# Practice Paper of the American Dietetic Association:

# Nutrition Intervention in the Treatment of Eating Disorders

# ABSTRACT

This paper provides concrete ideas about the role of the RD working with people diagnosed with eating disorders (EDs) as well as updates on current research and controversies within the eating disorders field, which serve to inform RDs about their role with this unique population. In addition it provides guidance on diagnostic criteria, symptoms, assessment and treatment. EDs are one of the hardest illnesses to treat, having both mental health aspects as well as medical and nutritional aspects. While multidisciplinary team treatment is considered best practice, there exists considerable variation in team composition depending on treatment setting and stage of illness. In addition, there is still debate over how to best treat the various EDs, where treatment needs to take place and who should be on a treatment team. Recent research advances have greatly improved understanding of EDs and consequently led to better treatment protocols. RDs need to pursue advanced training, mentoring, case consultation

Eating disorders (EDs), including anorexia nervosa (AN), bulimia nervosa (BN), and eating disorders not otherwise specified (EDNOS) are a group of mental illnesses that have challenged treatment providers, caregivers, and indeed, people diagnosed with an eating disorder. These disorders are defined by clinical criteria in the *Diagnostic and Statistical Manual* of Mental Disorders (DSM-IV) (1). Presently, the definitions of EDs are being redefined as the DSM task force works on creating the DSM-V criteria, which may include specific criteria for binge eating disorder (BED) and revised thresholds for diagnoses. A discussion of the American Psychiatric Association proposed changes to the DSM diagnostic criteria for eating disorders can be found in the American Dietetic Association (ADA) Position Paper "Nutrition Intervention in the Treatment of Eating Disorders" (2).

While these working definitions are used to diagnose eating disorders, effective treatment has been limited and remains elusive (3). Reasons for this include treatment providers working alone rather than in a multidisciplinary team, professionals lacking a common understanding of the illnesses, outdated assumptions about the nature of the illnesses, and lack of adequate insurance coverage for this spectrum of disorders. Currently there is no professional consensus on how to restore weight and health in AN, stop binge–purge behaviors in BN or halt the ED thoughts common to both

and supervision in order to be effective in treating this population. They also need to be aware of the controversies in EDs etiology and treatment and engage in the discussion worldwide about most effective treatment. RDs must be clear about their role on multidisciplinary teams and offer the best possible evidence based treatment to clients diagnosed with EDs. It is the position of the American Dietetic Association that nutrition intervention, including nutritional counseling, by a RD, is an essential component of the team treatment of patients with anorexia nervosa, bulimia nervosa, and other EDs during assessment and treatment across the continuum of care. This paper supports the "Position of the American Dietetic Association: Nutrition Intervention in the Treatment of Eating Disorders" published in the August 2011 Journal of the American Dietetic Association. To access the entire practice paper, go to: http://www.eatright.org/practice.

disorders. Recently, research has lead to promising new developments in understanding EDs and in treatment modalities (4,5).

It is the position of the ADA that nutrition intervention, including nutritional counseling, by a registered dietitian (RD), is an essential component of the team treatment of patients with AN, BN, and other EDs during assessment and treatment across the continuum of care. This practice paper supports the "Position of the American Dietetic Association: Nutrition Intervention in the Treatment of Eating Disorders" (2).

#### **THEORIES ON ETIOLOGY**

Theories about etiology of EDs have advanced from thinking the eating disorder was purely a matter of personal choice to thinking the ED was a result of family dysfunction to the current understanding that EDs are a result of a complex interaction between genetics, neurobiology, personality characteristics, and environment (5,6). Results of twin studies have shown that 50% to 83% of the variance in AN, BN, and BED, a form of EDNOS, can be accounted for by genetic factors. Chromosomes 1, 4, and 10 may contain risk genes for AN and BN, and chromosomal regions are being identified that may be involved in the serotonin, brain-derived neurotrophic factor and opioid systems, implicated in AN.

observations	Reduced spontaneity and flexibility concerning food intake Avoidance of specific foods Poor food variety Statements about being or eating "healthy" Avoidance of social situations with food Abnormal speed of eating a meal Attempt to "bargain" about foods (eg, I will eat this if I do not have to eat that) Inability to identify hunger or satiety Unusually small portions Inability to define or eat a balanced meal
Behavioral	Active and restless, stands frequently when most people would sit Disproportionate time spent thinking about food and body weight Interest in recipes, food channels, and food shopping Prepares food for other people without eating themselves Subjective or objective binge eating Hoards food or rations until the end of the day Food seems to go missing, especially sweets, cereals, high-carbohydrate foods Appears to be angry, tense, or hostile at meals Abnormal timing of meals and snacks "Debiting" food intake (eg, with exercise/food choices)
Rituals when eating	Excessive use of condiments (eg, salt, hot sauce) Cutting food into very small pieces before eating Inappropriate food utensils with preference for eating with fingers Picks, blots, and tears food apart Inappropriate food combinations and concoctions Eats food in a certain order Hides food in napkins, handbags, gives to dog, throws food away Doesn't let food touch lips
Physiological	<ul> <li>General: Marked weight changes or absence of expected weight gain in children or adolescents, growth delay in child/adolescent; weakness, fatigue, or lethargy.</li> <li>Cardio pulmonary: Low pulse, dizziness, low blood pressure, slow capillary refill</li> <li>Gastrointestinal: Abdominal pain, constipation, reflux, vomiting, delayed gastric emptying (feels full immediately after eating small amounts)</li> <li>Endocrine: loss of menstrual cycle, delayed menarche, or hypogonadism for boys/men</li> <li>Neuropsychiatric: Poor concentration, memory loss, insomnia, depression, anxiety, obcessiveness, over concern with weight and shape</li> <li>Integument: Dry skin, brittle nails, hair loss, yellow orange skin tone, white downy hair growth (lanugo), dull eyes, pale skin, cold intolerance</li> </ul>
Medical findings	<b>Anorexia Nervosa:</b> Bradycardia, orthostasis by pulse or blood pressure, hypothermia, cardiac murmur, atrophic breasts and vaginitis (postpubertal), pitting edema of extremities, emaciated, cold extremities, slowed capillary refill time <b>Bulimia Nervosa:</b> Sinus bradycardia, orthostatic by pulse or blood pressure, dry skin, parotid gland swelling, Russell's signs, mouth sores, dental enamel erosion, cardiac arrhythmias, may be normal weight

In both AN and BN there are alterations in brain serotonin, neuropeptide systems, and also brain neurocircuitry, which are present when the disorder is in its active state as well as in apparent recovery (7,8). Alteration in brain serotonin function seems to play a role in the odd appetite, mood, and impulse control observed in EDs. Consistent personality traits seen in AN and BN such as perfectionism, obsessive compulsiveness, and dysphoric mood sometimes seen in a child before the ED sets in, and persisting after weight restoration, suggest heritability (9). Taken together these data provide reasons for clinicians describing EDs as "brain disorders."

There is evidence that the hypothalamus plays a role in appetite regulation. Animal studies have shown an intense hunger drive when the lateral hypothalamus is stimulated by glutamate and glutamate agonists, suggesting a role for glutamate in hunger sensations (10). Individuals with BN or BED may have a higher expected sense of reward from food intake, leading to overeating, and the overeating may lead to habitual use of compensatory behaviors (11).

From the early starvation study by Ancel Keys came the understanding that starvation in and of itself produces many of the cognitive changes, mood disturbances, and peculiar behaviors characteristic of AN (12). Underweight adolescents with AN score higher on measures of depression and anxiety than when weight restored (13). The idea that families cause EDs has been dispelled (14). Recent research has shown that once the emotional impact of the ED is controlled there are no differences between families of girls diagnosed with AN and those diagnosed with insulin dependent diabetes mellitus, another childhood disease that can cause family distress. This suggests that families with a member diagnosed with an ED function differently as a result of the ED (15).

