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CHAPTER 1

Diagnosis, Assessment, and Treatment Planning for Anorexia Nervosa

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Overview of Anorexia Nervosa

Description and Diagnostic Criteria

The term "anorexia nervosa" (AN) was first introduced into the medical literature by Sir William Gull in 1874 (Gull, 1874). However, an illness strikingly similar to AN appears in various historical accounts of fasting girls and Catholic saints dating back to the 14th century (Keel & Klump, 2003). Across accounts, common features have included deliberate self-starvation resulting in low weight, denial of the seriousness of weight loss, excessive activity, amenorrhea, and, in modern Western cultures, a morbid fear of weight gain or being fat. The DSM-IV-TR (American Psychiatric Association [APA], 2000, p. 589) criteria for AN include:

- A. Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g., weight loss leading to maintenance of body weight less than 85% of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected).
- B. Intense fear of gaining weight or becoming fat, even though underweight.
- C. 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.
- D. In postmenarcheal females, amenorrhea, i.e., the absence of at least three consecutive menstrual cycles. (A woman is considered to have amenorrhea if her periods occur only following hormone, e.g., estrogen, administration.)

DSM-IV-TR also includes subtype specifications for individuals who do not regularly engage in binge-eating or purging behaviors (the restricting subtype) as well as those who do regularly engage in binge-eating or purging behaviors (the binge-purge sub-type).

In addition to patients who meet full DSM-IV-TR criteria for AN are many patients who meet partial criteria and would be diagnosed with an eating disorder not otherwise specified (EDNOS) (Dalle Grave & Calugi, 2007). Examples include females who meet all criteria for AN with the exception that they do not experience amenorrhea as well as individuals whose weight loss, though significant, is not considered to be below a "minimally normal" threshold for their age and height. Importantly, the DSM-IV-TR does not provide a strict threshold for low weight. Instead, 85% below expected weight is provided as a guideline, and no standard approach for determining expected weight has been provided. Thus, a patient diagnosed with DSM-IV-TR AN in one setting might receive a diagnosis of EDNOS in another, depending on how "low weight" is defined (Thomas, Roberto, & Brownell, 2009).

Differential Diagnosis

Medical conditions leading to significant weight loss should be excluded when making a diagnosis of AN, because low weight in these conditions is not deliberate. Such general medical conditions may include, but are not restricted to, gastrointestinal diseases, brain tumors, and acquired immunodeficiency syndrome (AIDS) (APA, 2000). Gastrointestinal disorders may be more common among individuals with AN due to the consequences of starvation and purging on gastrointestinal function. Thus, a second feature that may distinguish patients with a diagnosis of AN is the presence of body image disturbance in the forms of fear of weight gain or denial of the seriousness of low weight. Significant body image disturbance and reluctance to gain weight should be absent in individuals whose weight loss is attributable to a gastrointestinal disease.

Mental disorders that lead to significant weight loss also should be considered when making a diagnosis of AN. Major depressive disorder can be associated with significant weight loss and is a common comorbid disorder for individuals with AN. The primary distinction is whether or not weight loss is intentional. In major depressive disorder weight loss is unintentional and attributable to a loss in appetite. Importantly, individuals with AN often report a loss of appetite that may be secondary to prolonged starvation or problems in interoceptive awareness. However, these individuals may demonstrate resistance to the idea that they need to gain weight in order to be healthy, which would distinguish them from those with a sole diagnosis of major depressive disorder. Individuals with schizophrenia who have delusions regarding food and eating also may lose substantial weight. For example, an individual with a paranoid delusion that her husband is trying to poison her may refuse to eat food in the household, resulting in substantial weight loss. However, the motivation behind food refusal would differ between this individual and one with AN. A patient with AN may express neardelusional beliefs regarding the effects of eating on her weight or shape; however, the theme of beliefs will center around weight and shape concerns.

Brief Overview of Research

Epidemiology

AN affects approximately 1 in 200 girls and women over their lifetime and 1 in 2,000 boys and men (APA, 2000). Two recent population-based epidemiological studies have

reported higher lifetime prevalence estimates for AN in women, including a lifetime prevalence of 0.9% in the United States (Hudson, Hiripi, Pope, & Kessler, 2007) and 2.2% in Finland (Keski-Rahkonen et al., 2007). In addition, one recent study reported a less dramatic, though still statistically significant, gender difference in prevalence between women and men in the United States (Hudson et al., 2007). Results from these studies may reflect detection of cases that are missed when ascertainment is based on clinical referral (Keski-Rahkonen et al., 2007). Alternatively, results may reflect an increasing prevalence of the syndrome. A meta-analysis of incidence studies indicated that the number of new cases per 100,000 population per year increased over the 20th century (Keel & Klump, 2003), suggesting that the disorder has become increasingly common. However, the size of this increase, though statistically significant, was modest, reflecting the extent to which AN remains a relatively rare illness rather than an epidemic, as sometimes suggested in the popular media.

Associated Features and Comorbidity

Individuals with AN often display disturbances in affect, including depressed mood and anxiety. Both mood and anxiety disorders are more common among individuals with AN compared to age-matched comparison samples (Hudson et al., 2006). Some characteristics of these comorbid disorders may be explained or exacerbated by the effects of starvation in AN. Specifically, depressed mood, anhedonia, and insomnia may result from malnutrition (Keys, Brozek, Henschel, Mickelson, & Taylor, 1950). Similarly, preoccupations with food and rituals around food and eating may emerge as a consequence of starvation (Keys et al., 1950). Thus, a diagnosis of a mood or anxiety disorder should be made when the symptoms do not appear to be explained by starvation and do not appear to be related to the primary diagnosis of AN. For example, compulsions related to the need to repeatedly calculate caloric intake and expenditure may be better conceptualized as a feature of AN, whereas compulsions related to the need to keep belongings in a certain order may be better conceptualized as a feature of obsessive–compulsive disorder or obsessive–compulsive personality disorder.

The diagnosis of social phobia may be particularly challenging in AN because some patients may report discomfort when eating in public for fear that they will be judged for eating too little. In such instances, the fear of judgment may not be irrational or excessive, given that friends and family sometimes adopt the role of "food police" when they are with a loved one who suffers from AN. Patients may also report a fear of being judged for eating too much when eating in public. If this fear centers on others observing their binge-eating episodes, it may not represent an irrational feargiven that binge-eating episodes, by definition, involve an amount of food that is definitely more than others would eat. Notably, patients with AN may fear judgment for eating too much even when they are eating a normal or small amount of food. In these instances, the fear may be judged excessive; however, the content of the fear may be better explained by a diagnosis of AN, given that individuals with the disorder may have disrupted perceptions of what constitutes a normal amount of food and would judge themselves as eating excessively even if no one else were observing them. Importantly, many patients with AN endorse premorbid fears of eating in public related to concerns of being critically evaluated or judged by others in ways that are not thematically related to others judging what or how much they eat. Instead, they are concerned that others will experience disgust in simply watching them engage in the act of eating. In such cases, the relationship between anxiety disorders and AN may reflect shared risk factors for these syndromes (Keel, Klump, Miller, McGue, & Iacono, 2005).

Cultural Influences

A recent review of eating disorders in a cross-cultural context (Keel & Klump, 2003) indicated that AN was not a culturally bound syndrome, although certain features of the illness appear to be culturally bound. Specifically, the endorsement of fear of gaining weight or becoming fat as a motivation for starvation appeared to be restricted to individuals exposed to ideals from a modern Western context. Of note, this feature was not included in the description of AN when it was first introduced into the medical literature in the late 19th century. However, research suggests that individuals who meet all criteria for AN with the exception of criterion B may have a less severe presentation and a better prognosis compared to those who meet full criteria (Strober, Freeman, & Morrell, 1999). Thus, debate continues on the centrality of this feature to the definition of the syndrome.

Significant differences have been observed between ethnic groups in prevalence estimates and incidence rates for AN. Research within the United States has indicated that the lifetime prevalence of AN is significantly lower in African American women than in European American women (Striegel-Moore et al., 2003). Similarly, research in Curacao has supported significant differences in the incidence of AN in the white/ mixed population versus the black population (Hoek et al., 2005), with an incidence of 9.08 per 100,000 population per year versus 0 per 100,000 population per year, respectively. Overall, the incidence of AN in the mixed/white population of Curacao was similar to that observed in the United States and the Netherlands (Hoek et al., 2005).

