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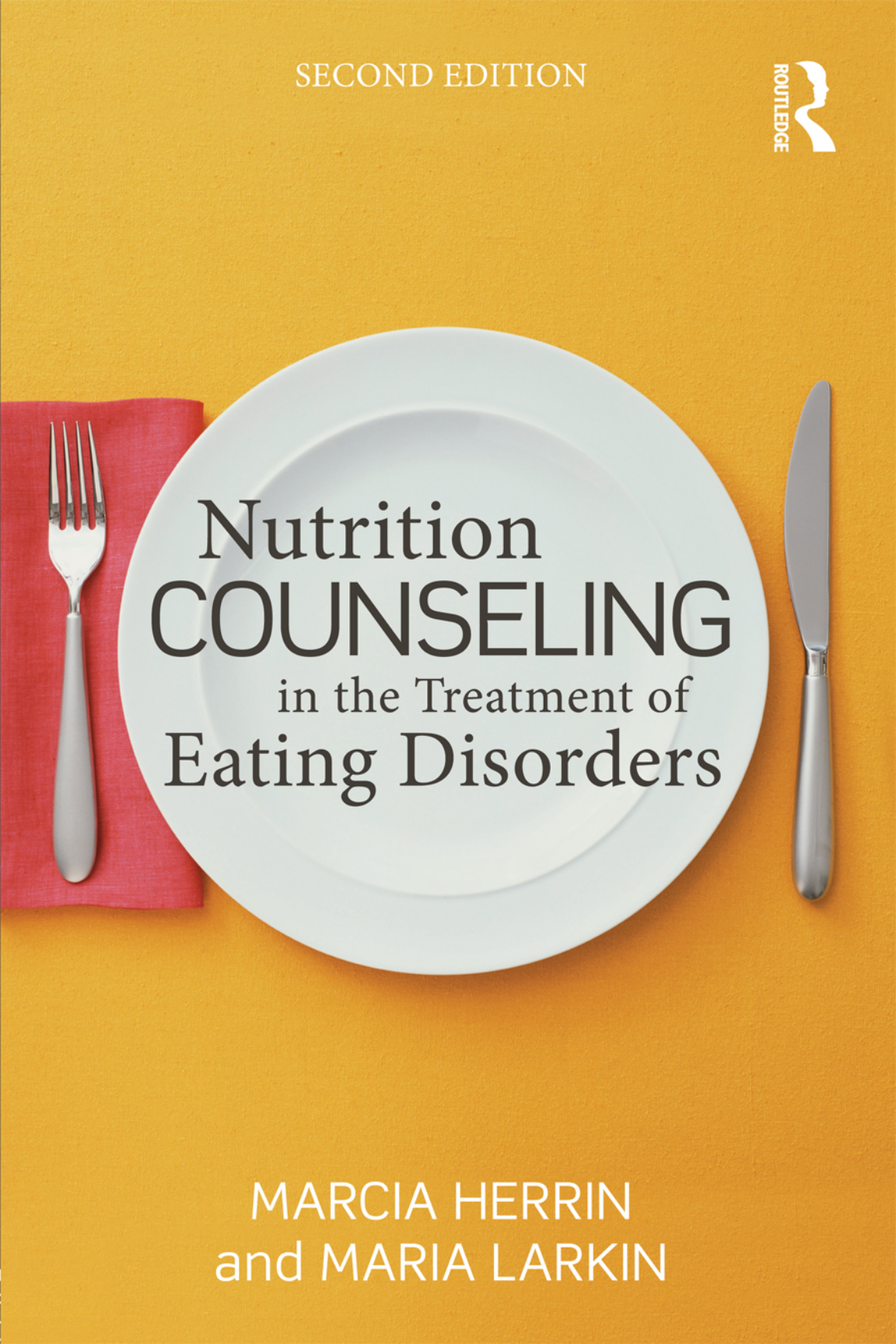

Nutrition COUNSELING in the Treatment of Eating Disorders

MARCIA HERRIN
and MARIA LARKIN

NUTRITION COUNSELING IN THE TREATMENT OF EATING DISORDERS

Marcia Herrin and Maria Larkin have collaborated on the second edition of *Nutrition Counseling in the Treatment of Eating Disorders,* infusing research-based approaches and their own clinically-refined tools for managing food and weight-related issues. New to this edition is a section on nutrition counseling interventions derived from cognitive-behavioral therapy-enhanced dialectical behavioral therapy, family-based treatment, and motivational interviewing techniques. Readers will appreciate the state of the art nutrition and weight assessment guidelines, the practical clinical techniques for managing bingeing, purging, excessive exercise, and weight restoration, as well as the unique food planning approach developed by the authors. As a comprehensive overview of food- and weight-related treatments, this book is an indispensible resource for nutrition counselors, psychotherapists, psychiatrists, physicians, and primary care providers.

Marcia Herrin, EdD, MPH, RD, LD, is the founder of Dartmouth College's eating disorders program. She has worked in the area of eating disorders since 1987 and is co-author of *The Parent's Guide to Eating Disorders*. Dr Herrin is a frequent presenter at conferences and offers supervision for other professionals. She has a private practice in Lebanon, New Hampshire.

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NUTRITION COUNSELING IN THE TREATMENT OF EATING DISORDERS

Second Edition

MARCIA HERRIN AND MARIA LARKIN

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To my grandmother, Catherine Giudici Burke, in loving memory.
Marcia Herrin

To my father, Giovanni Salvatore Miccio, for believing in me.
Maria Larkin

CONTENTS

PREFACE

We recognize the world is a changing place. Indeed, there are few constants in life. This truth has permeated our experience over the last two and a half years as we have laboriously researched and written this book. As our book underwent a transformation from the first edition to this new second edition, as new references replaced the old, and as chapters were renamed, reorganized and updated, we simultaneously observed many developments in the field of nutrition and eating disorders. In the last few months, the American Dietetic Association (ADA) changed its name to the Academy of Nutrition and Dietetics (AND) and produced its first position statement on and standards of practice for the treatment of eating disorders. Recently, the American Psychiatric Association (APA) proposed the DSM-5, new revisions to the DSM-IV diagnostic criteria for eating disorders.

We even hesitate to say our book is finished, as research in the field of eating disorders is emerging by leaps and bounds. Two days before our book deadline, while opening her mail, Larkin found in the *Journal of the Academy of Nutrition and Dietetics* (formerly the *Journal of the American Dietetic Association*), a new research study and shared it with Herrin over the phone. The next day Herrin applied the research information in a counseling session with a patient. This example of applied research is just one of many that occurred during the course of our researching and writing of this book. In fact, we feel the strength of our book lies in our research-based recommendations and in our successful application of information that has, time after time, improved outcomes for our patients. As a result of writing this book, we have become more skilled and knowledgeable about nutrition counseling in the treatment of eating disorders. Yet, we still consider ourselves students—less "green" than before (Larkin's masters program used the first edition as a text)—and still learning from our patients, colleagues, and researchers in the field.

Waterhous and Jacob remind us in their excellently written practice paper, *Nutrition Intervention in the Treatment of Eating Disorders*, "There is no professional consensus on how to restore weight and health in AN or stop binge and purge behaviors in BN or halt the eating disorder thoughts common to both" (Waterhous & Jacob, 2011, p. 1). And so, in this second edition, we offer not only our knowledge, skills, and insights but also pertinent techniques from cognitive-behavioral therapy-enhanced dialectical behavioral therapy, family-based treatment, and motivational interviewing techniques. Although we are not credentialed psychotherapists, we are skilled nutrition counselors and registered dietitians who garner from the evidence-based field of psychotherapy as well as from the field of nutrition that which is instructive to our patients. We have provided here detailed, clinically-oriented guidelines for assessing and monitoring weights and for weight restoration in the eating-disordered patient. We added to this edition a chapter on family-based treatment and levels of care for eating disorder treatment.