### **THEORIES ON TREATMENT**

Treatment theories have paralleled thinking about etiology. EDs require early diagnosis (16) then immediate and effective treatment. Current research shows that the sooner ED behaviors are stopped and nutrition and physical health restored the better the prognosis and the better a person responds to psychotherapy (16,17).

Multidisciplinary treatment teams have not always been standard in care of EDs. A multidisciplinary team, including nutrition, medical, and mental health professionals, provides the comprehensive treatment needed and each team member contributes best when grounded in advanced training in EDs. Methods for restoring physical and mental health can vary but need to be based in sound science. RDs involved in ED treatment have a responsibility to obtain advanced training, continuing education and case consultation, as well as read the most current research findings and apply evidence-based treatment modalities.

Current research has shown that family-based therapy (FBT) or the Maudsley method is very effective for anorexic and bulimic patients under age 18, with short duration of illness (less than 3 years), and that the family unit can provide effective treatment in the home setting (18). Similarly, research is ongoing with college-aged individuals and use of FBT; and another model is being researched with married couples, using concepts from FBT (19). These treatment models employ the use of social support concepts and the observation that individuals with an ED have difficulty refeeding or stopping binge-purge behaviors alone. Research also supports other modalities such as day treatment programs for those adolescents who do not improve in FBT (20). An overlooked component of care is that of the caregivers, especially parents, who experience tremendous stress in dealing with the protracted course of the illness and need support from the health care team as well as others in their lives (21). RDs can be an important source of caregiver support, by understanding the difficulty of their situation, educating them about EDs, the course of recovery and, for adolescents and preadolescents, instructing families on how to refeed at home.

An underappreciated aspect of treatment is the time involved in "return to normal" or a "maintenance period" when the person recovers not only physically but cognitively, and learns to eat normally, and to rely on internal regulation of hunger, appetite, satiety, and pleasure when eating. This period, which begins after an individual is physically and nutritionally restored, can be prolonged due to persistent psychological disturbances (22). RDs have the potential to be instrumental during this time by helping patients with weight maintenance, making adjustments to caloric intake as exercise is resumed or increased, and assisting the process of relearning normal eating patterns and engaging in social eating activities. Treatment for BED is evolving as diagnostic criterion has been developed (23,24). Binge eating, defined as eating a very large amount of food accompanied by a sense of loss of control has been observed in the adult population and recently observed in children ages 6 to 13 (25). BED seems to have a significant genetic component (26). Treatment for EDs has been very challenging due to lack of understanding about the nature of these disorders, and insurance company limitations on reimbursement for treatment (27). With recent research describing the etiology and nature of EDs, changes in treatment modalities have occurred (4). More research is needed in the area of effective treatment along the spectrum of severity of these illnesses.

## THE NATURE OF EATING DISORDERS

Eating disorders are now known to be biologically-based serious mental illnesses (9). People with EDs often lack insight into their illness (anosognosia), may be very resistant to treatment, and prone to deception, which can leave treatment providers feeling frustrated by the slow progress (6). The clinician who understands the nature of EDs will understand that individuals with EDs are not purposefully attempting to be uncooperative. Clinicians will know to not take personally the resistance to treatment and so be able to provide treatment that is associated with best prognosis. RDs who are ED specialists are psychologically literate, obtain supervision from clinical psychologists to help with issues of transference and countertransference, and understand the following:

- Eating disorders have an emotional/behavioral component and a neurophysiological/genetic component. The latter may set the stage for ED development, be triggered by weight loss, and may be the reason many ED psychological symptoms resolve with physical restoration (9).
- People do not choose to have EDs.
- Eating disorders are spectral disorders; they exist on a continuum of severity and often become more severe the longer they are present (28).
- Family dynamics might be part of the environmental influences that cause stress and stressful environments can exacerbate EDs, but families are not causative in ED etiology (29).
- EDs often co-exist with other psychiatric illnesses especially anxiety related disorders (9).
- The longer a person remains in a state of suboptimal nutrition or continues with ED behaviors, the more persistent and severe the disorder can become (30).
- Both physical restoration and cognitive/emotional restoration have to occur; physical restoration alone does not constitute recovery (4).
- Nutrition education alone is ineffective as is therapy/ counseling alone (31).

	Low abnormalities	High abnormalities
Sodium (Na <sup>+</sup> )	Reduced sodium levels in water loading or water intoxication. Low Na levels also seen in Addison's disease, excessive fluid loss due to vomiting, diarrhea, sweating. Proceeds during refeeding syndrome due to hyperglycemia.	Hypernatremia with dehydration and insufficient water intake. Note: for all electrolytes there are numerous other disease states that can affect levels. These are the common reasons seen in eating disorders.
Potassium (K+)	Hypokalemia is seen in malnutrition, malabsorption, vomiting, diarrhea, excess sweating (can be seen with excessive exercise), The most common cause of low K is gastrointestinal loss due to purging behaviors.	Hyperkalemia is seen in Addison's disease, which should be ruled out if eating disorders are suspected. Dehydration from water withholding and uncontrolled diabetes elevate K.
Chloride (Cl <sup>.</sup> )	Chloride is lost from the gastrointestinal tract with purging behaviors such as vomiting which results in lower blood levels. Blood chloride is also lower with water loading/water intoxication.	
Bicarbonate (CO <sub>2</sub> )	Low with laxative use.	Elevated bicarbonate with vomiting.
Phosphate (PO <sub>4</sub> <sup>2-</sup> )	Low phosphate occurs due to vomiting and diarrhea, malnutrition, malabsorption, respiratory alkalosis due to cellular use of phosphorus for increased glucose metabolism (this is basis of refeeding syndrome) or continued infusion of high-glucose solution in a non- diabetic patient.	Phosphate levels can be elevated due to a variety of conditions, most notably kidney dysfunction, fractures in the healing stage, Addison's disease (which can mimic bulimia and therefore needs to be ruled out), excess vitamin D.
Magnesium (Mg²+)	Low Mg levels are seen in states where excess loss of fluids occurs such as diarrhea, diuretic use or misuse, protracted vomiting.	Increased Mg levels are seen in dehydration. Note: serum Mg levels can remain normal even when body stores are depleted 20%.
Calcium (Ca <sup>2+</sup> )	Hypocalcemia can occur due to alkalosis, malnutrition, hyperphosphatemia due to laxative misuse. Increased Mg and PO4 intake and excess use of laxatives can lower blood Ca <sup>++</sup> level due to excess intestinal loss of calcium.	Hypercalcemia is seen in prolonged use of diuretics- thiazides, excessive vitamin D, milk-alkali syndrome, hyperparathyroidism, cancer, Addison's disease, hyperthyroidism, Paget's disease, bone fractures w/bed res
Thyroid function test	Referred to as sick euthyroid syndrome. Low thyroid- stimulating hormone, low thyroxine. Considered adaptive as a nonthyroidal disorders due to fasting, starvation, protein-energy under nutrition. Do not treat.	
Liver function test		Elevated in acute and chronic malnutrition
Cholesterol		Elevated levels are relatively common with malnutrition and do not need dietary manipulation. Cholesterol levels resolve with weight restoration but may require a year. Lipid panel may elevate with binge eating.
Amylase		Inflammation of salivary glands or pancreas will increase amylase in the blood.
Lipase		Elevated with pancreatitis
Hormone levels	Low luteinizing hormone, follicle-stimulating hormone, and estradiol with amenorrhea, low testosterone in males.	Elevated testosterone in females with polycystic ovarian syndrome.
Complement component 3	Decreased in malnutrition.	Increased with acute inflammation.