Gender Influences

As noted in the section on epidemiology, AN is approximately 10 times more common in women compared to men. Some of this prevalence difference can be understood in terms of gender differences in body image ideals. In modern Western cultures, thinness represents an aesthetic ideal for women whereas muscularity represents an aesthetic ideal for men. Of interest, a preponderance of cases in women appears to be present in cross-historical and cross-cultural descriptions of the syndrome (Keel & Klump, 2003), suggesting that factors other than culturally bound ideals of beauty may contribute to the observed gender difference.

An emerging line of research is beginning to examine the role of gonadal steroid hormones in contributing to possible sex differences in risk for developing eating disorders (EDs). This work has demonstrated that genetic effects on the development of EDs are activated at puberty (Klump, Burt, McGue, & Iacono, 2007) and that prenatal exposure to testosterone may serve an organizational role in protecting individuals from the development of disordered eating (Culbert, Breedlove, Burt, & Klump, 2008). Of note, these studies have examined a broadly defined phenotype of disordered eating rather than AN.

Course and Outcome

AN has a variable course. A minority of patients achieves remission early in the course of illness (i.e., within 1 year) and sustains recovery throughout life. This course appears to be most likely to occur in individuals diagnosed at a younger age, who experience a shorter delay between onset of illness and initiation of treatment (Steinhausen, 2002). Indeed, the only evidence-based treatment identified for AN, a specific form ("Maudsley model") of family-based therapy (Russell, Szmukler, Dare, & Eisler, 1987), has demonstrated efficacy in the treatment of children and adolescents (Keel & Haedt, 2008). In contrast, no evidence-based treatment has been consistently supported for the treatment of AN in adults (Bulik, Berkman, Brownley, Sedway, & Lohr, 2007; Wilson, Grilo, & Vitousek, 2007). Although a better prognosis is associated with an earlier age of onset, most cases first develop in mid- to late adolescence (APA, 2000).

With longer durations of follow-up, such as 10–20 years following diagnosis, just under half of patients achieve full recovery, another third remain symptomatic but demonstrate some improvement, and 20% remain chronically ill (Steinhausen, 2002). Complicating the attempt to interpret findings across studies is the variability in how recovery is defined, with some studies defining full recovery by 8 consecutive weeks of no or minimal symptom levels (Herzog et al., 1999). In addition, there is variability in how changing clinical presentation is defined. Longitudinal studies suggest that a majority of patients who do not recover from the restricting subtype of AN go on to develop binge-eating or purging behaviors (Eddy et al., 2002). Among these, some experience weight gain resulting in a shift in diagnosis (i.e., "diagnostic crossover") from AN to bulimia nervosa (BN). Although these individuals may be considered "improved" because they no longer meet full criteria for the binge–purge subtype of AN, it is difficult to consider them recovered. Similar issues emerge for those whose illness changes over time to meet partial criteria for AN. Such individuals may be viewed as partially recovered (Herzog et al., 1999), or they may be viewed as having an EDNOS.

Mortality has been observed in approximately 1 in 20 patients (Steinhausen, 2002; Sullivan, 1995) across studies, reflecting a standardized mortality ratio of approximately 10.0—or a 10-fold increase in risk of premature death (Keel et al., 2003; Löwe et al., 2001). Primary causes of death include the physical consequences of starvation and suicide (Nielsen et al., 1998). Predictors of fatal outcome include poor psychosocial functioning, longer duration of follow-up, and severity of alcohol use disorders (Keel et al., 2003).

Psychobiology

Symptoms and associated features of AN can have a profound impact on neuroanatomical structures and function. Several studies have demonstrated brain matter reductions in patients with AN, compared to controls, that appear to result from starvation (Keel, 2005). Recent work has focused on reductions in the right dorsal anterior cingulate cortex (ACC) that are related to both weight loss and weight recovery and prospectively predict eating disorder outcome, independently of body mass index (BMI) (McCormick et al., 2008). This region is of particular interest because it is a phylogenetically recent structure, containing a category of spindle-shaped neurons distinct to humans and great apes. This structure integrates cognitive and emotional information—

particularly with regard to reward-based learning. In a study comparing women ill with AN, recovered from AN, and healthy control women, Uher and colleagues (2003) found increased activation of the dorsal ACC specifically in response to food stimuli in recovered women, compared to both controls and ill patients, and increased dorsal ACC activation in women with a lifetime history of AN (ill and recovered), compared to controls. Consistent with structural changes related to weight recovery reported by McCormick and colleagues (2008), Uher and colleagues (2003) found that dorsal ACC activity was positively correlated with current body weight-explaining activation differences between recovered and ill patients. However, this finding does not explain differences found between recovered and control participants who did not differ in BMI, nor does it explain why differences were specific to food stimuli versus aversive emotional stimuli. Overall, results suggest some potential involvement of the dorsal ACC in the processing of information, which may leave individuals vulnerable to develop AN. This hypothesis is supported by the prospective association found between right dorsal ACC volume following weight recovery and risk for relapse in patients treated for AN (McCormick et al., 2008).

Considerable work has focused on neurotransmitters and neuropeptides that regulate eating and weight. Studies generally find reduced levels of neurochemicals that inhibit food intake (e.g., serotonin, leptin, melanocortin-stimulating hormone, and brain-derived neurotrophic factor) and increased levels of neurochemicals that promote food intake (e.g., ghrelin and neuropeptide-Y) in patients with AN, compared to controls (Favaro, Monteleone, Santonastaso, & Maj, 2008; Keel, 2005). Thus, when differences have been found, they have suggested that the bodies of patients with AN may respond to starvation by reducing signals that might inhibit food intake and increasing signals that would stimulate food intake. This interpretation is supported by normalization of most of these alterations with weight restoration (Favaro et al., 2008).

The serotonin system has received considerable attention in the search to understand the psychobiology of AN and has produced findings that deviate somewhat from that described for other neurochemicals. Specifically, serotonin levels do appear to be reduced during illness with AN. However, some evidence suggests elevated activity of this neurotransmitter system following weight recovery (Favaro et al., 2008). One interpretation of this pattern of findings is that individuals vulnerable to developing AN may have increased serotonin receptor activity, particularly the serotonin 1A receptor, which contributes to premorbidly higher levels of anxiety and harm avoidance. When individuals with these features initiate a weight loss diet, they may find that reducing their food intake helps to regulate their anxiety (Kaye, Frank, Bailer, & Henry, 2005). Of note, a key challenge to this model is that patients who are actively ill with AN report high levels of anxiety and harm avoidance (Klump et al., 2004), suggesting that starvation does not ameliorate these features.

Behavior and Molecular Genetic Findings

Family studies support the hypothesis that AN runs in families affected by eating disorders (Becker, Keel, Anderson-Fye, & Thomas, 2004). There is also evidence of shared familial transmission of obsessive–compulsive personality disorder (Lilenfeld et al., 1998) and anxiety disorders (Keel, Klump, et al., 2005). Twin studies further support the role of genes in explaining why AN runs in families. Specifically, twin concordance for broadly defined AN is greater in monozygotic compared to dizygotic twins (Becker et al., 2004). Molecular genetic studies have pointed to a number of possible genetic variations that may be linked to risk for development of AN. However, most findings have not been replicated. An exception to this pattern are findings related to the serotonin 2A receptor gene (Becker et al., 2004; Klump & Culbert, 2007), for which multiple independent association studies have supported increased A allele frequency in probands with AN. The functional significance of this finding has yet to be determined.

Risk and Maintenance Factors

Some risk factors for the development of AN have been covered in previous sections, specifically those concerning epidemiology, psychobiology, and genetic findings. For example, being an adolescent or young adult female represents a risk factor for the development of AN. Genes confer risk for developing the disorder as well, though it is unclear exactly which genes are key in the diathesis or what these genes influence. A vulnerability to perturbations in the function of the right dorsal ACC also may increase risk for developing the disorder. In support of AN as a neurodevelopmental disease, several studies have demonstrated a link between obstetric complications during the perinatal period and later development of AN, with a recent study demonstrating that the number of obstetric complications was inversely associated with age of AN onset (Favaro, Tenconi, & Santonastaso, 2006).

Beyond these considerations, there has also been a search to understand psychosocial risk factors for the development of AN. Perfectionism has been associated specifically with the development of AN and related syndromes in retrospective (Fairburn, Cooper, Doll, & Welch, 1999) and prospective longitudinal studies (Tyrka, Waldron, Graber, & Brooks-Gunn, 2002). Most other studies have examined prospective risk factors for the development of disordered eating, which is heavily weighted toward syndromes characterized by binge eating at normal or elevated weight, or have been cross-sectional studies that examined correlates of AN (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). This latter set of studies cannot provide information regarding the temporal association between a posited risk factor and the development of AN. Thus, correlates may reflect consequences of the illness. Among a host of correlates that may contribute to risk for developing AN are a parenting style marked by high concern, difficulties with sleeping or eating during infancy and early childhood, childhood anxiety disorders, negative self-evaluation, and adverse life events (Jacobi et al., 2004).

