CHAPTER 1

What Is the Problem?

Anorexia Nervosa

Beatrice is a 13-year-old girl who lives with her family and younger brother. About 6 months ago Beatrice decided to eat "more healthily" and stopped eating desserts and sweets. At the time Beatrice was at a normal weight and had been so her entire life to that point. Her parents did not intervene in her decision to stop eating sweets because they agreed that sweets were not necessary for her health. Over the following 2 months, Beatrice decided that bread and other complex carbohydrates were not healthy and began eating fewer of them. She started to read labels on all the foods that her mother bought and complained about eating many of them that she thought were two fatty. Although she said she was not trying to lose weight, Beatrice weighed herself daily and began keeping a record of her weight and the number of calories she consumed each day. Beatrice also spent a significant amount of time in front of her full-length bedroom mirror examining her body for any sign of fat. She was particularly focused on fat on her belly and thighs. Although Beatrice had started menstruating 2 years previously, her periods had recently ceased. As her parents became concerned about her weight loss, they tried to encourage her to eat, but she refused, often becoming tearful and yelling at them, which was very out of character for her. Her parents also noticed she spent less time with friends from school and avoided eating with the family if at all possible. Her parents took Beatrice to her pediatrician under her protest. The pediatrician found Beatrice

had lost 16 pounds from her last visit a year before and recommended that she eat more and regain the weight. Beatrice promised to do so, but she could not do it. Her parents continued to encourage her, but when she tried to eat she was overwhelmed with fear and anxiety and became tearful.

Relatively recent epidemiological studies suggest that the overall prevalence of AN in the United States is about 1–2% among all females. In teenage girls, the prevalence is 0.3–0.7% (Hoek & Hoeken, 2003; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011), with some evidence suggesting an increasing rate over the past half century (Keski-Rahkonen et al., 2007; Lucas, Beard, & O'Fallon, 1991). Clinical reports suggest that about 10% of cases of AN are in males, but the rate may actually be higher due to underdetection (Norris et al., 2012); estimates have ranged from as low as 0.3% to as high as 1:1 (Braun, Sunday, Huang, & Halmi, 1999; Hudson, Hiripi, Pope, & Kessler, 2007; Lock, 2008; Strober, Freeman, Lampert, Diamond, & Kaye, 2001). In younger persons, subthreshold presentations are more common, with an estimated prevalence rate of 1.5% in adolescent females and 0.1% in adolescent males (Hudson et al., 2007; Swanson et al., 2011). The clinical severity of these subclinical cases appears to be similar to that of those who meet full diagnostic criteria, however. While AN can begin in preadolescence (Joergensen, 1992; Lucas et al., 1991), the peak age of onset is between 14 and 18 years (Halmi, Brodland, & Loney, 1973). AN rarely begins after age 25 (Lucas et al., 1991).

Etiology and Risk Factors

No specific cause of AN has been identified. Some studies support a possible genetic basis. AN occurs at a rate that is about five times greater in families where another first-degree relative has had the disorder, and heritability estimates from twin studies are in the 30–75% range (Bulik et al., 2006). It appears that temperament and personality type may be risk factors for AN (Bulik et al., 2006; Klump et al., 2000), specifically perfectionistic, obsessive, and avoidant personality features. These temperaments and personality characteristics are also likely heritable (Cassin & von Ramson, 2005; Wade et al., 2008). One study found that early-childhood eating behavior (e.g., picky eating) was associated with AN in adolescence (Marchi & Cohen, 1990).

In addition to these genetic and early behavioral risks, psychosocial

challenges associated with adolescence are implicated in the etiology of AN. While it is clear that AN disrupts physical, emotional, and social development, it remains unclear if anxiety about taking on adolescent developmental challenges is the cause or result of the AN (Bruch, 1973; Crisp, 1997). The overall cultural and social context is also likely relevant to the development of eating disorders, specifically the impact of pressures related to thinness and appearance that can trigger extreme dieting in vulnerable individuals (Anderson-Fye & Becker, 2004; Attie & Brooks-Gunn, 1989; Killen et al., 1994; McKnight Investigators, 2003; Stice, 2002). These social pressures may also result in increased risks in non-Western groups as the "thin ideal" is transmitted to them (Gunewardene, Huon, & Zheng, 2001; Lake et al., 2000; Wildes & Emery, 2001). Participation in particular vocations and sports is also associated with an increased risk for AN. Ballet, gymnastics, wrestling, and modeling are examples of such activities.

Clinical Presentation and Course

According to the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013), the specific criteria for the diagnosis of AN are reduction of food intake that leads to weight loss and clinically significant low body weight; extreme fear of gaining weight or behaviors (i.e., not eating, overexercising, extreme dieting) that prevent weight gain; and a view of the self that is based primarily on low weight and a thin body shape. In younger persons, a refusal to acknowledge the impact of severe weight loss is also a common feature of AN (Couturier & Lock, 2006). In some persons with AN, the disorder can be accompanied by episodes of overeating (i.e., binge eating) and purgative behaviors (e.g., vomiting, laxative use, diuretic use, and compensatory excessive exercise). Although amenorrhea is a common result of malnutrition in women, it is not applicable to younger persons or boys and is no longer a diagnostic requirement in DSM-5 (Roberto, Steinglass, & Walsh, 2008).