We hope the reader (nutrition counselors, psychotherapists, cognitive behaviorists, psychiatrists, physicians, and primary care providers, or whoever you may be) will benefit from the techniques, literature reviews, and clinical accounts we present in this book. We highlight these clinical accounts as case examples throughout the book. One remarkable account is the story of Annie. Annie marked each month of abstinence from bulimic behaviors with a piece of artwork. One month, she brought in a dramatic drawing of an eagle landing on a dinner plate, a fork and knife in its talons (Figure 0.1). For Annie the eagle represented aspects of her recovery. Initially, eating was frighteningly like a fierce eagle, but as she progressed, eating became a powerful eagle-like force for recovery. Her transformation and the hard work, bravery, and dedication of all our patients who struggle to recover from eating disorders are the inspiration behind the writing of this book.

Marcia Herrin, EdD, MPH, RD, LD
Maria Larkin, MEd, RD, LD

FIGURE 0.1 A Reproduction of Annie's Drawing of an Eagle Landing on a Dinner plate.

PART I

Nutrition Counseling

1

CLINICAL FEATURES OF EATING DISORDERS

Introduction

Eating disorders (EDs) are biologically-based mental disorders classified and defined in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association, 2000). This chapter relies on the DSM-IV and the soon to be published next edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5). We conclude this chapter with a discussion of the most significant of the proposed DSM-5 criteria for EDs.

The diagnostic criteria for anorexia nervosa (AN), bulimia nervosa (BN), binge-eating disorder (BED), and eating disorder not otherwise specified (EDNOS) are based on psychological, behavioral, and physiological characteristics with the same core features: overvaluation of shape and weight and serious disturbances in eating behaviors. The severity of EDs increases over time, and individuals with EDs have significantly elevated mortality rates, especially those with AN due to associated medical complications or suicide (Arcelus, Mitchell, Wales, & Nielsen, 2011). Like other psychiatric illnesses, EDs have a strong heritability factor, as emerging studies identify chromosomal regions and genes for AN. Twin studies confirm that approximately 50–70% of risk factors for ED are heritable. A family history of an ED, obesity, or anxiety, and depression increases risk. Individuals with a diagnosis of anxiety, depression, post-traumatic stress disorder (PTSD), obsessive–compulsive disorder (OCD), and attention deficit/hyperactivity disorder (ADHD) also have an increased risk (Mehler & Andersen, 2010). (See Chapter 3: The Process of Counseling for more information on these disorders, pp. 59–78.)

The highest onset of AN and BN is during adolescence in westernized societies which value thinness. BED occurs into adulthood, with a growing trend among overweight and obese middle-aged women (Ozier & Henry, 2011; Walsh, 2011).

A recent study by Marques et al. (2011) compared the prevalence of EDs across all major ethnic minority groups in the United States. The study confirmed that the lifetime prevalence of AN and BED is similar for all major ethnicities (American African, Asian, Hispanic, and non-Hispanic Whites). BN, however, has higher prevalence rates among Latinos and African Americans. A number of studies indicate that bisexual and gay men are at a higher risk for EDs than heterosexual men. One population-based study (Feldman & Meyer, 2007) found that 15% of gay or bisexual men had at some time suffered from disordered eating, AN, BN or BED compared to less than 5% of heterosexual men. These researchers reported no differences in rates of ED between lesbian, bisexual women, and heterosexual women. Nevertheless, studies of the prevalence of EDs show them to be relatively uncommon, as illustrated in Table 1.1, in part because they are frequently underreported and undertreated.

In both AN and BN, alterations in brain structure and function as well endocrine abnormalities contribute to many of the clinical features of EDs we describe in this chapter. These clinical features are not only disabling but come with significant medical and social costs. For example, women with AN have higher rates of pregnancy complications than women without an ED, and have higher healthcare utilization than those with other forms of mental illness (Klump, Bulik, Kaye, Treasure, & Tyson, 2009). It is important to recognize the unique eating-disordered characteristics of special populations such as pregnant women. Table 1.2 describes these populations and clinical features. In addition two tables are included to define the classic clinical features of AN, BN, and BED. We further elaborate on the major complications of EDs we find useful for the assessment and treatment (including making medical referrals) as well as for the purposes of psycho-education. We end this chapter by introducing the proposed DSM-5 criteria for EDs and comparing it to the DSM-IV.

Features of Anorexia Nervosa

In western societies, AN afflicts from one-third to 3% of women and is the third most prevalent chronic disease afflicting adolescent girls. Males and older women are affected, but at lower rates. There are two subtypes of AN: restricting and binge/purge. The binge/purge subtype is distinguished from BN by low weight.

TABLE 1.1 Lifetime Prevalence of Eating Disorders in the United States

	Men	*Women*
AN	.3%	.9%
BN	.5%	1.5%
BED	2%	3.5%

Note. Adapted from "Position of the American Dietetic Association: Nutrition Intervention in the Treatment of Eating Disorders," by Amy D. Ozier and Beverly W. Henry, 2011, *Journal of the American Dietetic Association*, 111, p. 1237. Copyright 2011 by American Dietetic Association. Adapted with permission.

TABLE 1.2 Characteristics of Special Populations at Risk for Eating Disorders

Risk factor	*Medical or lay term★*	*Characteristics*
Type I diabetes mellitus (DM)	Diabulemia★ Diabetic EDO patients: (Mathieu, 2008)	Intentionally omit insulin in an attempt to lose weight or compensate for a binge. Higher rates of premature diabetic complications and hemoglobin A1c levels. Hyperglycemia results from omission of insulin or binge eating. Most common in BN.
Type II DM		Most common in EDNOS and BED. ED often is undetected because symptoms of DM and ED show similar features.
Athletics	Female athlete triad (FAT)	FAT: Low energy availability, menstrual irregularities, and low bone density.
	Anorexia athletica★ (AA)	AA: Female athletes who exercise beyond what is necessary for good health, with extreme focus on weight and diet. Exercise becomes a burden, does not satisfy performance goals, and takes up too much time.
Pregnancy	Pregorexia★	Fear of normal expected weight gain during pregnancy results in reducing calories and increasing exercise.
Food allergies or intolerances	Gluten-free or lactose-free diets	Food avoidance, food fears.
Middle age and menopause		Normative age-related changes and biological shifts may increase body dissatisfaction and disordered eating. Evidence is unclear whether body mass index (BMI), age, or menopause are responsible (Slevec & Tiggeman, 2011).
College students	Drunkorexia★, beer bulimia★	Calorie restriction prior to consumption of alcohol to avoid weight gain. Purging after drinking to get rid of calories.
Vegetarian and vegans		High prevalence of veganism and vegetarianism in disordered-eating and ED populations (Sullivan & Damani, 2000).
Interest in health and nutrition	Orthorexia★	Obsession with healthy eating versus the desire to be thin. Anxiety with eating that impacts quality of life.
Obesity		BED leads to obesity; BED is associated with dieting in the obese; 30% BED in obese patients (Freitas, Lopes, Appolinario, & Coutinho, 2006); 30–60% of BED patients are obese (de Zwann, 2010; Dingemans & van Furth, 2012).
Hypoglycemia		Hypoglycemia can indicate undereating or overexercise.

TABLE 1.2 (continued)

Risk factor	*Medical or lay term*★	*Characteristics*
Ematophobia		Avoids certain foods due to fear of choking or vomiting
Picky eating	Food avoidance emotional disorder (FAED) (Bravender et al., 2007).	Avoidance of food to a marked degree in the absence of the characteristic psychopathology of eating disorders.

Many individuals with the restricting subtype develop purging or binge-eating symptoms, with more than 50% developing bulimic behaviors (Mehler & Andersen, 2010).

A markedly low weight is a unique feature of AN and sets the stage for the clinical complications we discuss below. Individuals with AN characteristically restrict food intake due to an intense fear of weight gain, resulting in an extremely low body weight and symptoms of starvation. They may count calories, weigh themselves obsessively, have body distortion, and see their bodies as bigger than they actually are. Individuals with AN typically tend to be sensitive, perfectionist, and self-critical. We have a number of AN patients who are writers and poets; those who are students do well academically, but feel socially isolated.

Factors that increase an individual's risk for developing AN consist of teasing or criticism about body size and shape, especially from peers, parents, significant others, or coaches. Other risk factors include: unintentional weight loss, military or sport weight standards, obesity at menarche, trauma or abuse, and a desire to improve athletic performance. The "female athlete triad" is characterized by disordered eating, menstrual irregularity, and loss of bone density, and is seen in competitive female athletes in sports such as running, ballet dancing, cycling and swimming (Ozier & Henry, 2011). (See Chapter 12: Managing Exercise, pp. 260–276.)

