Eating Disorders

A Comprehensive Guide to Medical Care and Complications

4th edition

Edited by

Philip S. Mehler, MD, FAED, CEDS
& Arnold E. Andersen, MD, FAED, DLFAPA

EATING DISORDERS

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***Note to the Reader*:** This book is not meant to substitute for medical care, and treatment should not be based solely on its contents. Instead, treatment must be developed in a dialogue between the individual and his or her physician. Our book has been written to help with that dialogue.

Drug dosage: The author and publisher have made reasonable efforts to determine that the selection of drugs discussed in this text conform to the practices of the general medical community. The medications described do not necessarily have specific approval by the US Food and Drug Administration for use in the diseases for which they are recommended. In view of ongoing research, changes in governmental regulation, and the constant flow of information relating to drug therapy and drug reactions, the reader is urged to check the package insert of each drug for any change in indications and dosage and for warnings and precautions. This is particularly important when the recommended agent is a new and/or infrequently used drug.

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To our patients with eating disorders—
Your struggles embolden us to put forth efforts to seek the best care
to cure your illnesses and achieve a permanent recovery

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This fourth edition of our book is one that we are both most proud of. This pride is attributable to the affirmations we have regularly received about the scientific knowledge void this book has filled for three decades and its great value to practitioners in this field. From an ephemeral afterthought in the midnineties to this fourth edition, we have strived to cover the always-growing body of medical findings related to eating disorders along with relevant associated topics. This edition is thus considerably larger than previous ones and contains four brand-new chapters on emerging topical areas of interest, in which we strove to be comprehensive, with just the right amount of detail. In addition, we focused on the book's accessibility to promote digestible explanations of complex medical and related concepts while filling in gaps and applying the medical information herein to novel circumstance. The issues addressed in the fourth edition are far from being only of academic interest. Rather, they are of practical import in the daily battle to help patients successfully extricate from the torments of their eating disorder and regain their health.

As coeditors and coauthors for all four editions of this book, we are blessed to be highly experienced in eating disorder practice, with almost 75 combined years of practice in the field of eating disorders, both from the well of the hospital-residential treatment center and from the hallowed halls of academia. We expect and receive no just compensation for our substantial investment of time and effort. Instead, this text is our donation to a beloved profession, a labor of love. We hope and trust that the ongoing need for a comprehensive, one-volume summary of the relevant medical and related facts remains self-evident.

We are indebted to our invited and esteemed coauthors for their industry and scholarship in adding this treatise to your library. In addition to

the aforementioned new chapters, we have augmented and updated all the other ones. Medicine is a human profession practiced by humans who have dedicated their lives not only to science but also to humanity and the suffering that eating disorders exact on its patients. Said humanity in medicine has inspired all our pens.

Throughout the task of writing and editing this book, we have constantly and faithfully been assisted by long-tenured experts from the field of eating disorders, numerous colleagues, and others. We are under obligation to our mentors from the 1980s and 1990s, to our patients, to our late parents, and to our growing families for the opportunities afforded us in this sacred field.

+ + +

We would like to thank Kelly Maebane, Millie Plotkin, and Keith Stenson for their administrative, librarian, and artistic skills, with which this edition of the book would not have come to fruition. We are deeply appreciative for your superb assistance. And special thanks also to Paul, Jess, and Ladi.

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Arnold E. Andersen, MD, FAED, DLFAPA

1

Diagnosis and Treatment of the Eating Disorders Spectrum in Primary Care Medicine

Arnold E. Andersen, MD, FAED, DLFAPA

COMMON QUESTIONS

What are eating disorders? Why are they a public health concern?
What changes in the field of eating disorders have occurred during the period 2016–2020?
How do eating disorders present to physicians, other primary care providers, psychologists, teachers, and coaches?
What risk factors increase the probability of an eating disorder?
How are eating disorders diagnosed?
How have *DSM-5* and ICD-11 changed diagnostic criteria?
What are the strengths and weaknesses of *DSM-5* and ICD-11?
How might a transdiagnostic approach be helpful?
Why are eating disorders best understood as a spectrum disorder?
What is orthorexia?
Which simple screening instruments are available?
What are the most common comorbid psychiatric disorders?
How does binge eating disorder relate to bariatric surgery?
What is the natural history development of eating disorders over time? What factors maintain and perpetuate eating disorders?
What are the most overlooked groups in diagnosis of eating disorders?
How serious are eating disorders? What are the causes of death?