Because children and adolescents are still developing the cognitive capacity to understand and express their thoughts and feelings, behaviors must often be interpreted as expressive of internal states and parental reports substituted for self-evaluation (Couturier et al., 2007). It is also less common for children and adolescents to binge-eat or purge (Peebles, Wilson, & Lock, 2006).

Most children and adolescents with AN come to the attention of mental health providers after a parent or pediatrician becomes concerned about unexplained weight loss or failure to gain expected weight commiserate with height. The behaviors and cognitions that precipitate AN are often insidious and difficult to parse from those associated with typical adolescent development. Thus, an adolescent may appear to be expressing normal adolescent concerns about physical appearance and health but in reality be initiating behaviors that will foment the development of AN. In the early stages it is almost impossible to detect the difference. Nonetheless, as restrictive eating, exercise, calorie counting, and fat avoidance become habitual, the fact that these are no longer explained by normal adolescent concerns about appearance becomes more apparent to those around them. Often the young person will become more irritable and temperamentally erratic as well as withdraw from friends and try to avoid being with family members, especially at mealtimes. At this point, the rate of weight loss often escalates and typically leads parents to contact their pediatrician (Pinhas, Morris, Crosby, & Katzman, 2011).

Over time, eating becomes more restrictive and avoidance of foods that contain fat or are perceived to be fattening increases. Sometimes the young person will claim to be not hungry or report experiencing physical pain or stomach upset to avoid eating. Behaviors such as excessive weighing, body checking, mirror time, calorie counting, food weighing, and ritualized eating patterns (e.g., special plates and utensils) often increase just as the amounts of the permitted foods decrease. Sometimes the young person will not be able to sit still or stop moving and will try to stand at all times. Others may drink water or chew calorie-free gum excessively to satisfy oral cravings, while others will restrict fluids to the point of dehydration. Some will become preoccupied with cookbooks and cooking, sometimes preparing elaborate meals for family members while refusing to eat themselves. Others will visit bakeries, grocery stores, and restaurants to see and smell the food but refuse to buy or eat any. As they lose weight, their ability to maintain a normal body temperature decreases and they will complain of feeling cold and wear many layers of baggy clothing. As they become increasingly malnourished they can appear depressed and anxious.

Differential Diagnosis and Comorbidity

When evaluating a young person for AN, it is important that other possible causes of weight loss and refusal to eat be considered. Common medical

reasons for weight loss include chronic infection, thyroid disease, Addison's disease, inflammatory bowel disease, connective tissue disorders, cystic fibrosis, peptic ulcer disease, disease of the esophagus, celiac disease, infectious diseases, disease of the small intestine, diarrhea, diabetes mellitus, and occult malignancies (Golden et al., 2003). For the most part, a thorough history and physical exam along with laboratory studies can rule out these possibilities. Psychiatric reasons for weight loss and restrictive eating include avoidant/restrictive food intake disorder (ARFID; Bryant-Waugh & Kreipe, 2012), bulimia nervosa (BN; Le Grange & Lock, 2007), and rumination disorder (Bryant-Waugh, Markham, Kreipe, & Walsh, 2010). A thorough psychiatric evaluation (see Chapter 3) should clarify whether the eating problem is best classified as AN or another type of eating problem.

Another challenge when diagnosing and treating persons with AN is the rate of comorbidity. In the National Comorbidity Survey Replication Adolescent Supplement (NCS-A), the lifetime rate of comorbidity with at least one other psychiatric disorder in AN was 55.2% (Swanson et al., 2011). The most common psychiatric disorders found associated in adults with AN were depression, social anxiety, separation anxiety, obsessive–compulsive disorder (OCD), generalized anxiety, and substance abuse. One of the more challenging is the co-occurrence of AN and OCD because they share obsessional preoccupations (e.g., overeating, food, weight, and shape obsessions) and compulsive behaviors (e.g., restricting and counting calories, overexercising, checking behaviors, mealtime rituals), making it difficult to differentiate these two disorders. In OCD, though, these preoccupations and compulsions are not exclusively focused on eating and weight. It appears that anxiety disorders are often present prior to and after recovery from AN (Bulik, Sullivan, Fear, & Joyce, 1997; Godart, Flament, Lecrubier, & Jeammet, 2000; Silberg & Bulik, 2005). It should be noted that some specific phobias (e.g., fear of swallowing) may lead to weight loss and be misdiagnosed as AN. In addition, while low mood often co-occurs with AN, especially when the person is at a low weight, this often improves with weight restoration. If low mood persists after weight restoration, treatment for depression may be indicated (Eisenberg, Wall, & Neumark-Sztainer, 2012).