Assessment

Interview at Intake Assessment

Clinical interviews are a central part of any intake assessment because they identify the problem for which patients are seeking treatment, and they form the basis of a treatment plan. This interview also is crucial for determining whether a given treatment setting can offer the services needed for the patient or whether a referral to another treatment facility would be needed. Different approaches may be employed for the intake interview, ranging from an unstructured case-history interview to a structured diagnostic interview. In practice, clinicians may adopt a combination of these approaches to

obtain information about patient background (age, education, employment, relationship status, family history, medical history, legal history) and life events (e.g., transitions that may have preceded the onset of the eating disorder, traumatic events) as well as more detailed information about specific mental disorders that might be the focus of treatment.

In addition to structured interviews designed to obtain diagnoses across of range of mental disorders, such as the *Structured Clinical Interview for DSM-IV Axis I Disorders* (SCID; First, Spitzer, Gibbon, & Williams, 1996), there are also structured clinical interviews designed specifically for the assessment of eating disorders, such as the *Eating Disorders Examination* (Fairburn & Cooper, 1993). Structured clinical interviews require intensive training in order to be administered reliably. Because of this requirement, full structured clinical interviews are most often obtained within research settings. Outside of a research setting, clinicians may prefer to supplement unstructured clinical interviews with psychometrically validated self-report assessments (discussed below).

Key features to assess during an interview to determine presence of AN include:

- 1. Current body weight and height, objectively measured
- 2. Attitudes about gaining weight, if underweight
- 3. Perception of current weight/amount of body fat, influence of weight and shape on self worth, concern over medical consequences of low weight if weight is extremely low
- 4. If female and after the age of menarche, pattern of current menstrual cycles and use of hormonal contraception

In addition to assessing for these features to evaluate the presence of AN, it is important to assess related eating disorder features, including:

- 1. Presence and frequency of binge-eating episodes—that is, episodes in which the person feels a loss of control over eating and consumes more food than most people would under similar circumstances (excluding fasting behavior as a circumstance surrounding binge eating)
- 2. Presence of purging behavior, including use and frequency of self-induced vomiting, ipecac, laxatives for weight control, diuretics for weight control, enemas, or omission of insulin if diabetic
- 3. Presence of nonpurging weight control behaviors such as frequency, period, and intensity of fasting and exercise

Misuse of prescription or over-the-counter medications to promote weight loss,

Oincluding diet pills, herbal agents, stimulants, or medications that stimulate the thyroid

Although weight and height can be objectively measured and signs of purging may be detected through physical examination, detection of other features relies on accurate self-report. Patients with AN are not always able or willing to provide accurate information about their illness for a variety of reasons including, resistance to treatment that might result in weight gain, fear of being stigmatized, and genuine belief that their thoughts and behaviors are healthy. Clinicians should be aware of the impact of secrecy or denial on accuracy of assessments that rely on patients as the sole source of information. Repeating assessments throughout treatment is a good way to recheck information because patients may become more willing to reveal symptoms as they improve and develop greater trust in their therapists. Repeating assessments also is crucial for determining whether patients are making progress in treatment.

Following thorough assessment of eating disorder features, including onset and course of disordered eating symptoms leading up to the intake assessment, it is important to assess for presence of comorbid syndromes that may require clinical attention. As noted above, AN is associated with elevated risk of mortality due to suicide (Keel et al., 2003). Thus, it is important to evaluate for the presence of passive thoughts of death, active suicidal thoughts, plans, availability of means, and intentions—whether or not the patient endorses depressed mood or meets criteria for a major depressive episode. Given that comorbid substance use disorders significantly increase the risk of fatal outcome (Keel et al., 2003), a careful assessment of drug and alcohol use should be completed as well. Information regarding evaluation of medical stability is provided in the following section.

Recent work has supported the utility of motivational interviewing during intake assessment as a predictor of changes during treatment and maintenance of changes after treatment (Geller, Drab-Hudson, Whisenhunt, & Srikameswaran, 2004). Proponents of this approach note that motivational interviewing is useful both for assessment and for enhancing outcomes by addressing the need for patients to be in the action phase of making a change before therapists implement interventions designed to effect changes.

Medical Evaluation

A medical evaluation is advisable for any patient diagnosed with AN, given the risk of medical complications associated with the illness. This type of evaluation is particularly important for patients with low weight (BMI < 16.0) or frequent purging (purging on a daily basis) who may require inpatient treatment to achieve medical stabilization. In addition to objective assessment of height and weight, medical evaluations should include:

- 1. Vital signs (pulse, blood pressure, temperature, respiration)
- 2. Electrolytes, glucose, calcium, magnesium, phosporus
- 3. Amylase

4. Complete blood count with differential

- 5. Thyroid function tests (T3, T4, and TSH)
- 6. Albumin, transferrin
- 7. BUN/creatinine
- 8. Urinalysis, stool guaiac
- 9. Liver function tests (SGOT, SGPT, bilirubin)
- 10. Bone densitometry
- 11. Electrocardiogram

In cases of unstable vital signs and EKG abnormalities or very low potassium levels (hypokalemia) or other signs of medical instability (kidney or liver dysfunction), inpatient treatment is warranted to achieve medical stability. Inpatient treatment is also warranted when weight is extremely low and cannot be safely normalized on an outpatient basis. Thresholds for determining treatment setting are discussed in more detail in the treatment planning section.

Psychological Assessment

Several widely used self-report measures can be administered to determine psychological functioning related to eating disorders and other disorders. Table 1.1 provides a list of measures and what they are designed to assess based on information provided in Carter, McFarlane, and Olmsted (2004). This list is not exhaustive but does provide an indication of some of the most widely used measures and their length. As noted above, it is important to repeat assessments to determine whether improvements are occurring with treatment, and this is more feasible with shorter assessments. Measures included in Table 1.1 have reasonably straightforward scoring systems and interpretation. However, it is worth noting that several measures use reverse-scored items to avoid response pattern bias. In addition, one of the more widely used eating disorder assessments, the Eating Disorders Inventory (EDI), measures features specific to eating disorders (in three subscales) as well as associated personality and psychopathology features (in five subscales). For all self-report measures, it is important to determine that a patient has an adequate reading level (usually eighth grade) to complete these measures.

Nutritional Assessment

A referral to a dietician is an important component of assessment and is crucial for developing a treatment plan (see Treatment Team Members below). A nutritional assessment can establish the patient's current pattern of eating and rules around what he/ she eats, when he/she eats, and how much he/she eats. This assessment can also focus on what physical sensations the patient experiences before, during, and after eating and how he/she interprets these signals. For example, does the patient interpret feelings of hunger as being "good" or as a sign that he/she is losing weight rather than as a signal that it is time to eat? Do feelings of hunger trigger a fear of losing control over eating? These experiences suggest a perturbation in the association between biological drives to eat and interpretation of these drives, which may contribute to disorderedeating behaviors. How quickly does a patient feel full once he/she commences eating? Premature feelings of extreme fullness may reflect delays in gastric emptying that are a consequence of long periods of fasting. These feelings may contribute to reduced food intake or self-induced vomiting. Does the patient believe certain myths regarding the effects of food on body weight or composition? What are the patient's feelings and thoughts (e.g., rules) about different food groups (carbohydrates, fats, dairy)? Does the patient follow a special diet for medical, religious, or cultural reasons?