What are the key principles of treatment? When should a primary care clinician refer a patient to a specialist in eating disorders?
What are the criteria for hospitalization?
What role does genetics play? Epigenetics?
Is prevention possible?
How effective are internet-based and other electronic-based methods for diagnosis, treatment, prevention, and education?
How can the burden on caregivers be alleviated?
What would ideal treatment of eating disorders look like?

A good physician treats the disease; a great physician treats the person who has the disease.

Whether this maxim was written by Sir William Osler or Maimonides is less important than the message it sends: while the illness characteristics of eating disorders and their medical complications follow patterns that astute clinicians learn to recognize, a vital aspect of diagnosis and treatment is to understand the unique features of the life and personality of each person who suffers from an eating disorder. No two patients are the same in their genetic endowment, developmental trajectory, life experiences, relationships, hopes and fears, temperament, psychodynamics, comorbidities, or response to the experience of illness and treatment. Hence, an eating disorder will present in myriad variations in different individuals, and their treatment response will vary in small if not large ways despite shared diagnoses and similar methods of treatment. An understanding and appreciation of each person's uniqueness and the effect of that person's individuality on all aspects of illness will result in an optimum approach to forming a healing therapeutic relationship without sacrificing evidence-based treatments.

The goal for patients suffering from any eating disorder is that they will not only attain full remission from their eating disorder but go on to develop a life that is flourishing in all aspects. The hope is that remission of their illness will be only the first step in the return, or the achievement, of a full and vibrant life of internal harmony, caring relationships, and skilled navigation of the complex psychosocial environment.

Each chapter in this book contains two primary components:

Patient case studies: Some of the brief examples are from cases we have evaluated and/or treated anywhere from 45 years ago to the recent past. In all cases, anonymity has been preserved by altering patient characteristics or by combining two similar cases.

Patient care: This chapter and other chapters are intended for the use of medical professionals and the general public to increase their understanding of multiple aspects of the diagnosis and treatment of patients with eating disorders. They are not intended for the treatment of any individual patient except after thorough evaluation by a medical professional.

Case Study

A.H., a 25-year-old female, was referred by her local psychiatrist for treatment of severe anorexia nervosa (AN), complicated by a fractured humerus. She had developed body image distress and perception of fatness starting at age 13. She had been slightly chubby and was teased by her peers, especially about her "thunder thighs." She restricted food and lost weight from 151 pounds at 5 feet 4 inches (body mass index [BMI] of 26) to 84.9 pounds (BMI 14.6) at admission. In addition to eating fewer than 500 calories daily, she ran 6–8 miles a day and lifted weights. She limited herself to no more than 5 grams of fat a day and became vegetarian, not a family tradition. She developed fearfulness about "dangerous foods," such as cheese.

A.H. stated: "I felt cold all the time. My skin was dry. My hair started to fall out, and my nails were brittle." Menses stopped at age 19. She was preoccupied with thoughts of food, weight, and shape for "80% to 90% of the day." She experienced a severe fear of becoming fat, even as her weight plummeted. Her mood became fragile, often tearful. Family walked on eggshells to avoid displeasing her and provoking irritability. Her temperament was that of a self-critical, perfectionistic young woman for whom expression of emotions was a foreign experience.

Three previous residential treatments resulted in only temporary improvement, followed by relapse into her anorexic pattern of

behavior and thinking. No binges or purges developed. Shortly before admission, she fell on an icy sidewalk and fractured her humerus. Bone mineral density by dual-energy X-ray absorptiometry (DEXA) scan for L1–L4 was 0.675 g/cm^3, T = –3.4. Left hip bone density was 0.674 g/cm^3, T = –2.2. Hemoglobin of 5.4 required four transfusions. Labs showed hyponatremia of 126 mEq/L. Calcium was low, but potassium and phosphorus were within normal limits. EKG showed bradycardia of 45. Her displaced fracture was treated surgically with a rod, followed by physical therapy. A.H. had never abused alcohol or street drugs. Her two previous relationships had ended poorly, and she had a sense of distrust about future relationships.