Clinical Complications of Anorexia Nervosa

Cardiac Function

Underweight AN patients are at risk for cardiovascular and neurological repercussions. The most serious of these are the electrocardiographic abnormalities that can signal the risk of sudden death. AN patients may experience chest pain indicative of mitral value prolapse as the heart reduces in size. Mitral value prolapse occurs in 30–50% of patients with severe AN (Mehler & Andersen, 2010, pp. 131–132). This condition is not usually medically dangerous, but as a precaution, patients should be medically assessed for signs of heart failure.

When caloric intake is extremely inadequate, the body adapts by losing cardiac muscle in an attempt to preserve other muscle. As a result, blood pressure drops and the likelihood of cardiac failure increases. Patients with heart rates below 40 beats/minute should be under close medical supervision. The following symptoms indicate cardiac impairment: weakness, dizziness, cognitive impairment, overall fatigue, and light-headedness on standing or changing positions (e.g., moving from lying down to sitting; Mehler & Andersen, 2010, pp. 131–132). If our patients complain of these symptoms, we express concern and inform the patient's medical practitioner immediately. If the patient also has low blood pressure and feels light-headed, he or she might need hospitalization. With refeeding and weight restoration, cardiac structure and function as well as exercise capacity return to normal without long-term consequences (Mont et al., 2003; Mehler & Andersen, 2010, p. 133).

Hormonal Changes

Many of the physical signs and symptoms in AN are the result of endocrine dysfunction (i.e., hypothalamic–pituitary dysregulation, hypothalamic amenorrhea, and hypothalamic–pituitary adrenal axis dysregulation). For example, sensitivity to cold, hypotension, and low heart rate indicate hypothyroidism. These symptoms are ameliorated with weight restoration and without the need for thyroid medications (Mehler & Andersen, 2010). Diminished libido may be the result of reduced androgen levels and, in men, low testosterone.

In AN, amenorrhea, also called hypothalamic or functional amenorrhea, occurs in some but not all patients, as there is a highly variable "individual susceptibility of the gonadal axis to undernutrition" (Miller, 2011, p. 2939). Further, amenorrhea may be masked by the use of prescription medications used for birth control. Hypothalamic amenorrhea reflects a state of estrogen deficiency, which can occur with dieting, with excess exercise, and with or without significant weight loss (Mehler, Cleary, & Gaudiani, 2011). Although no longer recommended as a diagnostic test, estrogen deficiency is sometimes assessed by a progestin challenge test. Withdrawal bleeding within 10 days of the challenge indicates that amenorrhea is caused by an estrogen deficiency. A lack of withdrawal bleeding, on the other hand, demonstrates a more profound estrogen deficiency or possibly an unrelated medical problem. It is now well accepted that the prescription of estrogen–progesterone medications do not result in clinical improvements in bone mineral density (BMD; Mehler & Andersen, 2010, p. 143). Of note, a person with amenorrhea can become pregnant, but with active AN, ovulation is unlikely. We refer the reader to as an excellent general reference, Mehler and Andersen's (2010) book, *Eating Disorders: A Guide to Medical Care and Complications,* for detailed guidelines for testing and treatment of hormone abnormalities secondary to EDs. Table 1.3 outlines the general endocrine changes seen in AN (Mehler & Andersen, 2010, p. 163; Miller, 2011, pp. 2939–2949, Table 3).

TABLE 1.3 Changes in Hormone Levels in Anorexia Nervosa

Hormone	*Change in hormone levels*
Gonadal hormones	Decrease in estrogen (females) and testosterone (males). Decrease in luteinizing hormone (LH) pulsatility.
Adrenal	Increase in plasma cortisol.
Growth hormones	Decrease in insulin-like growth factor-1 (IGF-1). Increased or normal fasting growth hormone (GH). Decrease in serum growth-hormone-binding protein (GHBP).
Appetite-regulating hormones	Decrease in leptin. Increase in grehlin. Increase in peptide YY (PYY).
Cholesterol	May be increased.
Glucose	Decrease in fasting blood sugar.
Thyroid hormones	Low or low normal thyroxine (T4) and triiodothyronine (T3). Normal thyroid stimulating hormone (TSH). Increase in reverse T3.

Bone Health

The endocrine alterations described above contribute to low BMD in AN (Miller, 2011). As a result, osteoporosis, characterized by loss of bone density and deterioration of the structure of bone, is common. Although the research is mixed, it appears possible that some individuals may recover from osteoporosis, while others will have only partial improvement in BMD despite resumption of hormone levels and weight restoration (Olmos et al., 2010). In both osteopenia and osteoporosis, bones become less dense and more fragile, but rarely is there any pain.

Achieving peak bone mass is important for preventing osteoporosis. On average, 90% of peak bone mass is acquired by the age of 19 years (Bogunovic, Doyle, & Vogiatzi, 2009). As a consequence, adolescence marks a critical period on which a lifetime of bone health is dependent. Failure to achieve peak bone mass is associated with an increased risk of osteoporosis and fracture in adulthood (Bogunovic et al., 2009). Adolescent girls with AN do not experience the usual linear increase in bone mass during puberty (Miller, 2011).

In addition, loss of BMD is thought to occur quickly and early in the course of AN. Significant losses occur in as little as six months of amenorrhea-associated with AN (Mehler & MacKenzie, 2009). But, conventional dual energy x-ray (DEXA) scans do not detect early loss of bone volume and trabecular thickness in adolescents (Miller, 2011). Both duration of amenorrhea and body mass index (BMI) predict bone density. Mehler et al. (2011) predict that BMI less than 15 and six months of amenorrhea are prognostic of significant loss of BMD.

Osteopenia occurs in 92% and osteoporosis in 40% of women with AN (Mehler, 2011). More than 50% of AN patients present with osteopenia at the time of diagnosis (Munoz & Argente, 2002). In normal populations, osteopenia is infrequently diagnosed before the age 50, at which point most people begin to lose

0.5% of BMD every year. The type of bone loss associated with AN is unique and is considered more damaging than that induced by menopause (Mehler et al., 2011). Males with AN often have more severe bone loss than females with a similar course of illness (Mehler, Sabel, Watson, & Andersen, 2008).

Individuals with osteoporosis are at greater risk for fracture even with a small trauma and have a higher lifetime risk for future fractures. The bones most affected are the hip, spine, and wrist. Advanced osteoporosis can lead to loss of height, stooped posture, humpback, and severe pain if fractures occur. Bone fractures occur in more than 40% of individuals with long-term AN. Biller's classic study found AN lasting 5 years leads to an annual fracture rate seven times greater than that of healthy women (Biller, Saxe, Herzog, Rosenthal, Holzman, & Klibanski, 1989).

The etiology of bone loss in AN is multifactorial. Low body weight, depleted fat cell mass (less than 10%), and the deficiency of estrogen and progesterone that occurs with amenorrhea all increase bone resorption and decrease bone formation. Young women with estrogen deficiency may lose bone mass at a rate of 3–5%/year. Other hormonal disturbances involved in bone formation are affected in AN, namely, low testosterone levels, low insulin-like growth factor-1 (IGF-1) levels and excess cortisol secretion (Mehler et al., 2011). Increased levels of ghrelin and Y peptide along with changes in leptin and endocannabinoid levels also affect bone formation and resorption (Horst-Sikorska & Ignaszak-Szczepaniak, 2011; Mehler et al., 2011). Although both recovery of menses and normalized weight improve BMD, the strongest indicator is normalization of menses (Miller, 2011).

There are currently no evidenced-based research studies for the treatment of osteoporosis in AN. The results of trials with hormonal therapies (estrogen and contraceptives) and bisphosphonates have been either unclear or the therapies have been found to be ineffective these patients. Most studies use adult subjects, but data from adult studies cannot easily be applied to adolescents, who are at greatest risk of osteoporosis. Individuals, who take contraceptive hormones or have had withdrawal bleeding with a progestin challenge, often have a false sense of recovery and strengthened denial of their illness.