There is likely to be considerable overlap in content of assessment for planning nutritional rehabilitation and psychological intervention. However, it is important to note that a psychologist may help a patient notice and challenge patterns of thinking about his/her eating, weight, and self-worth (e.g., dichotomous thinking—"I am only a person of worth if my weight remains below 100"), whereas a dietician's role is to provide healthy eating guidelines and to serve as the expert on what patterns of food intake result in healthy weight and nutritional status.

| | | , | |
|---|--|---------------------------------|---|
| Measure (authors) | Construct assessed | Age | Scoring and interpretation |
| Eating Attitudes Test (EAT) (Garner & Garfinkel, 1979); EAT-26 (Garner, Olmsted, Borh & Garfinkel, 1982) | Anorexia nervosa symptoms Dieting (EAT-26) Bulimia (EAT-26) Oral Control (EAT-26) | ≥ 16 years | Items (40 or 26) with responses ranging from "never" to "always" on 6-point Likert scale; least pathological responses receive 0 points and remaining items scored 1, 2, and 3 to denote increasing severity; on EAT-26, cutoff > 20 indicative of an eating disorder |
| Eating Disorders Inventory (EDI) (Garner, Olmsted & Polivy, 1983); EDI-2 (Garner, 1991) | Drive for Thinness Bulimia Body Dissatisfaction Ineffectiveness Perfectionism Interpersonal Distrust Interoceptive Awareness Maturity Fears Asceticism (EDI-2) Impulse Regulation (EDI-2) Social Insecurity (EDI-2) | ≥ 12 years | Scored similarly to EAT and EAT-26; a cutoff of 14 for Drive for Thinness recommended for screening purposes |
| Eating Disorders Examination Questionnaire (EDE-Q) (Fairburn & Beglin, 1994) | Global eating disorder severity Restraint Eating Concern Shape Concern Weight Concern | Older adolescents and adults | 36 items with responses ranging from 0 "never" to 6 "every day" over previous 28 days; item scores averaged, and scores between 4 and 6 are considered to be clinically significant |
| Mizes Anorectic Cognitions Scale (MACS-R) (Mizes, Christiano, Madison et al., 2000) | Self-control self-esteem Weight and approval Rigid weight regulation/ fear of weight gain | Adults | Items rated from "strongly disagree" to "strongly agree" on 5-point Likert scale |
| Shape- and Weight- Based Self-Esteem Inventory (Geller, Johnson, Madson, 1997) | Influence of weight and shape on self-evaluation | ≥ 13 years | Patient completes pie chart indicating size of self-esteem pie influenced by different domains, including weight shape; score is determined by angle of pie shape that reflects influence of shape and weight on self-esteem |
| Beck Depression Inventory–2 (BDI- 2) (Beck, Steer & Brown, 1996) | Depression severity | ≥ 13 years | 21 items endorsed for degree of depressive symptoms in previous 2 weeks; total score determined by sum of highest response to each item; cutoff of 29 and higher indicates severe depression; 20–28 moderate depression; 14–19 moderate depression |

TABLE 1.1. Self-Report Measures Used in the Psychological Assessment of AN

(cont.)

| Measure (authors) | Construct assessed | Age | Scoring and interpretation |
|---|--------------------|---------------------------------|---|
| Beck Anxiety Inventory (BAI) (Beck, Epstein, Brown & Steer, 1988) | Anxiety severity | Older adolescents and adults | 21 items endorsed for degree of anxiety symptoms in previous week; total score determined by sum of highest response to each item |
| Rosenberg Self- Esteem Scale (RSE) (Rosenberg, 1965) | Self-esteem | Older adolescents and adults | 10 items endorsed for feelings of self-worth; higher scores indicate higher self-esteem |
| | | | 25 |

TABLE 1.1. (cont.)

Family Assessment

In the preceding sections we have emphasized assessing the patient, assuming in some cases that the patient is an adult. Indeed, many of the psychological assessments included in Table 1.1 are validated specifically in adult populations. For younger patients still living with parents or guardians, family assessment can provide information about the family environment and about the patient.

First, family assessment gives information about the patient's immediate living environment, which may not be available through traditional, patient-focused assessments. For example, the Family Assessment Measure–III (FAM-III; Skinner, Steinhauer, & Santa-Barbara, 1995) is a self-report questionnaire that is completed by each family member, including children and siblings 10 years of age or older. The FAM-III produces three subscale scores: the General Scale taps the family system; the Dyadic Relationships Scale assesses relationship quality between pairs within a family (e.g., Mother– Daughter Dyad); and the Self-Rating Scale measures how the individual completing the scale perceives his/her function within a family (Carter et al., 2004).

Second, family assessment can provide additional information about the patient. Indeed, regardless of a patient's age, any assessment can be significantly enhanced by talking with a family member, spouse, or friend of the patient. This family inclusion is particularly important for younger patients because minors are more likely to have been brought into treatment by their family rather than being self-referred. Thus, younger patients may not believe that they have a problem that requires intervention and may be less forthcoming with their thoughts, feelings, and behaviors. Conducting assessments with the family can reveal behavioral patterns that provide crucial information for understanding the patient's motivations and fears.

As noted above, a specific family-based intervention (Russell et al., 1987) has demonstrated efficacy in treating AN in younger patients. This intervention requires a careful assessment of family processes to identify how the child's illness has impacted family functioning and ways in which these alterations serve to maintain the illness. Initially, assessment focuses on factors that would directly impact the patient's ability to eat and gain weight. One part of this assessment occurs during a family meal in which the therapist observes what foods are prepared, how they are presented, and interactions among family members around eating (or the lack thereof). Of note, this is not a purely observational assessment. The therapist uses this event to coach parents *in vivo* in ways to improve their child's food intake. Later sessions of family-based therapy focus on how the eating disorder impacts not only the patient's medical and psychological well-being but also family interactions and developmental processes that should occur during adolescence. Again, this assessment occurs within the context of intervention to improve patient outcome.

Treatment Planning

Treatment Setting Options

Services available for treating EDs range from intensive inpatient programs (in which general medical care is readily available), to residential and partial hospitalization programs, to varying levels of outpatient care (in which the patient receives general medical treatment, nutritional counseling, and/or individual, group, and family psychotherapy). Specialized ED programs are unavailable in many geographic areas, and the financial burden associated with specialized multidisciplinary treatment is significant. Thus, many patients and health professionals face the dilemma of identifying the best treatment available, given treatment needs and resources available.

Recent work by Gowers and colleagues (2007) found no outcome differences when comparing adolescent patients with AN who had been initially randomized to inpatient treatment, specialized outpatient treatment, or general outpatient treatment. Instead, greater differences were found for those who transitioned from their assigned treatment condition. Specifically, those randomized to inpatient treatment who gained weight and "bargained their way out" of inpatient treatment had better outcomes compared to those who remained in that treatment arm. Conversely, those randomized to either outpatient arm but subsequently transferred to inpatient treatment had worse outcomes compared to those who remained in outpatient treatment. Generally, Gowers and colleagues' findings paint a bleak picture for the effectiveness of inpatient treatment for adolescent patients with AN. However, their findings should be interpreted in light of the impact of patient factors (specifically, severity of illness) on treatment utilization (Keel et al., 2002).

Inpatient Treatment

Factors suggesting that hospitalization may be appropriate include rapid or persistent decline in oral intake, a decline in weight despite maximally intensive outpatient or partial hospitalization interventions, the presence of additional stressors that may interfere with the patient's ability to eat, reaching a weight at which instability previously occurred in the patient, co-occurring psychiatric problems that merit hospitalization (e.g., suicidality), and the degree of the patient's denial and resistance to participate in his/her own care in less intensively supervised settings. Legal interventions, including involuntary hospitalization and legal guardianship, may be necessary to address the safety of treatment-reluctant adult patients who have developed, or are at imminent risk for developing, life-threatening medical sequelae from starvation. Many adult patients with AN have considerable difficulty gaining weight outside of a highly structured program, such as that provided by an inpatient program. Within an inpatient program, patients are initially prescribed 1,200–1,500 calories per day, depending on their weight at admission, and then intake is increased by 500 calories every 4–5 days until patients are consuming between 3,500–4,500 calories per day (Bowers, Andersen, & Evans, 2004). This schedule is designed to achieve weight gain of 3 pounds per week in females and 4 pounds per week in males without risk of medical complications of refeeding.

Partial Hospitalization

Although studies suggest that weight restoration is more efficient during inpatient hospitalization, growing pressure from insurance companies to transition patients to an outpatient setting once they are medically stabilized has placed more emphasis on partial hospitalization to provide intensive treatment over extended periods (Howard, Evans, Quintero-Howard, Bowers, & Andersen, 1999; Treat, McCabe, Gaskill, & Marcus, 2008). This pressure has caused a shift in focus from long-term inpatient programs with outpatient follow-up to day treatment care with hospital backup. A partial hospitalization program is appropriate for individuals discharged from inpatient care who have achieved at least 85% of their expected body weight but who still require intensive treatment to regain 95-100% of their body weight (Bowers et al., 2004). Partial hospitalization is also useful for addressing the cognitive features of AN that persist after weight restoration and increase risk of relapse (Keel, Dorer, Franko, Jackson, & Herzog, 2005). A partial hospital program offers a structured environment for most of the day-typically from 8:00 A.M. to 6:00 P.M.-and thus offers patients the opportunity to consume two to three meals and snacks to achieve continued weight gain. For example, within a partial hospital program, patients may consume 3,500 calories per day in order to gain approximately 2 pounds per week (Bowers et al., 2004). However, steady gains of even 0.5 pounds per week have been associated with better outcome at 6-month followup (Treat et al., 2008). Partial hospital treatment may be appropriate for patients who have not responded to outpatient treatment but who remain medically stable.