Figure 2. Abnormal laboratory values associated with eating disorders. Initial Laboratory assessment typically includes: complete blood count, comprehensive serum metabolic profile, electrolytes, liver function, enzymes and thyroid function test. Adapted from references 31 and 40.

It is important to engage individuals with an ED in a manner that preserves their respect as people with EDs have suffered from stigma and blame. Even health care providers have maintained a negative view of people with EDs (32). Know that the person with an ED also has more to them than their ED. They have a family, interests, and careers. Show interest in other aspects of their life. Understand that the person with an ED has a very real fear, however delusional, about weight gain, certain foods, and body dysmorphia, and that their symptoms may be made worse by other psychological issues such as obsessive compulsive disorder and anxiety. Clinicians need to know about the various "pro-ana" and "pro-mia" Web sites that some clients access as the content of these sites can give clinicians important information about strategies used by clients to maintain ED behaviors (33). The RD may want to read case examples of ED treatment (34) to gain further understanding of the illnesses.

#### **ROLE OF THE TREATMENT TEAM AND RD**

Multidisciplinary treatment for people with EDs is considered best practice in all treatment settings. Treatment teams minimally consist of a primary care physician, a mental health professional, a RD, and in cases involving preadolescents and adolescents, the family, unless there is compelling reason to exclude family members. In addition, the family may be important in treatment of older patients. Often a psychiatrist is involved to manage medication and to assess and treat comorbid conditions. Multidisciplinary teams need to be coordinated for consistent communication and to avoid "splitting." Frequently medical doctors do not have the time for team coordination; this is a role appropriate for RDs, nurses, therapists, and physician assistants with specialty training in EDs. Using these health care providers in this role represents a cost savings.

EDs frequently exist with psychological comorbid conditions such as depression, anxiety, borderline personality disorder, obsessive compulsive disorder, and substance use (9). Mental health professionals will identify these conditions, and discuss pertinent information with the RD as a treatment plan is developed (see "ROLES AND RESPONSIBILITIES OF THE REGISTERED DIETITIAN" in reference 2). Mental health conditions as well as degree of illness severity must be considered as the RD assesses the client and develops nutrition interventions. In FBT a professional needs training to coach the family in how to accomplish refeeding at home. The physician's role includes diagnosing, obtaining, and evaluating laboratory tests, physical status, and assessing progress. In outpatient settings many physicians are not ED specialists; this presents the opportunity for RDs who specialize in EDs to offer useful information to them.

EDs exist on a continuum of severity (28), becoming more severe as time passes. Depending on the severity of the illness, age of patient, and place of treatment, RDs will have varying levels of responsibility. The American Psychiatric Association (APA) has developed treatment guidelines for levels of care, dependent on illness severity (31). The APA guidelines contain important physical and psychological markers staging the illness. While not all ED treatment has utilized RDs as a routine part of treatment, patients and families have identified nutrition counseling as an important component of ED treatment (35).

The unique contribution of RDs is their vast knowledge of foods, food science, and how foods contribute to the health and well being of individuals. RDs have the capacity to evaluate diets and make changes based on an individual's medical condition, physical activity, food preferences, and religious beliefs as well as to evaluate whether food beliefs and behaviors fall into what might be considered the normal range. In addition, RDs are food and nutrition experts, able to understand the complex relationship of food intake to overall physiological health, which can be used to form nutrition education, and offer knowledge of foods, food products, formulas as needed by families and other health professionals.

All RDs, including entry-level RDs, need to know the basics about recognition of EDs (Figure 1). To effectively assess a client suspected of having an ED, RDs must be competent in analyzing labs and anthropometric data, knowing signs and symptoms of EDs, and understanding the need for immediate treatment. Every RD needs to know where and to whom to refer a suspected client with an ED within or outside of their community.

#### DIAGNOSIS, WARNING SIGNS, AND SYMPTOMS OF EATING DISORDERS

Eating disorders are considered serious mental illnesses and diagnosis is based on both psychological and medical symptoms. Warning signs, physical symptoms, as well as the formal DSM-IV diagnostic criteria serve to orient the RD to ED identification; however, the diagnosis of an ED is made by a physician or mental health professional. In cases of pediatric EDs, age appropriate classification, with consideration of the child's level of maturation, should be considered (36). Research shows that a person diagnosed with EDNOS does not have a less serious illness, although in many instances treatment is delayed because clinicians think the person is less ill (37). The DSM-V diagnostic criteria may remedy this situation. Being familiar with identification of EDs is a service to the multidisciplinary team and to health care providers who may not be ED specialists, as the RD can then bridge gaps in knowledge.

One area where RDs can identify warning signs and be instrumental in interventions to reduce disordered eating is in dieting behaviors in youth that have been identified as a risk factor in ED development (38). It is not possible to determine in advance who is genetically predisposed to an ED, thus it is important to intervene in school-based efforts to combat the obesity epidemic and ensure delivery of information to reduce childhood obesity does not do more harm than good. Project EAT (Eating Among Teens), a 5-year longitudinal study of overweight adolescents examined risk and protective factors for disordered eating. Risk factors included exposure to weight control methods in print media, irregular eating patterns, higher levels of weight concern, unhealthy weight control measures (skipping meals, fasting, smoking, use of food substitutes) and higher levels of physical activity. Eating meals on a regular basis, having more family meals, body satisfaction, were protective (39). The number of people who go on to develop full blown EDs is not determined in this study but it does suggest that at least disordered eating can be prevented. The RD can be instrumental in community efforts to reduce disordered eating in adolescents and to help identify disordered eating versus an ED.

#### ASSESSMENT

The nutrition assessment will have different components depending on the stage of illness and the treatment setting, whether inpatient, intensive outpatient, or outpatient. Inpatient programs, residential programs, and intensive outpatient programs generally have a protocol and will ask the RD to perform varying assessment tasks. A full nutrition assessment reveals current dietary intake, present eating patterns, history related to foods, nutrient deficiencies, supplement use, risk of refeeding syndrome, beliefs about food, binge purge patterns, and physical activity patterns. In children and adolescents, it is important to find out about family meals, who buys and prepares food and who will be providing care to the young person with an ED. In pediatric cases, where FBT is used, family norms about food, barriers to care, strengths, and needs are assessed. Parents will provide information about their child such as eating history, typical food intake, weight history, temperament, and behavior changes. In general, the younger a child, the more the RD will rely on parental report.

Letting clients tell their story provides an in-depth and personal picture of the ED. Evaluation of weight history and lifetime growth charts can provide information for estimation of goal weight. Inquiring about frequency of weight monitoring, desired weight, and body checking helps to assess fear of weight gain and body image concerns.

Assessing for typical meal patterns includes asking about timing of food intake, how holidays or family gatherings affect food intake, and information about food rules and rituals (Figure 1). A review of purging behaviors includes asking about amount, frequency, and type, including selfinduced vomiting, laxative use, use of enemas, suppositories, diuretics, and diet pills. Review physical activity routines including type of activity, sessions per week, intensity, and duration of exercise. Obtain information about exercise compulsions or exercise purging behaviors. The RD might ask: "How does your eating change if you take a day off? Do you do more training than recommended by your coach?" When exercise patterns interfere with social activities, become less enjoyable, continue despite injury and become more solitary, they can be a clue for presence of an ED.

In addition, RDs should assess level of counting calories, focusing on numbers, rigid food patterns, dietary extremes, or missing food groups. Vegetarian eating patterns which have not been part of family norms may be a warning sign for an ED. Macro and micronutrient deficiencies are evaluated by dietary intake, diet history, physical signs and symptoms, and laboratory values (40) (Figure 2). In uncomplicated starvation, laboratory values may be normal and then change once feeding begins. It is important to identify those at risk for refeeding syndrome and bring this to the attention of the treatment team.