Gastrointestinal Problems

Gastrointestinal symptoms are very common in individuals with AN and are often misdiagnosed as irritable bowel syndrome rather than recognized as symptomatic of an ED. Some gastrointestinal disturbances may cause permanent damage to the gut even after the ED has resolved. Gastrointestinal complications in AN patients are known to impede treatment and recovery by perpetuating and reinforcing disordered eating behaviors.

Gastrointestinal symptoms in both AN and BN individuals are considered by most medical experts to be a direct result of the hypometabolic state of chronic

starvation in addition to the gastrointestinal disturbances caused by binging and purging. For instance, delayed gastric emptying is well documented in AN. Researchers speculate starvation and protein malnutrition causes smooth muscle atrophy in the gastrointestinal tract, affecting gastric emptying and intestinal transit time. Others hypothesize that gastric dysrhythmias occur because of AN, contributing to delays in gastric emptying. Symptoms of delayed gastric emptying include: early satiety, bloating, and abdominal distention. These symptoms may make patients "feel fat" and curtail food intake further, or trigger purging or laxative abuse. Bloating is often the most severe for AN individuals who consume vegetarian diets high in fiber.

Constipation is another distressing gastrointestinal complication in which the underlying pathophysiological mechanisms remain unclear. Constipation that results from restrictive eating may stimulate rectal distention followed by a reflex mechanism that inhibits gastric emptying. What is certain is that AN slows gut transit time by approximately 50%. BN patients have significant delays as well (Benini, Todesco, Dalle Grave, Deiorio, Salandini, & Vantini, 2004). Tricylic antidepressants, in particular, and other medications used to treat co-morbid diseases may also contribute to constipation.

Malnutrition or purging leads to hypokalemia (low potassium) and hypomagnesemia (low magnesium). These deficiencies slow colonic transit time by interfering with nerve function in the bowel wall. Laxatives used as a solution for bloating and constipation cause electrolyte imbalances, pancreatic damage, interstitial nephropathy, and delayed intestinal motility. Salivary gland hypertrophy, typical of BN, has also been known to occur in malnourished anorexics who do not binge or purge. It may take several months for glands to normalize after weight restoration. Salivary gland hypertrophy, bloating, abdominal distention, and constipation contribute to the "I feel fat" lament we often hear from our underweight patients.

AN patients are at risk for developing yet another gastrointestinal complication known as superior mesenteric artery syndrome (SMA). Loss of normal intra-abdominal fat allows compression of the third portion of the duodenum by the overlying superior mesenteric artery. Abdominal pain made worse by eating is a major symptom of SMA. SMA resolves with weight gain. Although relatively rare in eating-disordered patients, pancreatitis can and does occur when weight loss is sudden or patients are rapidly refed. To further add to the risk, pancreatitis can be masked by the elevated serum amylase levels found in approximately 10–20% of patients who binge and vomit frequently (Mehler & Andersen, 2010).

Brain Health

Cerebral atrophy has been described in patients with AN and in some patients with BN. Patients with a history of adolescent AN show significant deficits in cognitive, emotional, and social function compared to healthy controls. These deficits

resemble those of mood and anxiety disorders and interfere with decision-making abilities. Patients with absent or irregular menses despite weight recovery also show cognitive impairments (Addolorato, Taranto, Capristo, & Gasbarrini, 1998; Chui et al., 2008; Krieg, Backmund, & Pirke, 1987). The impairments in social cognition affect interpersonal relationships, especially during the acute phases of AN, but can remain after recovery (Klump et al., 2009). It is postulated that the body is not able to protect the brain at low weights as the brain requires approximately 20% of the body's total energy expenditure to function (Treasure, Wack, & Roberts, 2008).

There is no debate that AN is associated with significant loss of cerebral gray and white matter, cerebral atrophy, and enlarged ventricles. Gray matter is a type of neural tissue associated with intelligence, concentration, emotions, personality, and creativity. White matter consists of the nerve fibers that connect various parts of the brain and determines how fast the brain can process information. The precuneus is an area of gray matter essential in self-processing, reflections upon self, and other aspects of consciousness and memory retrieval. Changes in the precuneus of AN patients is currently under study since it is the brain tissue most affected by weight loss. Recent research shows that loss of volume in the precuneus region is associated with distorted body image and it appears not to respond to weight recovery (Sachdev, Mondraty, Wen, & Gulliford, 2008). The severity of cerebral changes appears to be directly related to the amount of weight loss. Similar but less pronounced structural brain abnormalities have been found in BN.

With weight restoration, partial restoration of brain tissue has been documented. A number of studies show brain restoration during active weight gain (Joos et al., 2011). In a series of German studies, brain-tissue volume increased in weight-recovered patients by 25% compared to the brain volume measured at patients' lowest weights (Schlegel & Kretzschmar, 1997). Yet, brain-tissue deficits, especially of gray matter, have been documented 5 years after recovery (Mühlau et al., 2007). Early consensus is that lowest lifetime BMI is the critical variable that predicts irreversible brain changes. There is ongoing debate about whether these brain changes associated with eating disorders are the cause or the consequence of restricted eating and malnutrition (Kaye, 2008).

We recently updated our knowledge about seizures after several patients experienced mild to serious convulsions. We learned that seizures in ED patients occur for the same reasons as they do in individuals without EDs, and that is as a reaction to a triggering or stress factor. In general, everyone has a threshold that, if breached, leads to a seizure. Seizure thresholds are lowered by low blood sugar, restrictive eating, purging, excessive exercise, elevated cortisol levels, decreased blood flow to the brain, and lack of sleep. In epilepsy, the unremittingly low seizure threshold is due to the brain abnormalities associated with the epilepsy itself. Angela, a low-weight high-school-aged patient, was diagnosed with epilepsy after several severe seizures led to ambulance trips to the emergency room. Even treated with epilepsy medications, Angela continued to have seizures. Angela's doctors explained that her low weight and restricted

eating lowered the blood glucose levels in her brain, leaving her at high risk for another seizure. Angela was told that, once she gained weight, her anti-seizure medication would keep her from having seizures and she could expect to safely go off to college in the fall.

Protein Deficiency

The semi-starvation of AN leads to protein depletion and loss of lean body mass. Adequate protein is necessary for maintenance of the immune system, bones, tendons, connective tissues, oxygen transport, and other essential functions. Low electrolyte levels, particularly, hyponatremia (low sodium) and hypokalemia, can occur as a result of protein malnutrition, which lowers blood urea nutrition and, in turn, alters renal glomerular filtration pressure, impairing sodium reabsorption (Bahia, Chu, & Mehler, 2011). Loss of lean body mass is associated with reduced overall metabolism, low body temperature, and depletion of brain, heart, and other organ tissues. Lean body mass losses of up to 40% have been documented in men in the well-known Keys Minnesota starvation studies (Keys, Brozek, Henschel, Mickelson, & Taylor, 1950). (See Box 1.1 for a summary of the Keys starvation studies.) More recent studies in both male and female adolescents, found 10–15% less lean body mass in adolescents with AN compared to healthy controls; male patients had greater losses than females (Haas et al., 2009; Misra et al., 2008). Haas' study (Haas et al., 2009) of an AN patient population found that one-third were severely protein depleted. Protein-depleted adolescents have linear growth retardation and chronic morbidity (Hass et al., 2009). Haas et al. (2009) conclude that a BMI less than 16.5 in female adolescents indicates protein depletion. In adults with AN, low protein status is associated with chronic morbidity.

BOX 1.1 KEYS' STUDY ON STARVATION

Familiarity with the Keys' classic study of starved, young, male conscientious objectors during World War II provides ample data on the effect of starvation on physical and mental functioning and food behavior (Keys et al., 1950). The Keys' study is often used to illustrate that most symptoms associated with AN and BN are the result of starvation. Keys' subjects entered the study in good physical and psychological health. After 6 months of consuming one-half of their normal food intake, the subjects developed labile mood, cognitive dysfunction, poor concentration, social withdrawal, obsessive, ritualized eating behaviors, insatiable appetites, binge eating, food cravings, apathy, anxiety, depression, irritability, and frequent outbursts of anger. These young men

became negative, argumentative, withdrawn, and exhibited low self-esteem and relationship problems. Physical changes included: hair loss, gastrointestinal discomfort, edema, dizziness, headaches, increased fatigue, cold intolerance, lowered body temperature, decreased heart and metabolic rates, and decreased need for sleep. Cognitive changes included: impaired concentration, comprehension, and alertness. Food became the principal topic of conversation, reading, and daydreams. Food behaviors changed. Of note were an increased interest in cooking, inordinate amount of time spent meal planning, food hoarding, increased gum chewing, increased consumption of liquids, and eventual bingeing. A very readable summary of Keys' study is given in Garner and Garfinkel's *Handbook of Treatment for Eating Disorders* (2nd ed.; Garner, 1997, pp. 153–161).

