherman RA. Abnormal eating behaviors in military women. *Med Sci Sports Exerc.* 1999;31:1265–1271.
59. Lauder TD, Williams MV, Campbell CS, Davis G, Sherman R, Pulos E. The female athlete triad: prevalence in military women. *Mil Med.* 1999;164:630–635.
60. McNulty PA. Prevalence and contributing factors of eating disorder behaviors in active duty Navy men. *Mil Med.* 1997;162:753–758.
61. McNulty PA. Prevalence and contributing factors of eating disorder behaviors in a population of female Navy nurses. *Mil Med.* 1997;162:703–706.
62. Babar N, Alam M, Ali SS, et al. Anorexic behaviour and attitudes among female medical and nursing students at a private university hospital. *J Pak Med Assoc.* 2002;52:272–276.
63. Szweda S, Thorne P. The prevalence of eating disorders in female health care students. *Occup Med (Lond).* 2002;52:113–119.
64. Peterson AL, Talcott GW, Kelleher WJ, Smith SD. Bulimic weight-loss behaviors in military versus civilian weight-management programs. *Mil Med.* 1995;160:616–620.
65. Warner C, Warner C, Matuszak T, Rachal J, Flynn J, Grieger TA. Disordered eating in entry-level military personnel. *Mil Med.* 2007;172:147–151.
66. Carlton JR, Manos GH, Van Slyke JA. Anxiety and abnormal eating behaviors associated with cyclical readiness testing in a naval hospital active duty population. *Mil Med.* 2005;170:663–667.
67. Vitousek K, Watson S, Wilson GT. Enhancing motivation for change in treatment-resistant eating disorders. *Clin Psychol Rev.* 1998;18:391–420.
68. American Psychiatric Association. Practice Guidelines for the Treatment of Patients with Eating Disorders [revised]. *Am J Psychiatry.* 2000;157(suppl):1.
69. McKenzie JM, Joyce PR. Hospitalization for anorexia nervosa. *Int J Eat Disord.* 1992;11:235–241.
70. Agras WS. The consequences and costs of the eating disorders. *Psychiatr Clin North Am.* 2001;24:371–379.
71. Russell GF. Involuntary treatment in anorexia nervosa. *Psychiatr Clin North Am.* 2001;24:337–349.
72. Rigotti NA. Eating disorders. In: Carlson KJ, Eisenstat SA, eds. *Primary Care of Women.* St Louis, Mo: Mosby-Year Book; 1995: 443–449.
73. Becker AE, Hamburg P, Herzog DB. The role of psychopharmacologic management in the treatment of eating disorders. *Psychiatr Clin North Am.* 1998;5:17–51.
74. Golden NM, Iglesias EA, Jacobson MS, et al. Alendronate for the treatment of osteopenia in anorexia nervosa: a randomized, double-blind, placebo-controlled trial. *J Clin Endocrinol Metab.* 2005;90:3179–3185.
75. Nakahara T, Nagai N, Tanaka M, et al. The effects of bone therapy on tibial bone loss in young women with anorexia nervosa. *Int J Eat Disord.* 2006;39:20–26.
76. Mitchell JE, Peterson CB, Myers T, Wonderlich S. Combining pharmacotherapy and psychotherapy in treatment of patients with eating disorders. *Psychiatr Clin North Am.* 2001;24:315–323.

77. Russell GF, Szmukler GI, Dare C, Eisler I. An evaluation of family therapy in anorexia nervosa and bulimia nervosa. *Arch Gen Psychiatry*. 1987;44:1047–1056.
78. Lock J, le Grange D, Agras WS, Dare C. *Treatment Manual for Anorexia Nervosa: A Family Based Approach*. New York, NY: Guilford Press; 2001.
79. Bowers WA. Basic principles for applying cognitive-behavioral therapy to anorexia nervosa. *Psychiatr Clin North Am*. 2001;24:293–303, x.
80. Mark M, Rabinowitz J, Rabinowitz S, Gaoni B, Babur I, Danon Y. Brief treatment of anorexia nervosa in military personnel. *Hosp Community Psychiatry*. 1993;44:69–71.
81. McIntosh VV, Jordan J, Carter FA, et al. Three psychotherapies for anorexia nervosa: a randomized, controlled trial. *Am J Psychiatry*. 2005;162:741–747.
82. Levenson CW. Zinc regulation of food intake: new insights on the role of neuropeptide Y. *Nutr Rev*. 2003;61:247–249.