If the RD is asked to help evaluate a patient for possibility of an ED they might consider using one of the many ED screening and assessment tools such as the Eating Disorders Inventory (EDI) or Eating Attitudes Test (EAT-26). Both of these are self report measures while the Eating Disorder Examination (EDE) is a clinician administered measure (41). A short questionnaire, the SCOFF, can be used by clinicians quickly as a screening tool (38).

After the nutrition assessment is complete the RD can determine a nutrition diagnosis, following the Nutrition Care Process. Sample nutrition diagnosis:

- 1. Protein calorie malnutrition related to [restrictive eating and excessive exercise] as evidenced by [state % weight loss, labs, anthropometric data, clinical signs and symptoms].
- Inadequate dietary intake, related to ED [insert specific diagnosis] as evidenced by [state dietary intake method used].
- 3. Micronutrient deficiency [select one] related to inadequate intake, as evidenced by labs or signs and symptoms.
- Excessive exercise with inadequate macronutrient intake, related to [use medically supplied diagnosis] as evidenced by [patient statements about exercise habit, logs of exercise, microfractures].
- 5. Disordered eating pattern related to bulimia nervosa as evidenced by binge–purge cycles of [times per day], [times per week].

#### TREATMENT

A majority of clients with EDs will be managed in an outpatient setting or will return to the outpatient setting from inpatient or residential settings. In the latter settings RDs have roles delineated within the multidisciplinary team. The outpatient or private practice RD may have less well defined tasks and therefore requires advanced training. Often the RD will have a suspected ED case referred from physicians, therapists, trainers, parents, or schools and need to complete the assessment, provide recommendations, and initiate the collaboration and communication. The RD needs to understand not to initiate treatment as a sole provider. The treatment team will continuously evaluate appropriateness of outpatient treatment. When the individual is unable to improve on an outpatient basis, referral to a higher level of care is in the client's best interest. Guidelines and criteria for hospitalization or residential treatment (eg, intensive out-patient) for a person with an ED have been listed in

Day	Calorie intake (all feeding routes)	Supplements
Day 1	10 kcal/kg/day For extreme cases (BMI <14 or no food >15 days) 5 kcal/kg/day Carbohydrate: 50%–60% Protein: 15%–20% Fat: 30%–40%	Prophylactic supplement PO <sub>4</sub> <sup>2</sup> - 0.5–0.8 mmol/kg/day K <sup>+</sup> : 1–3 mmol/kg/day Mg <sup>2+</sup> : 0.3–0.4 mmol/kg/day Na <sup>+</sup> : <1 mmol/kg/day (restricted) IV fluids-Restricted, maintain "zero" balance IV thiamin + vitamin B complex 30 minutes prior to feeding
Day 2 to 4	Increase by 5 kcal/kg/day	Check all biochemistry and correct any abnormality Thiamin + vitamin B complex orally or IV until day 3 Clinical and biochemical monitoring
Day 5 to 7	20–30 kcal/kg/day	Check electrolytes, renal, and liver functions and minerals Fluid: maintain zero balance Consider iron supplement from day 7
Day 8 to 10	30 kcal/kg/day or increase to full requirement	Clinical and biochemical monitoring
Table 1. Refeeding	regime for patients at risk of refeeding syndrome <sup>a</sup>	
<sup>a</sup> Reprinted with pe <sup>b</sup> BMI=body mass in	rmission from reference 43. Idex.	

many documents (31,38) and do vary between adults and adolescents. With case collaboration and clinical judgment this decision is made by considering medical stability, motivation and ability to make progress in outpatient settings and degree of safety (suicide plan or intent).

Medical decisions regarding stability in AN cases are based on pulse, blood pressure, body temperature, orthostatic changes, electrolyte imbalance, refusal to eat or lack of progress in the outpatient setting, percent of weight lost and percent body fat and cardiac arrhythmias (38). With bulimia nervosa different criteria for medical stability are taken into consideration and include syncope, serum electrolyte concentrations, esophageal tears, hematemesis, body temperature, suicide risk, cardiac arrhythmias, and also failure to progress in outpatient treatment (38). Often an adult patient has to agree to a higher level of care and this can bring up difficult ethical dilemmas. Written contracts agreed upon early in treatment can be helpful in making this determination.

Best treatment practices in general for AN and BN include early detection, early and aggressive intervention, and early return to physical stability. Service users in both the United States and the United Kingdom have identified lack of early intervention as being a barrier to recovery (35). Normalization of attitudes and eating behaviors usually follows physical restoration but this aspect of recovery can take years (27,42). Clinicians vary in terms of what they define as full recovery from EDs.

Social support can be very helpful to persons with both AN and BN. A recent study of user satisfaction of ED services identified supportive relationships with friends and family as being very important to the recovery process (35). In addition, social support has been shown to be helpful in alleviating caregiver stress (21). Evidence supports use of cognitive approaches (cognitive behavioral therapy and dialectical behavior therapy) in treatment of eating disorders. In persons with AN these therapies are often more effective once a person has initiated nutrition restoration. RDs can develop nutrition-related psychoeducation to educate patients, families, and friends (22); and RDs should elect to have training in therapy modalities to become more effective in delivery of nutrition advice, education, and use of food and food-related events in nutrition therapy. Many RDs choose to use the transtheoretical model and motivational interviewing to assess readiness to change and enhance motivation to change. RDs need to be cautious in the use of these approaches because in people who are very starved, motivation and insight into their disorder is lacking, due to starvation effects on cognition.

Some patients, once physically restored, want information about dining out, cooking, normal eating, and sports nutrition. With input from the therapist, the RD determines when and what information would benefit the patient. Patients with a long history of illness frequently report that they can't recall how to eat normally and they benefit from qualitative nutrition information. Meal plans, presented in a qualitative manner, can be an effective tool for some individuals, especially those who state they "need permission to eat." The RD and the treatment team will know detailed nutrition information (ie, calories, fat grams). This information can be harmful to the patient, leading to obsessive thoughts and symptoms. Parents of adolescents and preadolescents can be provided with the nutritional guidance to help them implement the nutritional plan at home.

#### **Refeeding Syndrome**

Refeeding syndrome is a cluster of metabolic events that occur in the starved catabolic individual when feeding is initiated. This syndrome is unpredictable, does not occur in every starved individual, but needs to be anticipated in any undernourished person as it is life threatening. Patients at risk for refeeding syndrome include those with anorexia nervosa, any child with prolonged under-nutrition over weeks and months, and individuals with a rapid and significant weight loss prior to hospital admission regardless of current weight. This includes individuals who are still clinically overweight but have recently lost a lot of weight, chronic diuretic users, those who have engaged in severe purging behaviors (vomiting, laxative misuse), and those with a concurrent medical condition such as sepsis or diabetes. The starved catabolic individual has low body stores of phosphate, thiamin, and potassium which are needed to synthesize metabolic intermediates, particularly of carbohydrate metabolism (44).

The main biochemical findings in refeeding syndrome are hypophosphatemia, hypomagnesemia, hypokalemia glucose intolerance, fluid overload, and thiamin deficiency. Serum electrolytes may be normal, while total body stores are low (44). The start of aggressive refeeding shifts serum electrolytes into the intracellular compartments, which drops serum levels (Table 1). Suggested protocols (40) for avoiding complications associated with refeeding include initiating replacement therapy for electrolytes and fluids prior to refeeding and monitoring electrolytes (K, P, Ca, Mg) every 4-6 hours at the beginning of refeeding for 3 days (some recommend 7 days) and replace suboptimal levels as needed. If serum levels of micronutrients are low then replacement is warranted.