Total body nitrogen (the standard indicator of body protein) improves with weight gain. Patients restore protein levels and regain lean body mass during the early stages of recovery, and regain fat mass during the later stages of recovery. Body temperature normalizes with gains in muscle mass. Distribution of lean body mass and adipose tissue match controls after a year of maintaining a normal weight (Mayer et al., 2009). During the acute phase of weight restoration, adipose tissue is preferentially deposited centrally in the abdomen and trunk.

Hair, Nails, and Skin

Patients often complain of bad nails and of hair loss. The most vocal complaint is for the noticeable loss of hair. Telogen effluvium, common in AN, is the name for temporary shedding of dead hair that occurs when the body suffers a shock such as weight loss or a high fever (Strumia, 2009). Paradoxically, dramatic hair loss usually does not occur until, in recovery, hair regrowth pushes out the dead hair. When this happens, we point out the sign of recovery that we call "recovery bangs," the fine fringe of new hair that can be seen along the forehead hairline. Weakened nails and fragile, dry hair are generally related to general malnutrition, particularly of calories, protein and zinc (Kim et al., 2010).

Hypercarotenemia (high carotene levels associated with yellow-orange skin) is seen frequently in AN. Carotene processing is slowed down in AN, especially in children, adolescents, and young adults, who already metabolize carotene more slowly than adults ("Hypercarotenemia," 2002). Overconsumption of fruits and vegetables contributes to hypercarotenemia. The unique yellow hue of hypercarotenemia is most obvious on the palms, cuticles and soles of the feet (Tung, Drage, & Ghosh, 2006). Birmingham (2012) recommends that counselors compare the color of their own palms to those of the patients to help them more fully grasp that they have a medically serious condition. Hypercarotenemia, in and

of itself, is not dangerous, but it does indicate slowed metabolic processes and an unbalanced diet. Birmingham also uses this opportunity to emphasize that, in addition, AN has robbed patients of the ability to maintain a healthy body temperature, as he predicts patients' hands will be either markedly cold or warm (Birmingham, 2012).

Metabolism

Metabolism is the sum of all cellular activities necessary to sustain life. The calories needed to maintain metabolism plus the calories expended in physical activity account for a patient's total caloric needs. The caloric cost of metabolism is affected by height (taller people use more energy to maintain body temperature than do shorter people), age (younger people have faster metabolisms than older people), and muscle mass (muscles require more energy to maintain than fat tissues). Undereating, fasting, starvation, and malnutrition lower an individual's metabolic needs as the body tries to compensate for the potentially life-threatening consequences of a low caloric intake. Normally, exercise increases metabolic rate. But when caloric intake is inadequate, the body lowers metabolic rate to conserve energy despite regular exercise. Depressed metabolic rates have been measured in AN and BN. In AN, this is no doubt due to the cumulative effect of loss of muscle mass and decreased caloric intake. Signs of lowered metabolic rate in anorexic patients are bradycardia (slow heart rate), dry skin, brittle hair, constipation, cold intolerance, and fatigue. Over time, improved calorie intake and weight gain normalize metabolic rate. It is not clear why metabolic rates measure low in BN, besides the fact that BN patients also restrict caloric intake.

Blood Chemistry

We feel it is important for nutrition counselors to be aware of signs and blood chemistry findings that indicate problems (Box 1.2). The Academy of Eating Disorders' Critical Points for Early Recognition and Medical Risk Management in the Care of Individuals with Eating Disorders (2011, pp. 9–11) provides guidance for interpreting laboratory values in ED patients.

BOX 1.2 THE EFFECT OF EATING DISORDERED BEHAVIORS ON LABORATORY VALUES

- Glucose: ↓(poor nutrition), ↑(insulin omission).
- Sodium: ↓(water loading or laxatives).
- Potassium: ↓(vomiting, laxatives, diuretics, refeeding).
- Chloride: ↓(vomiting), ↑(laxatives).
- Blood bicarbonate: ↑(vomiting), ↓(laxatives).

- Blood urea nitrogen: ↑(dehydration).
- Creatinine: ↑(dehydration, renal dysfunction), ↓(poor muscle mass). "Normal" may be "relatively elevated" given low muscle mass.
- Calcium: slightly ↓(poor nutrition at the expense of bone).
- Phosphate: ↓(poor nutrition or refeeding).
- Magnesium: ↓(poor nutrition, laxatives, refeeding).
- Total protein and albumin: ↑(in early malnutrition at the expense of muscle mass), ↓(in later malnutrition).
- Total bilirubin: ↑(liver dysfunction), ↓(poor red blood cell (RBC) mass).
- Aspartate aminotransaminase (AST), alanine aminotransaminase (AST): ↑(liver dysfunction).
- Amylase: ↑(vomiting, pancreatitis).
- Lipase: ↑(pancreatitis).

Note. Comprehensive serum metabolic profile and important electrolytes and enzymes. Reprinted from *Eating Disorders Guide to Medical Management. Critical Points for Early Recognition and Medical Risk Management in the Care of Individuals with Eating Disorders*, by The Medical Care Standards Task Force of the Academy of Eating Disorders, co-chaired by O. Bermudez and M. Warren. Academy of Eating Disorders. Copyright 2011 by Academy of Eating Disorders. Reprinted with permission.

Emotional and Cognitive Changes

AN patients present with a variety of psychological concerns, such as fear of maturation, anxiety, depression, low self-esteem, trauma, and interpersonal problems. AN provides an identity that is the embodiment of the value of will-power and self-control, which once well-established has a self-reinforcing life of its own (Andersen, 2006; Fairburn, 2008). The limiting of the type and amount of food that is characteristic of AN becomes an effective coping mechanism.

ED patients without a primary history of OCD commonly have OCD-like tendencies (Box 1.3). The difference in these ED patients is that their obsessions and compulsions are limited to food, weight, or body image, and these behaviors decrease with adequate food intake and weight restoration. These patients believe their obsessions and compulsions to be rational and necessary, and not obtrusive or senseless. Patients often engage in compulsive behaviors such as cutting food into small pieces, eating only cold foods, using strong non-caloric condiments such as mustard, pepper, and vinegar, or drinking large amounts of non-caloric liquids. They may acknowledge a new interest in cooking, recipes, and food shopping, though they rarely eat what they purchase or prepare. It is not unusual for ED patients to be employed in food-related jobs or even to feel compelled to "shoplift" food. A surprising number remain perplexed about why they engage in these behaviors and are not able to relate them to their ED. They may

express relief to know that their behaviors are characteristic of an ED, and that the behaviors usually resolve as their ED resolves. Besides EDs, OCD-like tendencies also occur with other DSM diagnoses such as psychosis, substance abuse, and obsessive–compulsive personality disorder. True OCD cannot be diagnosed in a patient with ED until weight is normalized.

BOX 1.3 OBSESSIONS AND COMPULSIONS ASSOCIATED WITH EATING DISORDERS

A. Rituals before, during, and after meals.
B. Rigid schedules and plans for meals or exercise.
C. Researching food and nutrition facts.
D. Excessive calorie counting.
E. Compulsive exercise or other behaviors to burn calories.
F. Perfectionism:

- striving for the perfect body, shape or weight
- exclusively eating "good" foods
- exercising until it feels "just right"
- criticizing lazy behaviors
- concern over changes in routines or meals
- striving for perfection in school, work, and relationships.

Note. Reprinted from "Managing OCD in severe eating disorders ," paper presented at the annual meeting of MultiService Eating Disorders Association (MEDA), Needham, MA, by S. D. Tsao, May 2009. Reprinted with permission.