83. Su JC, Birmingham CL. Zinc supplementation in the treatment of anorexia nervosa. *Eat Weight Disord*. 2002;7:20–22.
84. Birmingham CL, Goldner FM, Bakan R. Controlled trial of zinc supplementation in anorexia nervosa. *Int J Eat Disord*. 1994;15:251–255.
85. Birmingham CL, Gritzner S. How does zinc supplementation benefit anorexia nervosa? *Eat Weight Disord*. 2006;11:e109–e111.
86. Szmukler GI, Young GP, Miller G, Lichenstein M, Binns DS. A controlled trial of cisapride in anorexia nervosa. *Int J Eat Disord*. 1995;17:347–357.
87. Attia E, Schroeder L. Pharmacologic treatment of anorexia nervosa: where do we go from here? *Int J Eat Disord*. 2005;37(suppl):s60–s63.
88. Bulik CM, Berkman ND, Brownley KA, Sedway JA, Lohr KN. Anorexia nervosa treatment: a systematic review of randomized controlled trials. *Int J Eat Disord*. 2007;40:310–320.
89. Halmi KA, Eckert E, LaDu TJ, Cohen J. Anorexia nervosa. Treatment efficacy of cyproheptadine and amitriptyline. *Arch Gen Psychiatry*. 1986;43:177–181.
90. Lacey JH, Crisp AH. Hunger, food intake and weight: the impact of clomipramine on a refeeding anorexia nervosa population. *Postgrad Med J*. 1980;56(suppl 1):79–85.
91. Ruggiero GM, Laini V, Mauri MC, et al. A single blind comparison of amisulpride, fluoxetine and clomipramine in the treatment of restricting anorectics. *Prog Neuropsychopharmacol Biol Psychiatry*. 2001;25:1049–1059.
92. Mitchell JE, Fletcher L, 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.
93. Kaye WH, Nagata T, Weltzin TE, et al. Double-blind placebo-controlled administration of fluoxetine in restricting- and restricting-purging-type anorexia nervosa. *Biol Psychiatry*. 2001;49:644–652.
94. Anderson IM, Parry-Billings M, Newsholme EA, Fairburn CG, Cowen PJ. Dieting reduces plasma tryptophan and alters brain 5-HT function in women. *Psychol Med*. 1990;20:785–791.
95. Walsh BT, Kaplan AS, Attia E, et al. Fluoxetine after weight restoration in anorexia nervosa: a randomized controlled trial. *JAMA*. 2006;295:2605–2612.
96. Cassano GB, Miniati M, Pini S, et al. Six-month open trial of haloperidol as an adjunctive treatment for anorexia nervosa: a preliminary report. *Int J Eat Disord*. 2003;33:172–177.

97. La Via MC, Gray N, Kaye WH. Case reports of olanzapine treatment of anorexia nervosa. *Int J Eat Disord.* 2000;27:363–366.
98. Hansen L. Olanzapine in the treatment of anorexia nervosa. *Br J Psychiatry.* 1999;175:592.
99. Vandereycken W. Neuroleptics in the short-term treatment of anorexia nervosa. A double-blind placebo-controlled study with sulphiride. *Br J Psychiatry.* 1984;144:288–292.
100. Vandereycken W, Pierloot R. Pimozide combined with behavior therapy in the short-term treatment of anorexia nervosa. A double-blind placebo-controlled cross-over study. *Acta Psychiatr Scand.* 1982;66:445–450.
101. Mondraty N, Birmingham CL, Touyz S, Sundakov V, Chapman L, Beumont P. Randomized controlled trial of olanzapine in the treatment of cognitions in anorexia nervosa. *Australas Psychiatry.* 2005;13:72–75.
102. Marrazzi MA, Bacon JP, Kinzie J, Luby ED. Naltrexone use in the treatment of anorexia nervosa and bulimia nervosa. *Int Clin Psychopharmacol.* 1995;10:163–172.
103. Crow S, Mussell MP, Peterson C, Knopke A, Mitchell J. Prior treatment received by patients with bulimia nervosa. *Int J Eat Disord.* 1999;25:39–44.
104. Thiels C, Schmidt U, Treasure J, Garthe R, Troop N. Guided self-change for bulimia nervosa incorporating use of a self-care manual. *Am J Psychiatry.* 1998;155:947–953.
105. Agras WS, Crow SJ, Halmi KA, Mitchell JE, Wilson GT, Kraemer HC. Outcome predictors for the cognitive behavior treatment of bulimia nervosa: data from a multisite study. *Am J Psychiatry.* 2000;157:1302–1308.
106. Pope HG Jr, Hudson JI, Jonas JM, Yurgelun-Todd D. Bulimia treated with imipramine: a placebo-controlled, double-blind study. *Am J Psychiatry.* 1983;140:554–558.