The golden rule to avoid complications of refeeding syndrome is to feed very conservatively at the beginning of refeeding (43). Monitor glucose—those with hyperglycemia requiring exogenous insulin are at risk for development of refeeding syndrome upon receipt of insulin because of the sequence of events leading to influx of electrolytes and glucose into cells (44). One treatment center has examined the role of macronutrient distribution on weight restoration and incidence of refeeding syndrome in 2,000 patients with AN. They identified three refeeding stages during which they manipulated macronutrients and by using this protocol along with rapid weight gain observed better outcomes and fewer instance of refeeding syndrome (45).

**Common vitamin and mineral deficiencies** Patients at risk for refeeding syndrome should receive adequate thiamin and magnesium (44,43). Thiamin and magnesium levels are frequently low in the starved patient and thiamin requirements will increase with refeeding as thiamin derived intermediates (thiamin pyrophosphate), used in carbohydrate metabolism are synthesized (Table 1). A multiple vitamin/ mineral supplement is often given at the start of refeeding and represents a conservative approach to supplementation.

Many people are deficient in vitamin D and the malnourished patient may also not be consuming adequate calcium. These individuals are at risk for osteopenia. Check vitamin D levels and correct with supplementation. Ensure adequate calcium in the diet; or, if not possible, suggest an age appropriate supplement. Numerous reports have documented low zinc in both AN and BN. Zinc supplementation is often recommended. Frequently patients with EDs are anemic due to deficiencies of iron and folate. Refeeding can correct this but if severe anemia is present, then replace using supplementation appropriate for age.

#### **Disease-Specific Treatment Guidelines**

Anorexia nervosa AN is characterized by varying degrees of starvation, so RDs need to understand the mechanics, physical adaptations, and refeeding involved in starvation from any cause and then understand the unique aspects of the anorexic mind. The first element of treatment for AN is medical stabilization, which, if the person meets hospitalization criteria will take place in the inpatient setting. This will involve supervised feeding and sometimes nutrition support, which can be nasogastric tube feeding (38,46). RDs need to be aware that in undernourished patients the gastrointestinal tract is compromised and malabsorption will be present, along with delayed gastric emptying, reduced gastric and pancreatic secretions, and villous atrophy, all of which can cause gastrointestinal distress which improves with refeeding (47).

Patients with AN may be cooperative as inpatients and in residential settings because of constant supervision and support. In outpatient settings, the person with AN often becomes resistant to further efforts to maintain feeding and weight. FBT makes use of the lessons learned from the constant supervision in the inpatient setting and empowers the family to take on the role of supervising all meals and snacks. Social support is a method where individuals may call upon family and friends to help them continue with recovery oriented behaviors. Research continues on various techniques helpful to patients recovering from anorexia. There is research suggesting relaxation techniques such as guided imagery and progressive muscle relaxation can reduce postprandial anxiety (48). Similarly, use of concepts from intuitive eating may be helpful later in treatment (49).

With EDs, the entire treatment team has to understand that not eating is not an option, that food is medicine, and that the person with an ED does not have a choice about whether to eat, they do have a choice in where they will eat—in the hospital or residential treatment center or at home. If hospitalized, the choice for the patient is to eat orally or via nasogastric tube (38,42.) See the section "Advancing Treatment in the Face of Denial of Illness" later in this paper for more discussion about refeeding when the patient does not want intervention. Once physical restoration is achieved the process of psychological restoration and return to normalized eating begins.

In the adult population with AN the rate of weight gain can vary but research has shown a gain of more than 0.8 kg/wk during inpatient stay is associated with better outcome (50). The APA recommends a weight gain of 2 to 3 pounds per week (0.9-1.4 kg/wk) during inpatient treatment (34,50). In outpatient treatment weight gain goals are ½ to 1 lb/wk. In chronically ill adults with AN the first step is stopping weight loss and maintaining weight before weight gain can be achieved. Resting energy expenditure can be greatly increased (51) during refeeding leading to high calorie dietary needs in order to promote weight gain. Higher energy density and a greater amount of dietary variety are associated with better outcome in weight restored people with AN (52). In order to promote weight gain, maintain or improve cardiac function, and normalize laboratory values it is often advisable to restrict or cease exercise (31).

Patients are frequently concerned about how weight is gained because in patients with very low body mass index (BMI) of 13-14, fat mass is preferentially gained over fat free mass (53). The patient might notice that they have weight gain in the abdominal area but this eventually redistributes (22,54). RDs can provide education to patients and/or the treatment team about the physiology of weight gain. Clinicians need to be aware of methods used by patients to manipulate their weight, such as water loading or hiding weights in clothing. Urine specific gravity below 1.010 may indicate water loading.

For all malnourished clients, adjustments to caloric needs are done on an individual basis based on desired rate of weight gain, treatment setting, labs, and vital signs. In pediatric clients weight gains of 0.5 to 2 lb/week are recommended (55). Calories often need to be monitored frequently and adjusted upward before any real weight restoration takes place (42). Rate of inpatient weight restoration is a predictor of outcome in AN and a threshold BMI of 20 is associated with better outcome (50).

One study concluded that recently weight restored female patients with AN had better prognosis when body fat reached at least 25% (56), and other studies have shown better prognosis when weight is gained in a shorter length of time and goal weights are set at higher rather than minimal levels (50,56,57). Goal weights for adults are more a state of health and can span a range of weight. The treatment team can determine the state of health based on endocrine status, percent body fat, cognitive improvements, labs, overall nutrition status, and food intake. Discussion of a weight range rather than a specific target weight may be in the patient's best interest as it allows clinical judgment some latitude in determination of weight.

The determination of appropriate goal weight for preadolescent and adolescent cases is more of an art than a science. Ideally growth records during childhood can aid in understanding weight trends and growth velocity. Setting a target weight that is too low may perpetuate the problem of an ED so it must be done collaboratively with the treatment team and parents. For a growing child, weight goals and energy needs must be readjusted every 3 to 6 months to account for growth. An excellent resource for determining pediatric target weights is available at http:// www.kartiniclinic.com/blog/determining-ideal-body-weight (57). Special considerations when determining desirable body weight for growing children and adolescents include: developmental stage when ED occurred, weight at loss of menses, menstrual history and athletic build. Preliminary research has shown that menses returns at an average of 4.5 lb greater than the weight where an individual experienced their last period (55). Research has shown that in some AN patients, menses does not re-establish, despite return to what is considered normal weight and that hypometabolism exists in these patients perhaps as a metabolic response to continued low energy intakes (58). Oral contraceptives to treat amenorrhea does not demonstrate efficacy in the treatment to aid in reproductive or bone health (4).

When looking at growth trends during adolescence it is important to understand normal growth and development. Weight gain expectations for girls age 10 to 14 years may be up to 37 lb. During high school from ages 14 to 18, expect to see an additional 20 lb on average. Similarly, the average boy gains 11 lb yearly for a total of 45 lb between ages 12 and 16 years (59).

Bulimia nervosa In the treatment of BN, the RD will most often be asked to provide information about appropriate calories, macro and micro nutrients, correction of electrolyte disturbances, provision of nutrition education in conjunction with psychotherapy, and assist with teaching a pattern of normal eating. Research indicates that adolescents with BN have better outcome if they reduce binge purge episodes early in treatment (60). Strategies reported to decrease purging include self monitoring (of eating behaviors, factors that precede and follow restricting, bingeing, and purging) and menu planning. RDs can provide a meal strategy to help break chaotic eating patterns of restricting or skipping meals (61). Consider timing of meals to reduce bingeing and purging due to starvation and to promote structure, satiety, and nutritional adequacy. Hypocaloric intake will cause deregulation of appetite, perpetuate binge eating and thus slow progress. While we have adhered to a philosophy of "all foods fit," there may be important research findings suggesting manipulation of macronutrients or highly palatable foods to avoid activation of certain brain systems involved in bingeing. Binge eating can be produced in a rat model exposed to food restriction followed by exposure to highly palatable foods, which represents "restraint stress" (62).