Outpatient Treatment

Although weight restoration may be faster in inpatient settings (Howard et al., 1999; Treat et al., 2008), findings from Gowers and colleagues (2007) suggest that inpatient and outpatient treatment achieve similar levels of weight gain and recovery. In addition, outpatient treatment provides more socialization, interactions in a real-world setting, and greater opportunities for patients to improve feelings of self-efficacy. However, in order to achieve weight restoration, it is important for outpatient treatment to provide repeated assessments of weight. Specifically, monitoring of weight should be done at least weekly (and often two to three times a week). Weight should be objectively measured in the same setting (same scale) after the patient voids and while the patient is wearing the same class of garment (e.g., hospital gown or standard clothing with pockets emptied and without shoes or heavy outer wear). In patients who purge, it is important to routinely monitor serum electrolytes. Urine-specific gravity, orthostatic vital signs, and oral temperatures may need to be measured on a regular basis as well.

Treatment Setting Transitions

Relapse occurs in 30% of patients with AN (Keel, Dorer, et al., 2005), and, as a result, patients may transition between inpatient, partial hospitalization, and outpatient treat-

ment and back again throughout the course of their recovery. Shorter duration of illness and higher BMI at the time of transition from inpatient to partial hospitalization predict better outcome (Howard et al., 1999; Treat et al., 2008), reinforcing the importance of patient variables in predicting treatment response. If the patient is going from one treatment setting to another, transition planning requires that the care team in the new setting be identified and that specific patient appointments be made. Similar guidelines apply to changes in locale that occur when patients require more intensive specialized treatment than is available to them locally. Changes in locale are particularly likely to occur in college-age patients who may initiate treatment where they attend school, return home for more intensive care, and then return to school when they are judged to be medically stable and able to benefit from a less intensive treatment approach. It is preferable that a specific clinician on the team be designated as the primary coordinator of care to ensure continuity and attention to important aspects of treatment.

Treatment Choices

Medications

At this time there is no empirical support for attempting to treat AN with medication alone. The decision about whether to use psychotropic medications and, if so, which medications to choose is based on the patient's clinical presentation. The limited empirical data on malnourished patients indicate that selective serotonin reuptake inhibitors (SSRIs) do not appear to confer advantage over placebo regarding maintenance of healthy weight (Walsh et al., 2006) or any advantage over psychotherapy for preventing relapse (Treasure & Schmidt, 2005). However, antidepressant medications are often used to treat comorbid depressive, anxiety, or obsessive–compulsive symptoms and for bulimic symptoms in weight-restored patients. Psychiatrists should evaluate the possible role of starvation in producing symptoms of depression and anxiety prior to initiating treatment with an antidepressant. Adverse reactions to tricyclic antidepressants and monoamine oxidase inhibitors are more pronounced in malnourished individuals, and these medications should generally be avoided in this patient population.

Recent randomized controlled trials of olanzapine (an antipsychotic) in AN have shown that weight restoration is faster when this medication is utilized (Attia, Kaplan, Haynos, Yilmaz, & Musante, 2008; Bissada, Tasca, Barber, & Bradwejn, 2008). There are mixed results for cyproheptadine's (an antihistamine) improvement of appetite, and other strategies that have not been investigated yet include the use of megastrol acetate (a steroid) or mirtazapine (an antidepressant), which have appetite-stimulating effects. Antianxiety agents used selectively before meals may be useful to reduce patients' anticipatory anxiety before eating. However, within the anxiety disorders literature, anxiolytics have been shown to reduce the efficacy of interventions that employ exposure and response prevention. Thus, the short-term benefits of using antianxiety medications may be offset by their potential to undermine long-term benefits of psychotherapeutic interventions. Promotility agents such as metoclopramide may be useful for bloating and abdominal pains that occur during refeeding in some patients. However, the risk of side effects such as sedation and motor abnormalities should be considered and discussed with patients before initiating medication use.

Although no specific hormone treatments or vitamin supplements have been shown to be helpful, supplemental calcium, vitamins, and hormones are often used. Calcium has been used in the hope that it will reduce problems with osteoporosis, but data have not substantiated this effect. Vitamin supplements have been used to balance nutritional deficiencies associated with prior food restriction. Finally, hormonal contraceptives have been used to trigger menstrual bleeding. However, given the lack of evidence that any of these improve patient outcomes, they are not a recommended part of treatment. Instead, emphasis should be placed on increasing food intake and achieving weight restoration. 10-

Psychotherapy

During the acute phase of treatment, the efficacy of specific psychotherapeutic interventions for facilitating weight gain remains uncertain. Table 1.2 includes randomized control treatment trials conducted in younger-adolescent as well as late-adolescent and adult samples with AN. Several observations can be made of the studies listed in this table. First, for younger patients, a specific family-based therapy has demonstrated superiority to alternative treatments in independent studies (Robin et al., 1999; Russell et al., 1987), making it a well-established evidence-based treatment for adolescents with AN (Keel & Haedt, 2008). However, this does not generalize to older patients with AN. Second, based on findings from two studies in Table 1.2 (Pike, Walsh, Vitousek, Wilson, & Bauer, 2003; Serfaty, Turkington, Heap, Ledsham, & Jolley, 1999), nutritional counseling alone is not beneficial for patients with AN compared to alternative interventions.

Third, few randomized controlled studies of psychosocial interventions have been conducted on AN in late-adolescent/adult samples, despite the fact that AN has been recognized as a form of mental illness for more than a century (Gull, 1874). Fourth, with few exceptions (Crisp et al., 1991), studies have compared two or more alternative treatments rather than using wait-list control or assessment-only conditions, due to ethnical constraints of withholding treatment from severely ill patients. As a consequence, studies have been unable to establish possible efficacy of a treatment in comparison to a no-treatment control (Chambless & Ollendick, 2001). Instead, treatments have been held to the highest standard of having to demonstrate superiority to alternative interventions. Fifth, sample sizes assigned to each treatment condition have been small and then further reduced by noncompletion, such that an average of 13 patients completed each treatment condition included in Table 1.2. These patterns contribute to a final, almost inevitable observation: The majority of comparisons from controlled treatment studies fail to identify any psychotherapy as being superior to another for the treatment of adult patients with AN.

In practice, modes of therapy (e.g., individual psychotherapies, family therapies, nutritional counseling, and group therapies) are often combined during hospital treatment and in comprehensive follow-up care. In addition, clinicians frequently attempt to blend features of different intervention models (cognitive-behavioral techniques, 12-step abstinence approaches, interpersonal, dialectical, and psychodynamic frameworks) resulting in an eclectic treatment approach (e.g., Johnson & Taylor, 1996). Although clinicians often view combined approaches as superior to a single-therapy approach and would endorse their effectiveness, no systematic data have been published regarding outcomes of using these combined integrated approaches to allow

| TABLE 1.2. Randomize (Mean Age > 17 Years) | ed Cor | trolled Tr | eatment S | tudies of AN in Young Adolescent So | amples (Mean Age < 17 Years) and Lat | e Adolescent/Adult Patients |
|---|----------|-------------|-----------------------------|---|--|---|
| Study | N | Sex (%F) | Age (<i>M</i> or range) | Inclusion criteria | Therapy conditions: N (% complete) | Results summary |
| Young adolescent (M age | e < 17 J | ears) | | | | |
| Eisler, Dare, Hodes, et al. (2000) | 40 | 97.5 | 15.5 | DSM-IV; ICD-10 AN | Conjoint family therapy (CFT): 19 (89.5%) Separated family therapy (SFT): 21 (90.5%) | <i>CFT</i> > <i>SFT</i> on EDI <i>CFT</i> = <i>SFT</i> on BMI, bulimic symptoms EAT, M-R scales, and outcome <i>CFT</i> > <i>SFT</i> on Depression, |
| | | | | 2 | | Obsessionality SMFQ <i>CFT</i> = <i>SFT</i> MOCI, RSE, Tension |
| Geist, Heinmaa, Stephens, et al. (2000) | 25 | 100 | 14.6 | Female Ages 12–17.4 yr Weight < 90% IBW Self-imposed food restriction | Family therapy (FT): 12 (100%) Family group psychoeducation (FPE): 13 (100%) | FT = FPE |
| Le Grange, Eisler, Dare, & Russell (1992) | 18 | 88.9 | 15.3 | DSM-III-R AN Age < 18 yr Duration of illness shorter than 3 yr | CFT: 9 (100%) SFT: 9 (100%) | CFT = SFT |
| Lock, Agras, Bryson, & Kraemer (2005) | 86 | 89.5 | 15.2 | DSM-IV AN Some partially weight restored Loss of one menstrual cycle | Short-term FT (STFT): 44 (96%) Long-term FT (LTFT): 42 (84%) | STFT = LTFT |
| Robin et al. (1999) | 41 | 100 | 14.2 | DSM-III-R AN Females Ages 11–20 yr Residing at home with one or both parents | Behavioral family systems therapy (BFST): 19 Ego-oriented individual therapy (EOIT): 18 90.2% completion across conditions | <i>BFST</i> > <i>EOIT</i> on BMI <i>BFST</i> = <i>EOIT</i> on EAT, EDI <i>BFST</i> = <i>EOIT</i> on BDI, CBCL |
| Russell, Szmukler, Dare, & Eisler (1987)ª | 21 | 91.3 | 16.6 | DSM-III AN Extreme loss of weight, no purging following overeating Amenorrhea or loss of sex interest/potency | FT: 10 (100%) Nonspecific individual treatment (NST): 11 (100%) | FT > NST on % ABW MR scales MR outcome (cont.) |