107. Agras WS, Dorian B, Kirkley BG, Arnow B, Bachman J. Imipramine in the treatment of bulimia: a double-blind controlled study. *Int J Eat Disord.* 1987;6:29–38.
108. Barlow J, Blouin J, Blouin A, Perez E. Treatment of bulimia with desipramine: a double-blind crossover study. *Can J Psychiatry.* 1988;33:129–133.
109. Hughes PL, Wells LA, Cunningham CJ, Ilstrup DM. Treating bulimia with desipramine. A double-blind, placebo-controlled study. *Arch Gen Psychiatry.* 1986;43:182–186.
110. Blouin AG, Blouin JH, Perez EL, Bushnik T, Zuro C, Mulder E. Treatment of bulimia with fenfluramine and desipramine. *J Clin Psychopharmacol.* 1988;8:261–269.
111. Walsh BT, Gladis M, Roose SP, Stewart JW, Stetner F, Glassman AH. Phenzelzine vs placebo in 50 patients with bulimia. *Arch Gen Psychiatry.* 1988;45:471–475.
112. Kennedy SH, Piran N, Warsh JJ, et al. A trial of isocarboxazid in the treatment of bulimia nervosa. *J Clin Psychopharmacol.* 1988;8:391–396.
113. Rothschild R, Quitkin HM, Quitkin FM, et al. A double-blind placebo-controlled comparison of phenelzine and imipramine in the treatment of bulimia in atypical depressives. *Int J Eat Disord.* 1994;15:1–9.
114. Walsh BT, Agras WS, Devlin MJ, et al. Fluoxetine for bulimia nervosa following poor response to psychotherapy. *Am J Psychiatry.* 2000;157:1332–1334.
115. 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.
116. Goldstein DJ, Wilson MG, Thompson VL, Potvin JH, Rampey AH Jr. Fluoxetine Bulimia Nervosa Research Group. Long-term fluoxetine treatment of bulimia nervosa. *Br J Psychiatry.* 1995;166:660–666.

117. Blouin J, Blouin A, Perez E, Barlow J. Bulimia: independence of antibulimic and antidepressant properties of desipramine. *Can J Psychiatry*. 1989;34:24–29.
118. Goldstein DJ, Wilson MG, Ascroft RC, al-Banna M. Effectiveness of fluoxetine therapy in bulimia nervosa regardless of comorbid depression. *Int J Eat Disord*. 1999;25:19–27.
119. Romano SJ, Halmi KA, Sarkar NP, Koke SC, Lee JS. A placebo-controlled study of fluoxetine in continued treatment of bulimia nervosa after successful acute fluoxetine treatment. *Am J Psychiatry*. 2002;159:96–102.
120. Milano W, Petrella C, Sabatino C, Capasso A. Treatment of bulimia nervosa with sertraline: a randomized controlled trial. *Adv Ther*. 2004;21:232–237.
121. Fichter MM, Krüger R, Rief W, Holland R, Döhne J. Fluvoxamine in prevention of relapse in bulimia nervosa: effects on eating-specific psychopathology. *J Clin Psychopharmacol*. 1996;16:9–18.
122. Leombruni P, Amianto F, Delsedime N, Gramaglia C, Abbate-Daga G, Fassino S. Citalopram versus fluoxetine for the treatment of patients with bulimia nervosa: a single-blind randomized controlled trial. *Adv Ther*. 2006;23:481–494.
123. Sundblad C, Landén M, Eriksson T, Bergman L, Eriksson E. Effects of the androgen antagonist flutamide and the serotonin reuptake inhibitor citalopram in bulimia nervosa: a placebo-controlled pilot study. *J Clin Psychopharmacol*. 2005;25:85–88.
124. Horne RL, Ferguson JM, Pope HG Jr, et al. Treatment of bulimia with bupropion: a multicenter controlled trial. *J Clin Psychiatry*. 1988;49:262–266.
125. Nathan PE, Gorman JM. *Treatments That Work*. 2nd ed. New York, NY: Oxford University Press; 2002: 560.
126. Walsh BT, Hadigan CM, Devlin MJ, Gladis M, Roose SP. Long-term outcome of antidepressant treatment for bulimia nervosa. *Am J Psychiatry*. 1991;148:1206–1212.
127. Hoopes SP, Reimherr FW, Hedges DW, et al. Treatment of bulimia nervosa with topiramate in a randomized, double-blind, placebo-controlled trial, part 1: improvement in binge and purge measures. *J Clin Psychiatry*. 2003;64:1335–1341.