With older adolescents and adults the RD might identify foods that elicit binge episodes as well as what situations promote bingeing. Favorite binge foods may or may not be incorporated into meals at the start of treatment based on client profile and in consult with a mental health professional. Laxative abuse can take months to resolve and is associated with disturbances of electrolytes, as well as hydration abnormalities. High fiber and increased fluids aid in restoration of bowel function.

Parents using the family-based approach to treat adolescents who are purging can implement protocols for meal support, post meal monitoring, and bathroom supervision. **Binge eating disorder** BED is currently being defined and diagnostic criteria developed (63,64). There is still controversy about what constitutes a binge eating episode. There is agreement that a binge eating episode occurs within a certain time frame, less than 2 hours, and that feeling loss of control is a central feature, but the size of the binge or food/caloric amount remains a topic of discussion (63). This is the objective (definition) verses subjective (what the client believes is a binge) definition of bingeing. Clinicians need to ask a patient to describe their binge episodes, as often the client report does not describe what would be considered a binge episode. Clients who are refeeding from malnutrition experience what they subjectively feel is a binge, but this may in actuality be a psychological or physiological response to starvation.

There is discussion about whether binge eating represents purely emotional eating or whether it is an abnormality in physiology, as evidenced by genetic studies and neurochemistry studies, such as the glutamate hypothesis.

The RD with experience can help the treatment team in identification of BED and in prevention of binges. A recent study of clients with BED found that interpersonal psychotherapy and cognitive behavior therapy-guided selfhelp are more effective than behavioral weight loss to decrease BED at 2 year follow-up (65,66). Recently, dialectical behavioral therapy showed promising results in treatment of BED (24). For individuals recovering from BED, the RD may be asked to provide nutrition education on normal eating and to evaluate the patient for nutrition requirements.

If the child has BED the RD can instruct the family on household environmental controls regarding food to reduce bingeing. The RD can provide nutrition education to the family regarding adequate nutrition for the age and physical activity level of the child.

#### **CONTROVERSIAL TOPICS**

#### Advancing Treatment in the Face of Denial of Illness

Denial of illness and avoidance of treatment are common in persons with EDs and yet, as people go through treatment and cognition returns to a healthy state, many convert to believing that treatment was necessary (67). Many treatment programs have no tolerance for not eating and FBT also advocates that eating must occur. Yet, some clinicians still feel that the person with an ED has to want to eat and so waiting for them to decide to eat is best. There is ongoing debate and lack of consensus about how to have a person eat when they absolutely refuse and then when to admit them to a hospital for care as they deteriorate. Some clinicians state that EDs need to be viewed as a form of self harm, no different from suicidal intent, and therefore admitting a person involuntarily ought to be viewed as a lifesaving part of treatment (68).

#### Weight as a Health Measure

The upcoming revision to the DSM-IV may drop weight as a criteria in the diagnosis of anorexia nervosa. This is because a person might have started out overweight and then rapidly lost weight, be normal weight, or even overweight while being medically compromised. Using weight as a diagnostic criteria has been criticized as arbitrary and insensitive to issues of gender, ethnicity, frame size, and age (69). In addition, when looking at a group of weight restored patients (58), those who had resumption of menses had a higher percent of ideal body weight, BMI, and resting metabolic rate then their amenorrheic counterparts. While weight is one measure to use for judging clinical progress it is helpful to think of weight as reflective of a state of health rather than an end goal in and of itself. The healthy weight becomes that weight where a person is physically and mentally healthy and has all body functions restored to normal. It is the end result of consuming an adequate diet and engaging in reasonable physical activity.

#### **Diagnostic Criteria**

Current diagnostic criteria for AN state "refusal to maintain body weight" at a certain level (70) and the term "refusal" is now being reconsidered as it is potentially misleading. The term refusal implies an act of will or stubbornness, while current research suggests an inability to maintain weight, rather than frank refusal (67). For example Hebebrand and colleagues pose the idea that the restlessness observed in many with anorexia is the result of a deficiency in leptin, leading to difficulty in energy balance (71). The DSM-V will drop the cessation of menses criteria because it is irrelevant in cases of young children and males and is inconsistently a marker of physical wellness in adult females. Finally, the current diagnostic systems, including the upcoming DSM-V do not fully describe the grey areas in the eating disorder spectrum and may define eating disorders too rigidly to be clinically useful (72).

#### SUMMARY STATEMENT

RDs have a unique role to play in the identification, assessment, and treatment of eating disorders. This responsibility calls for a higher level of training and clinical judgment as a protection for patients with EDs. The field of dietetics needs to continuously participate in research demonstrating the effectiveness of RDs in treatment of EDs, where in the continuum of care RDs are most effective, and what unique clinical practices RDs develop and implement in ED treatment. The newly published standards of practice can serve as a guide for RDs pursuing this realm of nutrition therapy (73).

#### Acknowledgements

The American Dietetic Association (ADA) authorizes republication of the practice paper, in its entirety, provided full and proper credit is given. Readers may copy and distribute this paper, providing such distribution is not used to indicate an endorsement of product or service. Commercial distribution is not permitted without the permission of ADA. Requests to use portions of the paper must be directed to ADA headquarters at 800/877-1600, ext 4835, or ppapers@eatright.org. This paper will be up for review in December 31, 2014.

*Authors:* Therese Waterhous, PhD, RD, LD (Willamette Nutrition Source LLC, DBA Willamette Eating Disorders Treatment Network, Corvallis, OR); Melanie A. Jacob, RD (Nutrition Therapy, LLC, Troy, MI).

*Project Lead:* Amy D. Ozier, PhD, RD, LDN, CHES (Northern Illinois University, DeKalb, IL).

Reviewers: Jeanne Blankenship, MS, RD (ADA Policy Initiative & Advocacy, Washington, DC); Jennifer Burnell, MS, RD, LDN (Carolina House, Durham, NC); Sharon Denny, MS, RD (ADA Knowledge Center, Chicago, IL); Sports, Cardiovascular and Wellness Nutrition dietetic practice group (DPG) (Pamela Kelle RD, LDN, CED-RD, Pamela Kelle Nutrition Consultant, Chattanooga, TN); Sharon McCauley, MS, MBA, RD, LDN, FADA (ADA Quality Management, Chicago, IL); Kimberli McCallum, MD (McCallum Place, St. Louis, MO); Eileen Stellefson Myers, PH, RD, LD (Private Practice, Nashville, TN); Esther Myers, PhD, RD, FADA (ADA Research & Strategic Business Development, Chicago, IL); Pediatric Nutrition DPG (Bonnie A. Spear, PhD, RD, University of Alabama at Birmingham, AL); Lisa Spence, PhD, RD (ADA Research & Strategic Business Development, Chicago, IL); Behavioral Health Nutrition DPG (Mary M. Tholking, MEd, RD, LD, self-employed, Clarksville, OH); Lisa Van Dusen, MS, RD, LDN (University of Massachusetts Memorial Medical Center, Worcester, MA).

*Association Positions Committee Workgroup:* Alana Cline, PhD, RD (chair); Connie B. Diekman, MEd, RD, LD, FADA; Ellen Lachowicz-Morrison, MS, RD, LDN, CEDRD (content advisor).

We thank the reviewers for their many constructive comments and suggestions. The reviewers were not asked to endorse this paper.