| TABLE 1.2. (cont.) | \mathcal{G} | - 093 | idh | G | | |
|---|---------------|-------------|--------------------|--|---|---|
| Study | Ν | Sex (%F) | Age $(M$ or range) | Inclusion criteria | Therapy conditions: N (% complete) | Results summary |
| Late adolescent/adult (A | 1 age > | - 17 year | $\overline{s})$ | | | |
| Bachar, Latzer, Kreitler, & Berry (1999) | 13 | 100 | 18.1 | DSM-IV AN | Self-psychological treatment (SPT): 7 (85.7%) Cognitive orientation treatment (COT): 6 (33.3%) | N too small for inferential statistics |
| Channon, de Silva, Hemsley, & Perkins (1989) | 24 | 100 | 23.8 | Russell's AN (17) Female | Cognitive-behavioral therapy (CBT): 8 (100%) Behavioral therapy (BT): 8 (87.5%) Routine outpatient: 8 (75%) | <i>CBT</i> = <i>BT</i> on All measures <i>Routine</i> > <i>CBT</i> , <i>BT</i> on Maturity fears |
| Crisp et al. (1991) | 06 | 100 | 22 | DSM-III-R AN Female Duration of illness < 10 yr Living near the service | <pre>Inpatient (IP): 30 (60%) Outpatient individual/family (OI): 20 (90%) Outpatient group (OG): 20 (85%) No further treatment (NT): 20 (100%)</pre> | <i>IP</i> , <i>OI</i> , <i>OG</i> > <i>NT</i> on Weight <i>IP</i> = <i>OI</i> = <i>OG</i> = <i>NT</i> on MR scales |
| Dare, Eisler, Russell, Treasure, & Dodge (2001) | 84 | 98 | 26.3 | DSM-IV AN Age > 18 yr | FT: 22 (72.7%) Focal psychoanalytic therapy (FP): 21 (57.1%) Cognitive-analytic therapy (CAT): 22 (59.1%) Routine treatment (RT): 19 (68.4%) | FT, $FP > RT$ on BMI FT = FP = CAT = RT MR Scales |

| NST > IPT on EDE restraint, Global outcome, GAF <i>CBT</i> > <i>IPT</i> on EDE restraint <i>CBT</i> = <i>IPT</i> on Global outcome, GAF <i>CAT</i> = <i>IPT</i> = <i>NST</i> on BMI, EDE (exc. restraint) EDI, HAM-D | CBT > NC | NST > FT for Weight gain NST = FT for MR outcome | CBT > DC on % remitted CBT = DC on BMI | CAT > EBT on Subjective rating CAT = EBT on BMI, MR Scales | |
|---|--|---|--|--|---|
| CBT: 19 (63%) Interpersonal psychotherapy (IPT): 21 (57%) NST: 16 (69%) | CBT: 18 (100%) Nutritional counseling (NC): 15 (80%) | FT: 19 (84.2%) NST: 17 (94.1%) | CBT: 25 (92%) Dietician control (DC): 10 (0%) | CAT: 14 (71.4%) Educational behavior therapy (EBT): 16 (62.5%) | |
| Female Ages 17–40 yr DSM-IVAN or lenient AN (BMI 17.5–19)—without amenorrhea criterion | DSM-IV AN–posthospitalization Lived within commuting distance | DSM-HI AN Extreme weight loss, amenorrhea/ loss of sex interest, posthospitalization | Ages > 16 yr DSM-III-R AN | Ages > 18 yr ICD-10 AN | |
| 17-40 | 25.2 | 24.2 | 20.9 | 25.0 | - |
| ≞ 09 ³ | 100 | 91 | 94 | 97 | |
| 56 | 33 | 36 | 35 | 30 | - |
| McIntosh et al. (2005) | Pike, Walsh, Vitousek, Wilson, & Bauer (2003) | Russell et al. (1987) ^a | Serfaty, Turkington, Heap, Lesham, & Jolley (1999) | Treasure et al. (1995) | |

Note: ABW, average body weight; BDI, Beck Depression Inventory; BMI, body mass index; CBCL, Child Behavior Checklist; EDE, Eating Disorder Examination; EDI, Eating Disorder Inventory; GAF, Global Assessment of Functioning; HAM-D, Hamilton Depression Rating Scale; IBW, ideal body weight; ICD, International Classification of Diseases; MOCI, Maudsley Obsessional Compulsive Index; MR Outcome, Morgan and Russell outcome; MR Scales, Morgan and Russell scales; RSE, Rosenberg Self-Esteem Scale; SMFQ, Short Mood and Feelings Questionnaire.

rdpress ^aRussell et al. (1987) included a group of young-adolescent patients with AN and adult patients with AN and is included in the table twice.

evaluation of their efficacy (i.e., their superiority in a randomized control trial) or their effectiveness (i.e., their success when employed in clinical settings).

Involvement of Family and Caregivers

A specific form of family therapy has demonstrated success in reducing symptoms in adolescent patients (Russell et al., 1987). Adolescent patients have been shown to improve most with conjoint family therapy (see Table 1.2), except in cases of highly critical parents, for which separate family groups may be more effective (Eisler et al., 2000). Patients with higher levels of obsessive–compulsive symptoms or those who come from a nonintact family have demonstrated improved outcomes when assigned to a 12-month, compared to a 6-month, family-based treatment (Lock, Agras, Bryson, & Kraemer, 2005).

Despite evidence supporting the importance of involving family and caregivers in the treatment of adolescents with AN, limited data support this approach in adult patients. In the original trial supporting the use of family-based treatment for younger patients with a shorter duration of illness (Russell et al., 1987), individual psychotherapy was associated with superior outcomes compared to family-based therapy in adult patients with AN (Russell et al., 1987). Of note, involving family and caregivers in the treatment of adult patients is complicated by ethical considerations to protect the confidentiality of information shared during treatment. However, given the potential for treatment resistance, family and caregivers often look to clinicians to provide them with tools they may use to support patients' efforts to make progress in treatment. Family and caregivers also may benefit from affiliation with support groups designed to acknowledge the stress of caring for an ill child, regardless of the age of that child. Goddard, MacDonald, and Treasure (Chapter 29, this volume) examine recent work addressing the needs of AN caregivers (see also Sepulveda, Lopez, Todd, Whitaker, & Treasure, 2008; Whitney et al., 2006).

Treatment Team Members

Among various forms of mental disorders, AN presents one of the greatest challenges because the illness impacts multiple domains of function. As such, a multidisciplinary team is required to address the various aspects of the illness. This treatment team should consist of at least a medical professional, a mental health professional, and a dietician.

The role of the medical professional is to monitor physical health and safety and ensure involvement of medical specialists as indicated. AN can trigger medical sequelae that require attention from specialists in endocrinology, cardiology, nephrology, gastroenterology, and orthopedics. The role of the mental health professional is to address disturbances in thoughts, feelings, and behaviors that form the core of the illness. A range of professions may fulfill this role, including psychiatrists, licensed clinical psychologists, clinical social workers, marriage and family therapists, and psychiatric nurses. While individuals in each of these professions are trained to provide psychotherapeutic interventions, a psychiatrist also may manage psychopharmacological treatments. The role of the dietician is to assess nutritional needs for healthy weight gain and maintenance, create an appropriate dietary plan, and assist in implementation of the plan.