During the inpatient portion of comprehensive treatment, A.H. restored her weight to 85% of her goal weight of 122–126 pounds. She accomplished the remainder of her weight restoration in partial hospital (day hospital, 5 days a week), achieving 100% of her goal weight. Her morbid fear of fatness diminished, and she developed an acceptance of a healthy weight and body image, learning cognitive behavioral skills in group, individual, and family therapy to challenge her overvalued beliefs. Her mood became euthymic and stable. No psychotropic medications were needed. She made considerable progress in catching up with normal development during a year of relapse prevention outpatient therapy after discharge.

Severe starvation in a woman with AN is shown in figure 1.1. A similar degree of severe starvation in a male with AN is displayed in figure 1.2.

Background and Overview

In the nineteenth century, tuberculosis and syphilis were called "great pretenders," presenting to clinicians in many disguised forms. In a similar manner, eating disorders in the twenty-first century appear in various disguises to physicians and other health professionals. They are frequently underdiagnosed and overlooked. For typical ways in which eating disorders present to primary care providers, see table 1.1.

Eating disorders are a public health concern with a greater prevalence than childhood diabetes or schizophrenia. These disorders, especially

anorexia nervosa, vie with substance use disorders for having the highest probability of death among all psychiatric disorders, resulting in 6 to 12 times the expected mortality for females 12–25 years old (Arcelus et al., 2011; Fichter and Quadflieg, 2016). The standardized mortality ratio (SMR) for both AN and substance use disorders is about 5 to 6 times the expected normal rate (Himmerich, Hotopf, et al., 2019; Fridell et al., 2019), although there is some evidence that mortality rates are decreasing. Even when eating disorders are subclinical by formal diagnostic criteria, they impose a significant burden on the normal development of a healthy identity and social functioning in young people entrained in their symptomatology. Preoccupation with weight, shape, and body image too often result in functional psychosocial impairments and a decreased quality of life during the adolescence and early adulthood of a large percentage of young women and significant numbers of young men. It continues as a threat to health in older decades as well.

Eating disorders, especially AN and bulimia nervosa (BN) are, at their core, disorders of eating behavior that develop as a way to deal with challenges in various areas of life, including self-esteem, emotional self-regulation, existential fears of development, shame, trauma, aversive experiences, societal norms, and relationship conflicts. They often develop from an overvaluation of the benefits of slimness or shape change as methods to deal with these issues. Some of these issues, especially emotional self-regulation, shame, and low self-esteem, are present in individuals suffering from binge eating disorder (BED) as well. AN and BN often occur in younger persons who are in vulnerable periods of development, especially in the transition from the preteen and early teen phase of achieving industry versus inferiority (the fourth stage of Erik Erikson's theory of psychosocial development; Erikson 1950) to the phase of forming a consolidated personal identity versus the adverse outcome of role diffusion. A high school girl struggling with a distorted body image is depicted in figure 1.3.

The fears and goals of patients with AN or BN have almost nothing to do with food in its essence, just as the fear of riding in elevators has little to do with the actual risk of elevators. The quest for extreme thinness or a perfect body serves as a strategy, as pseudo-solution, to deal

Figure 1.1. A typical very ill woman with anorexia nervosa. Note the extreme emaciation and lack of soft tissue.

with various distressed emotions and personal conflicts, especially a sense of inadequate control and effectiveness in life situations. At times these disorders appear to be responses to authority figures requiring weight loss for vocational, athletic, or familial concerns. Not uncommonly in the past but not unknown today, AN has been a response to a search for personal holiness or a means for a woman to gain a voice in a male-dominated culture. Theories advocating a predominantly sociocultural,