#### References

- American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4<sup>th</sup> ed (text revision). Arlington, VA: American Psychiatric Association; 2000.
- 2. American Dietetic Association. Position of the American Dietetic Association: Nutrition intervention in the treatment of eating disorders. *J Am Diet Assoc.* 2011;111:1236-1241.
- **3.** Attia E, Walsh T. Anorexia nervosa. *Am J Psychiatry.* 2007;164:1805-1810.
- O'Toole J. *Give Food A Chance: A New View on Childhood Eating Disorders.* Portland, OR: Perfectly Scientific Press; 2010:71-109,195-222.
- 5. Insel T, Wang P. Rethinking mental illness. *JAMA*. 2010;303:1970-1971.
- **6.** Schmidt U, Treasure J. Anorexia Nervosa: Valued and visible. A cognitive-interpersonal maintenance model and its implications for research and practice. *Br J Clin Psychol.* 2006;45:1-25.
- 7. Kaye W, Fudge J, Paulus M. New insights into symptoms and

neurocircuit function of anorexia nervosa. *Nat Rev Neurosci.* 2009;10:573-584.

- 8. Kaye W. Neurobiology of anorexia and bulimia nervosa. *Physiol Behav.* 2008;94:121-135.
- **9.** Klump KL, Bulik C, Kaye W, Treasure J, Tyson E. Academy for Eating Disorders position paper: Eating disorders are serious mental illnesses. *Int J Eat Dis.* 2009;42:97-103.
- Ploj K, Albery-Larsdotter S, Arlbrandt S, Kjaer MB, Skantze PM, Storlien LH. The metabotrophic glutamate mGluR5 receptor agonist CHPG stimulates food intake. *Neuroreport*. 2010;21(10):704-708
- **11.** Bohon C, Stice E, Burton E. Maintenance factors for persistence of bulimic pathology: a prospective natural history study. *Int J Eat Disord.* 2009;42:173-178.
- Kalm LM, Semba RD. They starved so that others be better fed: Remembering Ancel Keys and the Minnesota experiment. *J Nutr.* 2005;135:1347-1352.
- Hatch A, Madden S, Kohn MR, Clarke S, Touyz S, Gordon E, Williams LM. In first presentation adolescent anorexia nervosa, do cognitive markers of underweight status change with weight gain following a refeeding intervention? *Int J Eat Disord*. 2010;43:295-306.
- **14.** LeGrange D, Lock J, Loeb K, Nicholls D. Academy for Eating Disorders position paper: The role of the family in eating disorders. *Int J Eat Disord*.2010;43:1-5.
- Sim LA, Homme JH, Lteif AN, Vande Voort JL, Schak KM, Ellingson J. Family functioning and maternal distress in adolescent girls with anorexia nervosa. *Int J Eat Disord*. 2009;42:531-539.
- **16.** Van Son GE, Van Hoeken D, Van Furth EF, Donker GA, Hoek HW. Course and outcome of eating disorders in a primary carebased cohort. *Int J Eat Disord*.2010;43:130-138.
- American Academy of Pediatrics, Committee on Adolescence. Identifying and treating eating disorders. *Pediatrics*. 2003;111:204-211.
- Le Grange D, Eisler I. Family interventions in adolescent anorexia nervosa. *Child Adolesc Psychiatric Clin N Am.* 2009;18:159-173.
- **19.** Bulik CM, Baucom DH, Kirby JS, Pisetsky E. Uniting couples (in the treatment of) anorexia nervosa (UCAN). *Int J Eat Disord*. 2011;44:19-28.
- **20.** Goldstein M, Peters L, Baillie A, McVeagh P, Minshall G, Fitzjames D. The effectiveness of a day program for the treatment of adolescent anorexia nervosa. *Int J Eat Disord.* 2011;44:29-38.
- **21.** Kyriacou O, Treasure J, Schmidt U. Understanding how parents cope with living with someone with anorexia nervosa: modeling the factors that are associated with carer distress. *Int J Eat Disord.* 2008;41:233-242.
- **22.** Garner D, Garfinkle P. *Handbook for Treatment of Eating Disorder. Psychoeducational Principals in Treatment.* New York, NY: The Gillford Press; 1997:145-177.
- **23.** Brennan B, Roberts J, Fogarty K, Reynolds K, Jonas J, Hudson J. Memantine in the treatment of binge eating disorder: an open-label, prospective trial. *Int J Eat Disord.* 2008;41:520-526.
- 24. Chen EY, Matthews L, Allen C, Kuo JR, Linehan MM. Dialectical behavior therapy for clients with binge eating disorder or bulimia nervosa and borderline personality disorder. *Int J Eat Disord.* 2008;41:505-512.
- 25. Hilbert A, Czaja J. Binge eating in primary school children:

towards a definition of clinical significance. *Int J Eat Disord*. 2009;42:235-243.

- **26.** Javaras KN, Laird NM, Reichborn-Kjennerud T, Bulik CM, Pope HG, Hudson JI. Familiality and heritability of binge eating disorder: results of a case-control family study and a twin study. *Int J Eat Disord.* 2008;41:174-179.
- 27. Rome ES, Ammerman S, Rosen DS, Keller RJ, Lock J, Mammel KA, O'Toole J, Rees JM, Sanders MJ, Sawyer SM, Schneider M, Sigel E, Silber TJ. Children and adolescents with eating disorders: the state of the art. *Pediatrics*. 2003;111:98-108.
- 28. Maguire S, Le Grange D, Surgenor L, Marks P, Lacey H, Touyz S. Staging anorexia nervosa: conceptualizing illness severity. *Early Interv Psychiatry.* 2008;2:3-10.
- **29.** Hamli K. The multimodal treatment of eating disorders. *World Psychiatry*. 2005;4:69-73.
- **30.** Keel P, Brown T. Update on course and outcome in eating disorders. *Int J Eat Disord.* 2010;43:195-204.
- **31.** American Psychiatric Association. APA practice guidelines: Treatment of patients with eating disorders, third edition; Level of care guidelines for patients with eating disorders. *Psychiatryonline* Web site. http://www.psychiatryonline.com/ popup.aspx?aID=139471. Accessed Jan. 2010 [JTS1].
- **32.** Currin L, Waller G, Schmidt U. Primary care physicians knowledge of and attitudes toward the eating disorders: do they affect clinical actions? *Int J Eat Disord.* 2009;42:453-458.
- **33.** Harshbarger J, Ahlers C, Mayans L, Mayans D, Hawkins J. Pro-anorexia websites: what every clinician should know. *Int J Eat Disord.* 2009;42:367-370.
- Alexander J, Le Grange D. My Kid is Back: Empowering Parents to Beat Anorexia Nervosa. Melbourne, Australia: Melbourne University Press; 2009.
- **35.** Escobar-Koch T, Banker J, Crow S, Cullis J, Ringwood S, Smith G, Furth E, Westin K, Schmidt U. Service user's views of eating disorder services: an international perspective. *Int J Eat Disord.* 2010;43:549-559.
- 36. J, Bryant-Waugh R, Herzog D, Katzman D, Kreipe RD, Lask B, LeGrange D, Lock J, Loeb K, Madden S, Nicholls D, O'Toole J, Pinhas L, Rome E, Sokol-Burger M, Wallen U, Zucker N; Workgroup for Classification of Eating Disorders in Children and Adolescents. Classification of child and adolescent eating disturbances. Workgroup for Classification of Eating Disorders in Children and Adolescents (WCEDCA). *Int J Eat Disord.* 2007;40:S117-S122.
- Peebles R, Hardy K, Wilson J, Lock J. Are diagnostic criteria for eating disorders markers of medical severity? *Pediatrics*. 2010;125:1193-1201.
- 38. Rosen DS; American Academy of Pediatrics Committee on Adolescence. Clinical report-identification and management of eating disorders in children and adolescents. *Pediatrics*. 2010;126:1240-1253.
- **39.** Neumark-Sztainer D, Wall M, Story M, Sherwood ME. Five year longitudinal predictive factors for disordered eating in a population-based sample of overweight adolescents: implications for prevention and treatment. *Int J Eat Disord*. 2009;42:664-672.
- 40. Fischbach F, Dunning M. A Manual of Laboratory and Diagnostic Tests. Philadelphia, PA: Lippincott, Williams and Wilkins; 2004.
- 41. Academy for Eating Disorders. Clinical measures toolkit.

http://www.aedweb.org/AM/Template.cfm?Section=Research\_ Practice\_Guidelines&Template=/CM/ContentDisplay. cfm&ContentID=1900#CIT. Accessed October 10, 2010.