Aside from these three team members, additional professions may be involved in the treatment of inpatients—who are often more severely impacted by illness than those who are seen on an outpatient basis. Occupational therapists are involved with inpatient and partial hospitalization programs to assist patients in learning how to prepare healthy meals, go grocery shopping, attend restaurant outings with peers, and engage in family meals. Recreational therapists have been utilized in inpatient and partial hospital settings to help patients learn to incorporate healthy exercise, as well as creative relaxation strategies to improve more balanced living. The components of the treatment team should be guided by the patient's needs. However, it is important for health professionals to fully appreciate the limits of their own professional training with respect to treating a patient with AN.

References

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.
- Attia E, Kaplan AS, Haynos A, Yilmaz Z, & Musante D. (2008, September). *Olanzapine vs. placebo for outpatients with anorexia nervosa: A pilot study.* Paper presented at the 14th Annual Eating Disorders Research Society Meeting, Montreal, QB.
- Bachar E, Latzer Y, Kreitler S, & Berry EM. (1999). Empirical comparison of two psychological therapies: Self psychology and cognitive orientation in the treatment of anorexia and bulimia. *Journal of Psychotherapy Practice and Research*, 8:115–128.
- Beck AT, Epstein N, Brown G, & Steer RA. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology*, 56:893–897.
- Beck AT, Steer RA, & Brown GK. (1996). *The Beck Depression Inventory Manual* (2nd ed.). San Antonio, TX: Psychological Corporation Harcourt & Brace.
- Becker AE, Keel PK, Anderson-Fye EP, & Thomas JJ. (2004). Genes (and/or) jeans?: Genetic and socio-cultural contributions to risk for eating disorders. *Journal of Addictive Diseases*, 23:81–103.
- Bissada H, Tasca GA, Barber AM, & Bradwejn J. (2008). Olanzapine in the treatment of low body weight and obsessive thinking in women with anorexia nervosa: A randomized, double-blind, placebo controlled trial. *American Journal of Psychiatry*, 165:1281–1288.
- Bowers WA, Andersen AE, & Evans K. (2004). Management for eating disorders: Inpatient and partial hospital programs. In TD Brewerton (Ed.), *Clinical handbook of eating disorders: An integrated approach* (pp. 349–376). New York: Marcel Dekker.
- Bulik CM, Berkman ND, Brownley KA, Sedway JA, & Lohr KN. (2007). Anorexia nervosa treatment: A systematic review of randomized controlled trials. *International Journal of Eating Disorders*, 40:310–320.
- Carter JC, McFarlane TL, & Olmsted MP. (2004). Psychometric assessment of eating disorders. In TD Brewerton (Ed.), *Clinical handbook of eating disorders: An integrated approach* (pp. 21–46). New York: Marcel Dekker.
- Chambless DL, & Ollendick TH. (2001). Empirically supported psychological interventions: Controversies and evidence. *Annual Review of Psychology*, 52:685–716.
- Channon S, de Silva P, Hemsley D, & Perkins R. (1989). A controlled trial of cognitive-behavioural and behavioural treatment of anorexia nervosa. *Behaviour Research and Therapy*, 27:529–535.

- Crisp AH, Norton K, Gowers S, Halek C, Bowyer C, Yeldham D, et al. (1991). A controlled study of the effect of therapies aimed at adolescent and family psychopathology in anorexia nervosa. *British Journal of Psychiatry*, 159:325–333.
- Culbert KM, Breedlove SM, Burt SA, & Klump KL. (2008). Prenatal hormone exposure and risk for eating disorders: A comparison of opposite-sex and same-sex twins. *Archives of General Psychiatry*, 65:329–336.
- Dalle Grave R, & Calugi S. (2007). Eating disorder not otherwise specified in an inpatient unit: The impact of altering the DSM-IV criteria for anorexia and bulimia nervosa. *European Eating Disorders Review*, 15:340–349.
- Dare C, Eisler I, Russell G, Treasure J, & Dodge L. (2001). Psychological therapies for adults with anorexia nervosa: Randomized controlled trial of out-patient treatments. *British Journal of Psychiatry*, 178:216–221.
- Eddy KT, Keel PK, Dorer DJ, Delinsky SS, Franko DL, & Herzog DB. (2002). A longitudinal comparison of anorexia nervosa subtypes. *International Journal of Eating Disorders*, 31:191–201.
- Eisler I, Dare C, Hodes M, Russell G, Dodge E, & Le Grange D. (2000). Family therapy for adolescent anorexia nervosa: The results of a controlled comparison of two family interventions. *Journal of Child Psychology and Psychiatry*, 41:727–736.
- Fairburn CG, & Beglin SJ. (1994). Assessment of eating disorders: Interview or self-report questionnaire. International Journal of Eating Disorders, 16:363–370.
- Fairburn CG, & Cooper Z. (1993). The Eating Disorder Examination (12th ed.). In C Fairburn & GT Wilson (Eds.), *Binge eating: Nature, assessment, and treatment* (pp. 317–331). New York: Guilford Press.
- Fairburn CG, Cooper Z, Doll HA, & Welch SL. (1999). Risk factors for anorexia nervosa: Three integrated case-control comparisons. *Archives of General Psychiatry*, 56:468–476.
- Favaro A, Monteleone P, Santonastaso P, & Maj M. (2008). Psychobiology of eating disorders. Annual Review of Eating Disorders (Part 2). Oxford, UK: Radcliffe.
- Favaro A, Tenconi E, & Santonastaso P. (2006). Perinatal factors and the risk of developing anorexia nervosa and bulimia nervosa. *Archives of General Psychiatry*, 63:82–88.
- First MB, Spitzer RL, Gibbon M, & Williams JB. (1996). Structured Clinical Interview for Axis I DSM-IV Disorders. New York: Biometrics Research Department, New York State Psychiatric Institute.
- Garner DM. (1991). Eating Disorder Inventory-2: Professional manual. Odessa, FL: Psychological Assessment Resources.
- Garner DM, & Garfinkel PE. (1979). The Eating Attitudes Test: An index of the symptoms of anorexia nervosa. *Psychological Medicine*, 9:273–279.
- Garner DM, Olmsted MP, Bohr Y, & Garfinkel PE. (1982). The Eating Attitudes Test: Psychometric features and clinical correlates. *Psychological Medicine*, 12:871–878.
- Garner DM, Olmsted MP, & Polivy J. (1983). Development and validation of a multidimensional Eating Disorder Inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders*, 2:15–34.
- Geist R, Heinmaa M, Stephens D, Davis R, & Katzman DK. (2000). Comparison of family therapy and family group psychoeducation in adolescents with anorexia nervosa. *Canadian Journal of Psychiatry*, 45:173–178.
- Geller J, Drab-Hudson DL, Whisenhunt BL, & Srikameswaran S. (2004). Readiness to change dietary restriction predicts outcomes in eating disorders. *Eating Disorders*, 12:209–224.
- Geller J, Srikameswaran S, Cockell S, & Zaitsoff Z. (2000). Assessment of shape- and weightbased self-esteem in adolescents. *International Journal of Eating Disorders*, 28:339–345.
- Gowers SG, Clark A, Roberts C, Griffiths A, Edwards V, Bryan C, et al. (2007). Clinical effectiveness of treatments for anorexia nervosa in adolescents: Randomised controlled trial. *British Journal of Psychiatry*, 191:427–435.