As of this writing, the typical ED patient avoids red meat, foods with fat and sugar, and carbohydrate-containing foods. Which foods are viewed as either "good" or "bad" or "healthy" or "unhealthy" depends on the latest popular diet or nutrition theory. ED patients tend to believe, "Maybe others can eat fat or meat and not get fat, but not me; my metabolism is just different." Generally, patients progressively limit their intake of food over the duration of their eating disorder. First, snacks are eliminated, then breakfast, followed by lunch. Patients who live with others may continue to eat near-normal dinners to avoid concerning family members. Some patients "justify" eating in the evening by limiting caloric intake during the day.

Anxiety and depression are two major co-morbid psychiatric disorders in AN. In women with AN, overnight blood cortisol levels are positively associated with severity of depression and anxiety symptoms. Depression is observed in 50–75% of AN patients (Klump et al., 2009). Treatment can be complicated by personality changes caused by chronic undernutrition, such as irritability, sullenness, dependency, obsessiveness, compulsivity, and passive–aggressive behaviors. Although these changes can persist after recovery, the majority improve with weight resto-

ration or cessation of binging and purging (Klump et al., 2009). Certain personality traits, including perfectionism and neuroticism, are also apparent before and during the course of the illness (Miller, 2011). Suicide and other self-harming behaviors are highly prevalent in patients with EDs. Twenty to 40% of deaths in AN are attributed to increased rates of suicide (Guillaume, 2011). Suicide rates are 40-fold higher in AN than in the general population (Preti, Rocchi, Sisti, Camboni, & Miotto, 2011). The research is less clear about rates of suicide in BN, but a 2009 longitudinal assessment of mortality over 8–25 years found nearly similar rates for BN and AN (Crow et al., 2009). Overall there has been a decline in suicide rates in ED in recent years. There is some evidence that this decline could be attributed to improved quality of and access to ED treatment (Preti et al., 2011). Tables 1.4 and 1.5 summarize the physiological characteristics, medical complications, and behavioral, emotional, and cognitive characteristics of AN.

TABLE 1.4 Physiological Characteristics and Medical Complications of Anorexia Nervosa

Body weight	Significantly low body weight. Intense fear of gaining weight or becoming fat, or persistent behavior that interferes with weight gain. 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 persistent lack of recognition of the seriousness of the current low body weight.
Brain and neurological problems	Decreased gray-matter volume. Increased fluid in brain. Dizziness, faintness, headaches, seizures. Disordered thinking. Numbness or odd nerve sensations in the hands or feet (peripheral neuropathy).
Heart problems	Dangerous heart rhythms (arrhythmias). Slowed heart rate below 60 beats/minute (bradycardia). Fatigue. Low blood pressure, electrolyte imbalances, dehydration. Weakened heart muscle, reduced size of heart, heart attack, heart failure. Death.
Blood problems	Low blood iron (anemia). Low vitamin B12 (pernicious anemia). Potentially fatal bone marrow problems (pancytopenia). Increased risk of infection due to low white blood cells (leucopenia).
Kidney problems	Increased urination.
Liver problems	High blood levels of liver enzymes causing liver damage.
Bone problems	Low bone mineral density (osteopenia and osteoporosis). Increased risk of stress fractures.
Digestive problems	Bloating, abdominal pain, constipation. Uncomfortable fullness.
Hormonal problems	Decreased reproductive hormones, including estrogen and dehydroepiandrosterone (DHEA, a male hormone). Increased stress hormones. Decreased thyroid and growth hormones. Retarded height growth in children and adolescents. Irregular or absent menstruation (amenorrhea).
Reproductive problems	Infertility. Higher risk for complications, miscarriage, cesarean section, low birth weight, birth defects, post-partum depression. Poor success with fertility treatments.

TABLE 1.4 (continued)

Immune problems	Lowered resistance to infection.
Muscular problems	Muscular weakness. Loss of muscle tissue.
Emotional problems	Difficulty concentrating. Increase in "bad moods."
Sleeping problems	Insomnia and early morning awakening. Sleep disturbances.
Hair and skin problems	Brittle, thinning hair on scalp. Thinning of hair or hair loss on scalp. Increased downy hair growth (lanugo) on face, neck, arms, back, legs. Dry skin. Blotchy or yellowed skin, especially on palms (hypercarotenemia). Brittle nails.
Body organ failure	Organs simply fail in long-term AN. Death.

TABLE 1.5 Behavioral, emotional, and cognitive characteristics of anorexia nervosa

Restrictive behaviors	Excessive dieting, food control, fasting. Avoidance of water or excessive water intake. Extreme interest in nutrition. Collects recipes. Likes to cook or bake; usually doesn't eat what cooks or bakes. Refuses to eat in front of others. Tension at mealtime. Fear of food. Vegan or vegetarian. Frequent weighing (multiple times a day).
Exercise behaviors	Compulsive exercising or excessive physical activity. Participation in a sport that requires thinness, e.g., gymnastics, wrestling, swimming, distance running, ballet, cross-country skiing, figure skating.
Bingeing behaviors	May eat food in secret. May binge on occasion.
Purging behaviors	May self-induce vomiting or abuse diuretics, laxatives.
Eating behavior	Calorie counting, rigid rules and schedules, unusual use of condiments, low calorie foods. Odd food rituals. Eats alone. Fear of eating in front of others. Plays games with food (e.g. cutting it into tiny pieces).
Dressing behaviors	Layering of clothes. Wearing large pants and sweaters to mask thinness.
Social behavior	Social withdrawal, physically and emotionally. Extreme focus on job or school work.
Self-destructive behavior	Slow suicide progression. Self-hatred and feeling of unworthiness. Substance abuse.
Body image problems	Intense fear of becoming fat. Distorted body image.
Perfectionist behavior	Perfectionist: thinnest, smartest, neatest. Dichotomous thinking: all or nothing, black or white.
Self-esteem	Depression and low sense of self-worth and self-esteem.
Sexuality	Decreased interest in sex.
Social behavior	Self-centered and non-social, isolation from others, irritable.
Cognitive symptoms	Difficulty thinking clearly, potential severe cognitive deficits due to malnourishment.

Features of Bulimia Nervosa

BN differs from BED in the use of a compensatory behavior following a binge such as self-induced vomiting (SIV), restricting food intake, excessive exercising, or laxative or diuretic abuse. SIV is by far the most dominant purging method. One variant of SIV is rumination, self-induced regurgitation from the stomach to the mouth of recently ingested food that is chewed and then re-swallowed. Chewing and spitting out of food is considered yet another means of purging. No purging method is effective in eliminating consumed calories. After SIV, the body retains 1,200 calories (Kaye, Weltzin, Hsu, McConaha, & Bolton, 1993). It appears that, if the binge is less than 1,200 calories, the body retains all of the consumed calories (See Chapter 11: Managing Purging for more information, pp. 246–259.) Most individuals who binge and purge eventually gain weight, though weight may stay within normal parameters.

SIV is usually accomplished by inserting fingers into the throat (Mehler, 2011). Eventually, abrasions, sores, and calluses form on several knuckles (Russell's sign) as they scrape against teeth. Several of our patients explained that this is why they always wear gloves or very long sleeves, embarrassed that someone would notice the telltale knuckles. Over time, SIV leads to a diminished gag reflex; then, feeling desperate, patients experiment with increasingly vigorous efforts to induce vomiting, often using elongated objects, such as spoons. One of Herrin's patients swallowed a toothbrush that subsequently had to be surgically removed.

The strain and force of vomiting can cause hemorrhages on the cornea (appearing as very bloodshot eyes) and petechial hemorrhages (appearing as small red dots caused by minute amounts of blood that are pushed through the skin around the eyes). While unsightly, neither are of medical significance. One of our patients suffered a more serious consequence from forcible vomiting: a retinal detachment which required laser surgery to repair.

Emotional and Cognitive Changes

Patients with BN tend to exhibit impulsivity, emotional intensity, and fluctuations in mood, but usually appear healthy and manage to maintain weight within a normal range. This, coupled with secretive behaviors, makes it relative easy to conceal BN. Some individuals suffer for years before seeking help. In BN, the cycle of binge followed by a purge is thought to demonstrate an inability to self-regulate negative emotions. Emotional states which may precipitate bingeing include: boredom, depression, disappointment, loneliness, procrastination, stress, anxiety, and anger. Bingeing may also serve as a vehicle for acting out, for disassociating, for expressing impulses, for self-soothing, and to inflict self-punishment. Psychological factors related to binge eating include low self-esteem, feelings of ineffectiveness, and extreme concerns about shape and weight. Co-morbid

illnesses common to BN include anxiety disorders, depression, substance-use disorders, and personality disorders.