- **42.** Fisher M. Treatment of eating disorders in children, adolescents and young adults. *Pediatr Rev.* 2006;27:16.
- 43. Khan LUR, Ahmed J, Khan S, MacFie J. Refeeding syndrome: a literature review. *Gastroenterol Res Pract*, 2011. http://www.hindawi.com/journals/grp/2011/410971/. Accessed February 22, 2011.
- 44. McCray S, Walker S, Parrish CR. Much ado about refeeding. *Pract Gastroenterol.* 2005;12:26-44.
- **45.** Anzai N, Hackert A. Accelerated healing: the benefits of rapid weight restoration in anorexia nervosa. *SCAN PULSE*. 2010;29:10-12.
- 46. Robb AS, Silber TJ, Orrell-Valente JK, Valadez-Meltzer A, Ellis N, Dadson MJ, Chatoor I. Supplemental nocturnal nasogastric refeeding for better short-term outcome in hospitalized adolescent girls with anorexia nervosa. *Am J Psychiatry*. 2002;159:1347-1353.
- **47.** Winter TA, The effects of undernutrition and refeeding on metabolism and digestive function. *Curr Opin Clin Nutr Metab Care.* 2006;9:596-602.
- 48. Shapiro JR, Pisetsky EM, Crenshaw W, Spainhour S, Hamer RM, Dymek-Valentine M, Bulik CM. Exploratory study to decrease postprandial anxiety: just relax! *Int J Eat Disord*. 2008;41:728-733.
- **49.** Tylka T. Psychometric evaluation of a measure of intuitive eating. *J Couns Psychol.* 2006;53:226-240.
- **50.** Lund BC, Hernandez E, Yates W, Mitchell J, McKee P, Johnson C. Rate of inpatient weight restoration predicts outcome in anorexia nervosa. *Int J Eat Disord.* 2009;42:301-305.
- 51. Van Wymelbeke V, Brondel L, Marcel Brun J. Rigaud D. Factors associated with the increase in resting energy expenditure during refeeding in malnourished anorexia nervosa patients. *Am J Clin Nutr.* 2004;80:1469-1477.
- 52. Schebendach JE, Mayer LE, Devlin MJ, Attia E, Contento IR, Wolf RL, Walsh BT. Dietary energy density and diet variety as predictors of outcome in anorexia nervosa. *Am J Clin Nutr.* 2008;87:810-816.
- **53.** Yamashita S, Kawai K, Yamanaka T, Inoo T, Yokoyama H, Morita C, Takii M, Kubo C. BMI, body composition, and the energy requirement for body weight gain in patients with anorexia nervosa. *Int J Eat Disord.* 2010;43:365-371.
- 54. Mayer L, Klein A, Black E, Attia E, Shen W, Mao X, Shungu D, Punyanita M, Gallagher D, Wang J, Heymsfield S, Hirsch J, Ginsberg H, Walsh, BT. Adipose tissue redistribution after weight restoration and weight maintenance in women with anorexia nervosa. *Am J Clin Nutr.* 2009;90:1132-1137.
- **55.** Society for Adolescent Medicine. Eating disorders in adolescents: position paper of the Society for Adolescent Medicine. *J Adolesc Health.* 2003;33:496-503.
- 56. Mayer L, Roberto C, Glasofer D, Etu S, Gallagher D, Wang J, Heymsfield S, Pierson R, Attia E, Devlin M, Walsh BT. Does percent body fat predict outcome in anorexia nervosa. *Am J Psychiatry*. 2007;164: 970-972.
- 57. O'Toole J. Determining ideal body weight. Kartini Clinic for Disordered Eating Web site. http://www.kartiniclinic.com/ blog/determining-ideal-body-weight/. Posted July 22, 2008. Accessed October 10, 2010.
- 58. Sterling WM, Golden NH, Jacobson MS, Ornstein RM,

Hertz SM. Metabolic assessment of menstruating and nonmenstruating normal weight adolescents. *Int J Eat Disord.* 2009;42:658-663.

- 59. Centers for Disease Control and Prevention. Weight for age tables, children ages 2 to 20 years selected percentiles. http://www.cdc.gov/growthcharts/html\_charts/wtage.htm. Accessed October 10, 2010.
- **60.** Le Grange D, Doyle P, Crosby RD, Chen E. Early response to treatment in adolescent bulimia nervosa. *Int J Eat Disord*. 2008;41:755-757.
- **61.** Shah N, Passi V, Bryson S, Agras WS. Patterns of eating and abstinence in women treated for bulimia nervosa. *Int J Eat Disord.* 2005;38:330-334.
- **62.** Bello NT, Guarda AS, Terrillion CE, Redgrave GW, Coughlin JW, Moran TH. Repeated binge access to a palatable food alters feeding behavior, hormone profile, and hindbrain c-Fos responses to a test meal in adult male rats. *Am J Physiol Regul Integr Comp Physiol.* 2009;297:622-631.
- **63.** Marcus MD, Kalarchian MA. Binge eating in children and adolescents. *Int J Eat Disord.* 2003;34:S47-S57.
- **64.** Wolf BE, Baker, CW, Smith AT, Kelly-Weeder S. Validity and utility of the current definition of binge eating. *Int J Eat Disord.* 2009;42:674-686.
- **65.** Wilson G, Wilfley D, Agras W, Bryson S. Psychological treatments of binge eating disorder. *Arch Gen Psychiatry.* 2010;67:94-101.

- 66. Lynch FL, Striegel-Moore RH, Dickerson JF, Perrin N, Debar L, Wilson GT, Kraemer HC. Cost-effectiveness of guided selfhelp treatment for recurrent binge eating. J Consult Clin Psychol. 2010;78:322-333.
- **67.** Guarda A, Pinto A, Coughlin J, Hussain S, Haug N, Heinberg L. Perceived coercion and change in perceived need for admission in patients hospitalized for eating disorders. *Am J Psychiatry.* 2007;164:108-114.
- **68.** Anderson A. Eating disorders and coercion. *Am J Psychiatry.* 2007;164:9-11.
- **69.** Wilfley D, Bishop M, Wilson T, Agras W. Classification of eating disorders. *Int J Eat Disord*. 2007;40:S123-S129.
- **70.** Becker A, Eddy KT, Perloe A. Clarifying criteria for cognitive signs and symptoms for eating disorders in DSM-V. *Int J Eat Disord*.2009;42:611-619.
- Hebebrand J, Muller TD, Holtkamp K, Herpertz-Dahlmann B. The role of leptin in anorexia nervosa: clinical implications. *Molecular Psychiatry*.2007;12:25-35.
- 72. Fairburn CG, Cooper Z. Eating disorders, DSM-5 and clinical reality. *Br J Psychiatry.* 2011;198:8-10.
- **73.** American Dietetic Association. Standards of practice and standards of professional performance for registered dietitians in disordered eating and eating disorders. *J Am Diet Assoc.* 2011;111:1242-1249.