A Brief History of AN Treatment

An early clinical description of "nervous consumption," a medical condition that appears similar to what would now be called AN was written in

1689 by a physician named Richard Morton. Sir William Gull in England and Charles Lasègue in France almost simultaneously named the disorder in 1874 ("anorexia nervosa" in English and *anorexie hystérique* in French) (Gull, 1874). At that time there was no definitive theory as to the cause of the disorder, but family factors were, as was the case with most psychiatric disorders, considered a likely etiological source. Also, treatment for psychiatric disorders at the time was largely institutionally based, that is, hospitalization in large asylums. This meant lengthy separations from family members who were, as a result, effectively excluded from treatment (McKenzie, 1992; Zhao & Encinosa, 2009). This trend continued well into the 20th century—many patients with AN were treated in hospitals between the 1980s and the 2000s. More recently, psychiatric hospital treatment, especially in the United States, has been replaced by specialized residential treatment programs (Frisch, Franko, & Herzog, 2006; Meads, Gold, & Burls, 2001; Twohig et al., 2016). As is the case with hospitalization (Byford et al., 2007; Gowers et al., 2007), there is no compelling evidence that these programs are more effective than outpatient care for most young patients with AN.

From the late 19th century until at least the middle of the 20th, the dominant approaches to the treatment of AN were psychoanalytic and psychodynamic. These approaches theorized that the symptoms of AN were the result of a range of unconscious conflicts (Thoma, 1967). An early pioneer in psychodynamic theory related to AN was Hilde Bruch, who saw the symptoms of AN as an attempt to manage experiences of neglect and being overcontrolled by parents. Food refusal was conceptualized as a means of self-preservation and self-assertion. Because parents were considered to be neglectful and intrusive, Bruch employed an individual approach aimed at restoring a sense of self and promoting increased autonomy from parents (Bruch, 1973, 1978).

From the late 1970s until today there has been a growing awareness of the possible role of families in *helping* their children with AN. This work began with the observations of Salvador Minuchin, who found structural family therapy helpful in a case series of adolescents (Minuchin, Rosman, & Baker, 1978). Minuchin's work theorized that AN symptoms emerged and were maintained by aberrant family processes he described as "psychosomatic" and consisted of enmeshment, rigidity, overprotectiveness, and conflict avoidance. However, these family processes have yet to be substantiated by systematic research. Further refinements of family therapy's possible role in treating AN have taken place over the past 30 years, initially by researchers at the Institute of Psychiatry and Maudsley

Hospital. Their approach was not based on a view of the family as in any way dysfunctional, but instead designed to utilize parental skills to disrupt the maintaining behaviors of AN (Dare & Eisler, 1992). This approach (manualized as family-based treatment [FBT]) has been refined and studied in a number of randomized clinical trials and shown to be effective for adolescent AN (Lock, 2015).

Chapter 3 reviews what is currently known about efficacious assessments and treatments for AN, particularly AFT. In summary, there are few studies of treatment for AN that support a specific treatment for the disorder, especially in adults (Berkman, Lohr, & Bulik, 2007; Hay, 2013). Several outpatient interventions have been studied for use with adolescent AN. Randomized trial evaluations of family treatments suggest that family therapy, specifically manualized FBT, are efficacious (Eisler et al., 2000; Le Grange, Eisler, Dare, & Russell, 1992; Lock, Agras, Bryson, & Kraemer, 2005; Lock et al., 2010; Robin et al., 1999; Russell, Szmukler, Dare, & Eisler, 1987). Although individual therapy was not as effective as family therapy in these studies (Robin et al., 1999), individual approaches were nonetheless beneficial and therefore could be offered for patients when FBT is not an acceptable or tenable option (Lock et al., 2010). In particular, manualized AFT focusing on individuation and self-efficacy was found to be useful (Fitzpatrick et al., 2010). Some data suggest that CBT might be effective for adolescent AN, but the database is even more limited and no randomized trials have been conducted (Dalle Grave et al., 2013).

Outcomes in AN

Unfortunately, the outcomes for persons who develop AN are extremely variable. Some studies suggest that if the disorder lasts for more than 5 years, recovery is unlikely (Hay, Touyz, & Sud, 2012). It is estimated that 7–15% of those with AN will develop a severe and enduring form of the disorder. Mortality in AN is one of the highest of any psychiatric disorder, with rates as high as 18% reported in some samples (Steinhausen, 2002). Death is most often secondary to medical complications of starvation (50%) or suicide (50%) (Arcelus, Mitchell, Wales, & Nielsen, 2011). However, the prognosis for adolescents with AN is better than for adult populations (Fichter, Quadflieg, & Hedlund, 2006; Herperz-Dahlmann et al., 2001). Full recovery rates range from 30 to 60% with early intervention (Treasure & Russell, 2011).