Clinical Complications of Self-Induced Vomiting

Oral/dental problems become evident as early as 6 months after the onset of SIV. These include dental erosion, dental caries, periodontal disease, and cheilosis (fissuring and dry scaling along the angels of the mouth; Mehler, 2011). The erosion of the enamel that occurs on the lingual surface of maxillary teeth (perimyolysis) will look smooth and unnaturally glossy (Mehler, 2011). After several years of vomiting several times a week, most patients suffer from serious dental erosion, decalcification of tooth enamel, and, eventually, erosion of the dentin. Chronic purgers will inevitably have painful, yellow-colored, dull, jagged-edged, and obviously eroded teeth, and costly dental bills. Some bulimic patients are bothered by painful sensitivity to hot and cold foods and beverages.

Parotid and salivary gland enlargement is a common in BN, occurring in up to 50% of patients (Mehler & Andersen, 2010). Patients complain of a swollen face and puffy cheeks that may exacerbate their fear of weight gain. Because the swelling is most evident 3–6 days after a binge–purge episode, patients may not always attribute it to BN (Mehler, 2011). Although the swelling is painless and harmless, it may take several months after abstinence from purging for glands to return to normal size. Parotid hypertrophy elevates serum amylase, which can be misdiagnosed as pancreatitis unless patients self-disclose SIV.

Multiple gastrointestinal complaints (i.e., bloating, heartburn, flatulence, constipation, and stomach pain) are common among purging patients. A rare but serious complication is pneumonia when food is aspirated into the lungs during SIV. Esophagitis, erosion of the esophagus, ulcerations, and strictures are consequences of recurring vomiting. SIV may also result in hoarseness, sore throat, odynophagia (painful swallowing), dysphagia (difficulty swallowing), dyspepsia (indigestion), and hematemesis (vomiting bright red blood). Nutrition counselors should insist on a medical evaluation for patients who report blood in their vomitus as it could indicate serious esophageal or gastric tearing. It is more likely, though, that the blood originates from minute lacerations in the esophageal or gastric walls that are not medically serious. Stomach acids can burn sensitive tissues in the tongue, mouth, and throat, leaving these tissues chronically sore. Hoarseness and sore throats are the most common complaints of patients who regularly vomit. Repeated SIV causes laxity of the lower esophageal sphincter and loss of the gag reflex, resulting in spontaneous gastro-esophageal reflux (Mehler, 2011). Abdominal tenderness can result from the strain of vomiting. Recurrent strenuous vomiting can lead to Mallory–Weiss syndrome, tears in the gastro-esophageal junction, leading to bleeding or hematemesis. In Barrett's esophagus, the esophageal mucosa becomes inflamed by constant exposure to stomach acids, leading to precancerous lesions and a

significant risk of adenocarcinoma. Most patients with Barrett's esophagus have acid reflux-like symptoms, but some do not. Screening by an upper endoscopy procedure is recommended for patients with long-standing BN (Mehler, 2011). SIV may cause a serious esophageal rupture, known as Boerhaave's syndrome, a rare but life-threatening condition (overall mortality is 20%). The most significant symptom is severe chest pain.

Clinical Complications of Laxatives and Diuretics

Purging (via SIV, or laxative or diuretic abuse) that occurs twice a week or more may result in fluid or electrolyte imbalances. Undereating adds to the risk of fluid and electrolyte imbalances, as does maintaining a low body weight. We remind ourselves not to be complacent about normal electrolyte lab results in a patient who purges frequently because electrolyte levels fluctuate and recover quickly. We have seen patients have normal electrolyte results despite very frequent purging behaviors. Table 1.6 shows how various methods of purging affect specific electrolytes.

ED behaviors often cause a cycle of dehydration followed by excessive water retention. Patients with dehydration present with increased thirst, decreased urinary output, and feelings of weakness, dizziness, or lightheadedness. Dehydration stimulates the renin–angiotensin–aldosterone system to retain fluid, causing "puffiness," edema, and temporarily increasing body weight. Patients, who interpret fluid retention as weight gain, feel compelled to restrict or purge, or both, and the cycle begins again. Hypokalemia (low blood potassium) is the most common electrolyte concern in BN (normal range 3.6–5.2 mmol/L). It occurs in about 5% of patients and is a good diagnostic indicator of BN in an otherwise healthy-appearing patient. Patients with hypokalemia present with muscle weakness, fatigue, constipation, and heart palpitations (Mehler & Andersen, 2010). Hypokalemia (less than 2.5 mmol/L) can cause life-threatening cardiac arrhythmias and death in patients, regardless of their body weight (Mehler, 2011). Of note, AN patients may also be hypokalemic without engaging in SIV, due to malnutrition.

Metabolic alkalosis, an increase in serum bicarbonate, is observed in patients who self-induce vomiting or take diuretics on a regular basis. Bicarbonate levels are used to diagnose SIV. Serum levels greater than 40 mmol/L generally indicate vomiting (Mehler, 2011). Metabolic alkalosis usually is not an immediate concern, but it can progress into loss of consciousness and coma. Early symptoms include lightheadedness, nausea confusion, and muscle twitches. Hypomagnesemia can exacerbate cardiac arrhythmias. Patients with magnesium and other electrolyte losses can be asymptomatic or experience muscle weakness, cardiomyopathy, seizures, acute renal failure, convulsions, and coma. Electrolyte levels of low-weight patients and patients who purge frequently should be monitored by frequent blood and urine tests.

TABLE 1.6 Electrolyte Levels Associated with Purging

Method of purging	*Serum levels*					*Urine levels*		
	Sodium	*Potassium*	*Chloride*	*Bicarbonate*	*pH*	*Sodium*	*Potassium*	*Chloride*
Vomiting	Increased, decreased or normal	Decreased	Decreased	Increased	Increased	Decreased	Decreased	Decreased
Laxatives	Increased or normal	Decreased	Increased or decreased	Decreased or increased	Decreased or increased	Decreased	Decreased	Normal or decreased
Diuretics	Decreased or normal	Decreased	Decreased	Increased	Increased	Increased	Increased	Increased

Note. Reprinted from "Medical complications of bulimia nervosa and their treatments," by P. S. Mehler, 2011, *International Journal of Eating Disorders*, 44, p. 99. Copyright 2011 by Wiley Periodicals, Inc. Reprinted with permission.

Laxative Abuse

Both AN and BN patients may purge by abusing laxatives, despite the fact that calorie absorption takes place in the small intestine and laxatives act on the large intestine. Only an estimated 10–12% of ingested calories are lost as a result of laxative use (Bo-Linn, Santa Ana, Morawski, & Fordtran, 1983). Complications of laxative abuse include the electrolyte and fluid imbalances mentioned above and gastrointestinal problems such as, nausea, vomiting, and diarrhea. Serious consequences of chronic use of laxatives to purge are: confusion, convulsions, skeletal muscle weakness, urinary tract infections, osteomalacia, osteoporosis, rectal bleeding, finger clubbing (swelling), increased pigmentation of the skin, pancreatic damage, malabsorption, and cathartic (flaccid and dilated) colon. Acute diarrhea associated with laxative abuse can cause hyperchloremic metabolic acidosis.

Laxative abuse produces a benign, reversible brown-black discoloration of the colonic mucosa and submucosa, called melanosis coli, and a low-grade inflammation of the colon (Mehler, 2011). It is seen in approximately half of patients who take anthraquinone-based laxatives (such as those containing cascara, buckthorn, senna, senokot, or aloe vera) but does not have significant medical consequences (Mehler, 2011).