128. Nickel C, Tritt K, Muehlbacher M, et al. Topiramate treatment in bulimia nervosa patients: a randomized, double-blind, placebo-controlled trial. *Int J Eat Disord*. 2005;38:295–300.
129. Faris PL, Kim SW, Meller WH, et al. Effect of decreasing afferent vagal activity with ondansetron on symptoms of bulimia nervosa: a randomised, double-blind trial. *Lancet*. 2000;355:792–797.
130. Pope HG Jr, Keck PE Jr, McElroy SL, Hudson JI. A placebo-controlled study of trazodone in bulimia nervosa. *J Clin Psychopharmacol*. 1989;9:254–259.
131. Goldbloom DS, Olmsted M, Davis R, et al. A randomized controlled trial of fluoxetine and cognitive behavioral therapy for bulimia nervosa: short-term outcome. *Behav Res Ther*. 1997;35:803–811.
132. Fichter MM, Leibl K, Rief W, Brunner E, Schmidt-Auberger S, Engel RR. Fluoxetine versus placebo: a double-blind study with bulimic inpatients undergoing intensive psychotherapy. *Pharmacopsychiatry*. 1991;24:1–7.
133. Mitchell JE, Pyle RL, Eckert ED, Hatsukami D, Pomeroy C, Zimmerman R. A comparison study of antidepressants and structured intensive group psychotherapy in the treatment of bulimia nervosa. *Arch Gen Psychiatry*. 1990;47:149–157.
134. Bacaltchuk J, Trefiglio RP, de Oliveira IR, Lima MS, Mari JJ. Antidepressants versus psychotherapy for bulimia nervosa: a systematic review. *J Clin Pharm Ther*. 1999;24:23–31.
135. Bacaltchuk J, Trefiglio RP, Oliveira IR, Hay P, Lima MS, Mari JJ. Combination of antidepressants and psychological treatments for bulimia nervosa: a systematic review. *Acta Psychiatr Scand*. 2000;101:256–264.

136. Arnold LM, McElroy SL, Hudson JI, Welge JA, Bennett AJ, Keck PE. A placebo-controlled, randomized trial of fluoxetine in the treatment of binge-eating disorder. *J Clin Psychiatry*. 2002;63:1028–1033.
137. Ricca V, Mannucci E, Mezzani S, et al. Fluoxetine and fluvoxamine combined with individual cognitive-behaviour therapy in binge eating disorder: a one-year follow-up study. *Psychother Psychosom*. 2001;70:298–306.
138. Guerdjikova AI, McElroy SL, Kotwal R, et al. High-dose escitalopram in the treatment of binge-eating disorder with obesity: a placebo-controlled monotherapy trial. *Hum Psychopharmacol*. 2008;23:1–11.
139. McElroy SL, Arnold LM, Shapira NA, et al. Topiramate in the treatment of binge eating disorder associated with obesity: a randomized, placebo-controlled trial. *Am J Psychiatry*. 2003;160:255–261.
140. Gadde KM, Franciscy DM, Wagner HR II, Krishnan KR. Zonisamide for weight loss in obese adults: a randomized controlled trial. *JAMA*. 2003;289:1820–1825.
141. Wilfley DE, Crow SJ, Hudson JI, et al. Efficacy of sibutramine for the treatment of binge eating disorder: a randomized multicenter placebo-controlled double-blind study. *Am J Psychiatry*. 2008;165:51–58. Epub December 3, 2007.
142. Bays H, Dujovne C. Pharmacotherapy of obesity: currently marketed and upcoming agents. *Am J Cardiovasc Drugs*. 2002;2:245–253.
143. Padwal R, Li SK, Lau DC. Long-term pharmacotherapy for obesity and overweight. *Cochrane Database Syst Rev*. 2004;3:CD004094.
144. Hulihan J, Ortho-McNeil Pharmaceutical. Important drug warning letter to healthcare professionals, December 18, 2003. <http://www.fda.gov/downloads/Safety/MedWatch/SafetyInformation/SafetyAlertsforHumanMedicalProducts/UCM169494.pdf>. Accessed March 2, 2010.
145. Clancy CP, Graybeal A, Tompson WP, et al. Lifetime trauma exposure in veterans with military-related posttraumatic stress disorder: association with current symptomatology. *J Clin Psychiatry*. 2006;67:1346–1353.
146. Lapp KG, Bosworth HB, Strauss JL, et al. Lifetime sexual and physical victimization among male veterans with combat-related post-traumatic stress disorder. *Mil Med*. 2005;170:787–790.