- Gull WW. (1874). Anorexia nervosa (apepsia hysterica, anorexia hysterica). *Transactions of the Clinical Society of London*, 7:22–28.
- Herzog DB, Dorer DJ, Keel PK, Selwyn SE, Ekeblad ER, Flores AT, et al. (1999). Recovery and relapse in anorexia nervosa and bulimia nervosa: A 7.5 year follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38:829–837.
- Hoek HW, van Harten PN, Hermans KM, Katzman MA, Matroos GE, & Susser ES. (2005). The incidence of anorexia nervosa in Curacao. *American Journal of Psychiatry*, 162:748–752.
- Howard WT, Evans KK, Quintero-Howard C, Bowers WA, & Andersen AE. (1999). Predictors of success or failure of transition to day hospital treatment for inpatients with anorexia nervosa. *American Journal of Psychiatry*, 156:1697–1702.
- Hudson JI, Hiripi E, Pope HG, & Kessler RC. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry*, 61:348–358.
- Jacobi C, Hayward C, de Zwaan M, Kraemer HC, & Agras WS. (2004). Coming to terms with risk factors for eating disorders: Application of risk terminology and suggestions for general taxonomy. *Psychological Bulletin*, 130:19–65.
- Johnson CL, & Taylor C. (1996). Working with difficult-to-treat eating disorders using an integration of twelve-step and traditional psychotherapies. *Psychiatric Clinics of North America*, 19:829–841.
- Kaye SH, Frank GK, Bailer UF, & Henry SE. (2005). Neurobiology of anorexia nervosa: Clinical implications of alterations of the function of serotonin and other neuronal systems. *International Journal of Eating Disorders*, 37:S15–S19.
- Keel PK. (2005). Eating disorders. Upper Saddle River, NJ: Pearson Prentice-Hall.
- Keel PK, Dorer DJ, Eddy KT, Delinsky SS, Franko DL, Blais MA, et al. (2002). Predictors of treatment utilization among women with anorexia and bulimia nervosa. *American Journal* of Psychiatry, 159:140–142.
- Keel PK, Dorer DJ, Eddy KT, Franko DL, Charatan D, & Herzog DB. (2003). Predictors of mortality in eating disorders. Archives of General Psychiatry, 60:179–183.
- Keel PK, Dorer DJ, Franko DL, Jackson SC, & Herzog DB. (2005). Post-remission predictors of relapse in eating disorders. *American Journal of Psychiatry*, 162:2263–2268.
- Keel PK, & Haedt A. (2008). Evidence-based psychosocial treatments for eating problems and eating disorders. *Journal of Clinical Child and Adolescent Psychology*, 37:39–61.
- Keel PK, & Klump KL. (2003). Are eating disorders culture-bound syndromes?: Implications for conceptualizing their etiology. *Psychological Bulletin*, 129:747–769.
- Keel PK, Klump KL, Miller KB, McGue M, & Iacono WG. (2005). Shared transmission of eating disorders and anxiety disorders. *International Journal of Eating Disorders*, 38:99–105.
- Keski-Rahkonen A, Hoek HW, Susser ES, Linna MS, Sihvola E, Raevuori A, et al. (2007). Epidemiology and course of anorexia nervosa in the community. *American Journal of Psychiatry*, 164:1259–1265.
- Keys A, Brozek J, Henschel A, Mickelson O, & Taylor HL. (1950). The biology of human starvation. Minneapolis: University of Minnesota Press.
- Klump KL, Burt SA, McGue M, & Iacono WG. (2007). Changes in genetic and environmental influences on disordered eating across adolescence: A longitudinal twin study. *Archives of General Psychiatry*, 64:1409–1415.
- Klump KL, & Culbert KM. (2007). Molecular genetic studies of eating disorders. Current Directions in Psychological Science, 16:37–41.
- Klump KL, Strober M, Bulik CM, Thornton L, Johnson C, Devlin B, et al. (2004). Personality characteristics of women before and after recovery from an eating disorder. *Psychological Medicine*, 34:1407–1418.
- Le Grange D, Eisler I, Dare C, & Russell GFM. (1992). Evaluation of family treatments in adolescent anorexia nervosa: A pilot study. *International Journal of Eating Disorders*, 12:347–357.

- Lilenfeld LR, Kaye WH, Greeno CG, Merikangas KR, Plotnicov K, Pollice C, et al. (1998). A controlled family study of anorexia nervosa and bulimia nervosa: Psychiatric disorders in first-degree relatives and effects of proband comorbidity. *Archives of General Psychiatry*, 55:603–610.
- Lock J, Agras WS, Bryson S, & Kraemer HC. (2005). A comparison of short- and long-term family therapy for adolescent anorexia nervosa. *Journal of the American Academy of Child and Adolescent Psychiatry*, 44:632–639.
- Lock J, & Le Grange D. (2005). Family-based treatment of eating disorders. *International Journal* of Eating Disorders, 37:26–30.
- Löwe B, Zipfel S, Buchholz C, Dupont Y, Reas DL, & Herzog W. (2001). Long-term outcome of anorexia nervosa in a prospective 21-year follow-up study. *Psychological Medicine*, 31:881– 890.
- McCormick LM, Keel PK, Brumm MC, Bowers W, Swayze V, Andersen A, et al. (2008). Implications of starvation-induced change in right dorsal anterior cingulate volume in anorexia nervosa. *International Journal of Eating Disorders*, 41:602–610.
- McIntosh VW, Jordan J, Carter FA, Luty SE, McKenzie JM, Bulik CM, et al. (2005). Three psychotherapies for anorexia nervosa: A randomized, controlled trial. *American Journal of Psychiatry*, 162:741–747.
- Mizes JS, Christiano B, Madison J, Post G, Seime R, & Varnado P. (2000). Development of the Mizes Anorectic Cognitions Questionnaire—Revised: Psychometric properties and factor structure in a large sample of eating disorder patients. *International Journal of Eating Disorders*, 28:415–421.
- Nielsen S, Moller-Madsen S, Isager T, Jorgensen J, Pagsberg K, & Theander S. (1998). Standardized mortality in eating disorders: A quantitative summary of previously published and new evidence. *Journal of Psychosomatic Research*, 44:413–434.
- Pike KM, Walsh BT, Vitousek K, Wilson GT, & Bauer J. (2003). Cognitive behavior therapy in the posthospitalization treatment of anorexia nervosa. *American Journal of Psychiatry*, 160:2046–2049.
- Robin AL, Siegel PT, Moye AW, Gilroy M, Dennis AB, & Sikand A. (1999). A controlled comparison of family versus individual therapy for adolescents with anorexia nervosa. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38:1482–1489.
- Rosenberg M. (1979). Conceiving of the self. New York: Basic Books.
- Russell GFM, Szmukler GI, Dare C, & Eisler I. (1987). An evaluation of family therapy in anorexia nervosa and bulimia nervosa. *Archives of General Psychiatry*, 44:1047–1056.
- Sepulveda AR, Lopez C, Todd G, Whitaker W, & Treasure J. (2008). An examination of the impact of "the Maudsley eating disorder collaborative care skills workshops" on the well being of carers: A pilot study. *Social Psychiatry and Psychiatric Epidemiology*, 43:584–591.
- Serfaty MA, Turkington D, Heap M, Ledsham L, & Jolley E. (1999). Cognitive therapy versus dietary counseling in the outpatient treatment of anorexia nervosa: Effects of the treatment phase. *European Eating Disorders Review*, 7:334–350.
- Skinner HA, Steinhauer PD, & Santa-Barbara J. (1995). *Family Assessment Measure–III*. Toronto: Multi-Health Systems.
- Steinhausen HC. (2002). The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry*, 159:1284–1293.
- Striegel-Moore RH, Dohm FA, Kraemer HC, Taylor CB, Daniels S, Crawford PB, et al. (2003). Eating disorders in white and black women. *American Journal of Psychiatry*, 160:1326–1331.
- Strober M, Freeman R, & Morrell W. (1999). Atypical anorexia nervosa: Separation from typical cases in course and outcome in a long-term prospective study. *International Journal of Eating Disorders*, 25:135–142.
- Sullivan PF. (1995). Mortality in anorexia nervosa. American Journal of Psychiatry, 152:1073– 1074.

- Thomas JJ, Roberto CA, & Brownell KD. (2009). Eighty-five per cent of what? Discrepancies in the weight cut-off for anorexia nervosa substantially affect the prevalence of underweight. *Psychological Medicine*, 39:833–843.
- Treasure J, & Schmidt U. (2005). Anorexia nervosa. Clinical Evidence, 14:1140-1148.
- Treasure J, Todd G, Brolly M, Tiller J, Nehmed A, & Denman F. (1995). A pilot study of a randomised trial of cognitive analytical therapy vs educational behavioural therapy for adult anorexia nervosa. *Behaviour Research and Therapy*, 33:363–367.
- Treat TA, McCabe EB, Gaskill JA, & Marcus MD. (2008). Treatment of anorexia nervosa in a specialty care continuum. *International Journal of Eating Disorders*, 41:564–572.
- Tyrka AR, Waldron I, Graber JA, & Brooks-Gunn J. (2002). Prospective predictors of the onset of anorexic and bulimic syndromes. *International Journal of Eating Disorders*, 32:282–290.
- Uher R, Brammer MJ, Murphy T, Campbell IC, Ng VW, Williams SCR, et al. (2003). Recovery and chronicity in anorexia nervosa: Brain activity associated with differential outcomes. *Biological Psychiatry*, 54:934–942.
- Walsh BT, Kaplan AS, Attia E, Olmsted M, Parides M, Carter JC, et al. (2006). Fluoxetine after weight restoration in anorexia nervosa: A randomized controlled trial. *Journal of the American Medical Association*, 295:2605–2612.
- Whitney J, Murray J, Gavan K, Todd G, Whitaker W, & Treasure J. (2004). Experience of caring for someone with anorexia nervosa: Qualitative study. *British Journal of Psychiatry*, 187:444– 449.
- Wilson GT, Grilo CM, & Vitousek KM. (2007). Psychological treatment of eating disorders. American Psychologist, 62:199–216.

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