Because chronic use of laxatives disrupts intestinal peristalsis, patients often complain of alternating diarrhea and constipation that may resemble irritable bowel syndrome. Tolerances build up to the effects of stimulant laxatives, necessitating larger and larger doses for stool evacuation (Mehler, 2011). As a result, patients are at risk for laxative dependence, diarrhea, rebound constipation, fluid retention, and cathartic colon. A cathartic colon, having suffered damage to neurological innervation, becomes permanently flaccid, dilated and atonic. The loss of normal colonic peristalsis leads to problems transporting fecal material, possibly requiring partial colonic resection, colectomy, or ostomy.

When laxatives are withdrawn, the ensuing constipation and rebound peripheral edema (especially in the legs) called, pseudo-Bartter's syndrome, presents a challenge for patients. Although the edema usually resolves in 10 days or less, patients are likely to be convinced that they are gaining weight. Salt restriction and leg elevation can help, but diuretics do not (Mehler & Andersen, 2010).

Diuretic Abuse

In our practices, we rarely see patients who use prescription or over-the-counter (OTC) diuretics. Neither cause calorie malabsorption; OTC products are not even effective for water retention. Patients who abuse diuretics are usually older and have access to prescription diuretics. Abuse of prescription diuretics increases risk of dehydration, weakness, nausea, palpitations, polyuria, hematuria (blood in urine), pyuria (pus in urine), constipation, hypokalemia, cardiac conduction defects, nephropathy, and abdominal pain. Continued abuse of prescription diuretics leads to

eventual renal impairment from direct toxicity to the kidneys or from the effects of severe dehydration. When prescription diuretics are discontinued, most experience transitory "reflex" water retention. As with laxatives, salt restriction and leg elevation is the treatment of choice when weaning off diuretics.

OTC diuretics rarely cause serious medical complications, but many of them contain substantial amounts of caffeine. High doses of such diuretics, if misused, can cause headaches, trembling, and rapid heart rate, among other caffeine-related symptoms. Despite the fact that non-prescription diuretics have few side-effects, even if abused, their use should be discouraged for several reasons: first, the use of OTC diuretics may evolve into abuse of more dangerous prescription diuretics; and, second, taking diuretics of any kind perpetuates the binge–purge cycle and an unhealthy approach to weight control.

The physiological characteristics and medical complications of BN and BED are summarized in Table 1.7, and the behavioral, emotional, and cognitive characteristics of BN and BED are summarized in Table 1.8.

Features of Binge-Eating Disorder

Binge-eating disorder (BED) is classified under "eating disorder not otherwise specified" (EDNOS) in the DSM-IV but is expected to be a discrete diagnosis in DSM-5. BED has nearly two times the lifetime prevalence rate of AN and BN. Bariatric surgery patients have a 27% lifetime prevalence of BED (Kalarchian et al., 2007).

One variant of BED is a grazing pattern of bingeing. Grazers may eat relatively small amounts of food throughout the day or most of the evening, never feeling hungry or full. Though grazers may not consume a large amount of food in a discrete period of time, they do feel out of control and may end up consuming a significant amount of calories over the course of a day (Anderson, Lavender, & De Young, 2010). We have had patients ask us about the differences between compulsive eating and BED. In clinical practice, compulsive overeating is similar to grazing in that eating may continue for hours. Compulsive eaters tend to repeatedly reach for food and to feel overfull and out of control. Like other patients with BED, patients with compulsive overeating become preoccupied with eating and worry about weight, contributing to feelings of low self-esteem, guilt, and depression (Matz & Frankel, 2004).

Individuals presenting with BED may actually be suffering from night eating syndrome (NES), currently proposed as an addition to EDNOS in DSM-5. Keel Brown, Holland, and Bodell. (2012) predict that NES will likely become a provisional, and then an independent, diagnosis in a future edition of the DSM. In NES, the individual undereats or does not experience hunger during the day. Consumption of at least 25% of daily caloric intake occurs after the evening meal or after awakening to eat during the night. Individuals with NES often believe that they must eat in order to get to sleep.

TABLE 1.7 Physiological Characteristics and Medical Complications of Bulimia Nervosa and Binge-Eating Disorder

Body weight*	Weight fluctuations with body weight below, at, or above normal range due to alternating bingeing and fasting.
Slowed heart rate	Potential heart arrhythmia; irregularities related to electrolyte imbalance.
Body fluid regulation	Swollen glands, "chipmunk cheeks," puffiness around the face (burst blood vessels in eyes). Edema (swelling due to retention of body fluids). Possible impaired renal function.
Blood pressure	Normal or fluctuating blood pressure.
Body temperature	No change.
Menstrual period	Menstrual irregularities.
Body hair and skin	Finger or hand calluses. Small red dots around the eyes (petechiae) after forceful vomiting episode due to increased facial pressure.
Movement and functioning	Chemical imbalance caused by low potassium and sodium, producing dehydration, muscular fatigue, cardiac rhythm irregularities, cardiac arrest.
Imbalances	Fluid and electrolyte imbalance. Edema accompanying refeeding. Swelling of hands and feet.
Heart problems	Possible heart arrhythmias and irregularities.
Muscular symptoms	Overall muscular weakness.
Gastrointestinal disorders*	Abdominal pain, esophageal burning, heartburn or gastric dilation or rupture, non-responsive bowel. Constipation, diarrhea, stomach distress, bloating, hiatal hernia, gastrointestinal bleeding, dry mouth, nausea. Symptoms may mimic irritable bowel syndrome.
Nutritional problems	Vitamin deficiencies, hypoglycemia, diabetes mellitus.
Bone and dental	Tooth decay, gum erosion from regurgitated stomach acids. Periodontal disease. Dental caries. Mouth ulcers. Enlarged salivary glands.
Other symptoms	Headaches, tiredness, fatigue, weakness. Damage to esophagus (sore throat, horse voice, difficulty swallowing).
Disease states	Increased incidence of BN in Type 1 diabetes mellitus (especially in young women).

* Characteristic of BED as well as BN.

Features of Bingeing

BN and BED are both defined by recurrent and uncontrollable episodes of binge eating. Most ED patients, including those with AN, will engage in binge eating at some time over the course of their disorder. Binge eating is one sign that AN patients are migrating along the ED continuum from AN to BN (Meh-

TABLE 1.8 Behavioral, Emotional, and Cognitive Characteristics of Bulimia Nervosa and Binge-Eating Disorder

Bingeing*	Eating, in a discrete period of time (within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances. A sense of lack of control over eating during the episode (a feeling that one cannot stop eating or control what or how much one is eating).
Purging	Recurrent inappropriate compensatory behavior in order to prevent weight gain: self-induced vomiting; misuse of laxatives, diuretics, or other medications (diuretic, thyroid or diet pills, or use of other emetics such as syrup of ipecac); fasting; or excessive exercise.
Frequency of bingeing* and purging	At least once a week for 3 months.
Eating behavior*	Secret food foraging and hoarding, especially at night. Shoplifting or petty stealing of money to buy binge food.
Sleeping behavior*	Sleep disturbances. Often binge eating occurs at night.
Clothing and dress rituals	Obsessive–compulsive patterns such as trying on clothes multiple times a day.
Social behavior	Social irregularities: alternating withdrawal with erratic need for social contact and approval. Chaotic relationships and interactions.
Abusive behavior	Drug or alcohol abuse. Suicidal gestures or attempts. Self-hatred and self-mutilation. Feelings of self-disgust.
Body image problems*	Preoccupation with appearance and "image." Overly concerned about body weight and size.
Perfectionist behavior	Perfectionist: high performance and achievement expectations. Perfectionist inside, but sometimes chaotic outside. Façade of normalcy. Seemingly "has it together."
Self-esteem*	Low self-esteem: self-loathing, self-disgust, depression.
Sexuality	May be promiscuous or confused about sexuality—a mask for a desire to be accepted or respected.
Social behavior	Constant feeling of being out of control; vacillates between isolation and extreme need for external validation.
Cognitive symptoms	Inability to accurately identify and express feelings. Out of touch with one's feelings (e.g., anger, affection, humor). Obsessive thoughts focused on the eating-disorder cycle.

* Characteristic of BED as well as BN.

ler & Andersen, 2010). Binge eating is a distinctive behavioral pattern in which attempts to restrict eating (which may or may not result in actual dietary restriction) are interrupted by repeated episodes of binge eating. This pattern is set in motion when self-evaluation is based almost exclusively on achieving a thin body,