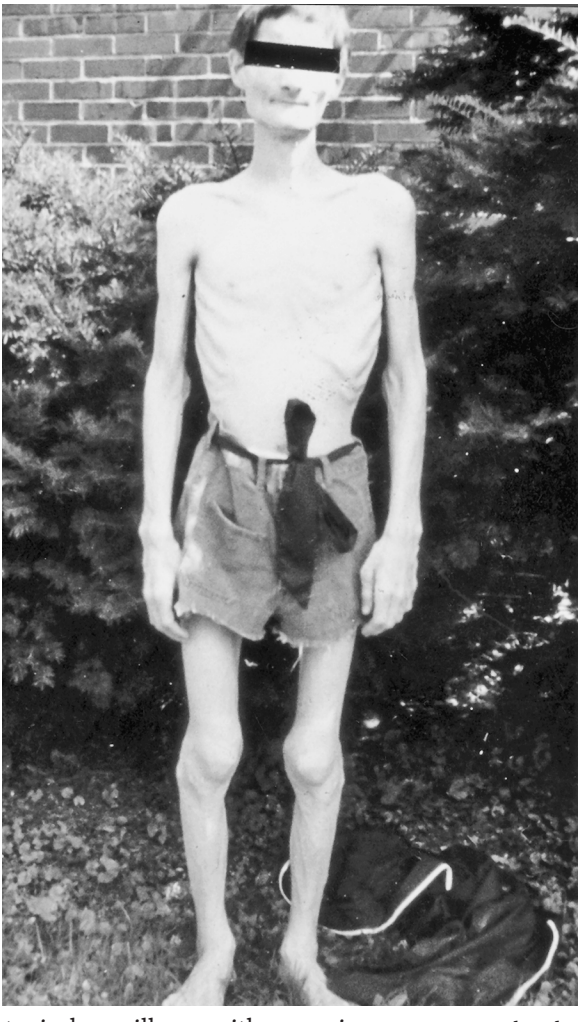

Figure 1.2. A typical very ill man with anorexia nervosa. Reproduced with permission of the patient.

biomedical, intrapsychic, or feminist origin of eating disorders all contribute some elements of understanding.

Our society promotes the belief that weight loss and shape change are ways to improve low self-esteem, to become respected and envied by others, and to feel effective, conferring the belief that the individual who can regulate body weight (women > men) and shape (men > women) is in control of life's challenges, and that regulation promises to be a means

TABLE 1.1. Typical ways in which eating disorders present to primary care providers

▪ Provider notes unexplained decreased weight; preoccupation with dieting or body shape; abnormal laboratory signs; failure to increase weight proportional to height in preteen or teenaged patient; unexplained amenorrhea in menstruating patient.
▪ Parent/teacher/coach or significant other expresses concerns about child/student/partner being preoccupied with weight or having unexplained weight loss; unexplained loss of periods in daughter; signs of vomiting without medical illness; excessive use of bathroom or shower after meals; signs of binge eating / disappearance of food; relentless workouts.
▪ Referral by coach, teacher, or psychologist for evaluation of a possible eating disorder.
▪ Presentation of patient *complaints* seemingly compatible with disorders of specific organ systems: thyroid (hyperactive, weight loss), malabsorption (diarrhea), other GI disorders (reflux, ulcers, lactose intolerance, stomach pain, bloating), cancer (weight loss, loss of appetite in older patients), parasites (unexplained weight loss), dermatologic (loss of hair), dental (multiple new caries, loss of enamel). *Rule in* eating disorders before *ruling out* medical causes of eating disorder symptoms.
▪ Positive answers to routine screening surveys, written or in person, for presence of an eating disorder; e.g., (modified) SCOFF questionnaire or SDE questionnaire.
▪ Routine history and examination of a patient in high-risk sports or interest groups, or with LGBTQ+ orientation.

of building self-esteem and respect, externally or internally. While often discussed as separate disorders, AN, BN, and BED are all part of one overarching, larger disorder of eating dysregulation driven by cognitive distortions—specifically, a shared overvalued belief in the benefits of thinness, usually combined with an excessive fear of fatness, and often associated with distortion in body perception (Andersen and Yager, 2009).

It may be helpful to conceptualize a novel terminology similar to that used for other disorders, such as autism spectrum disorders: the eating disorders spectrum (EDOS). These disorders form a diagnostic spectrum with many shared features. They occur on a spectrum of severity and most often present with comorbidities, changing in severity and associated symptoms over time in response to life events and therapeutic interventions. They also evolve on a spectrum of migration between different subgroups, which have porous boundaries. Three of the most frequent transitions are from AN, restricting subtype (AN-R), to AN, binge-purge subtype (AN-BP); from AN of any subtype to BN; and from BN to BED. As autism has recently come to be understood as a spectrum

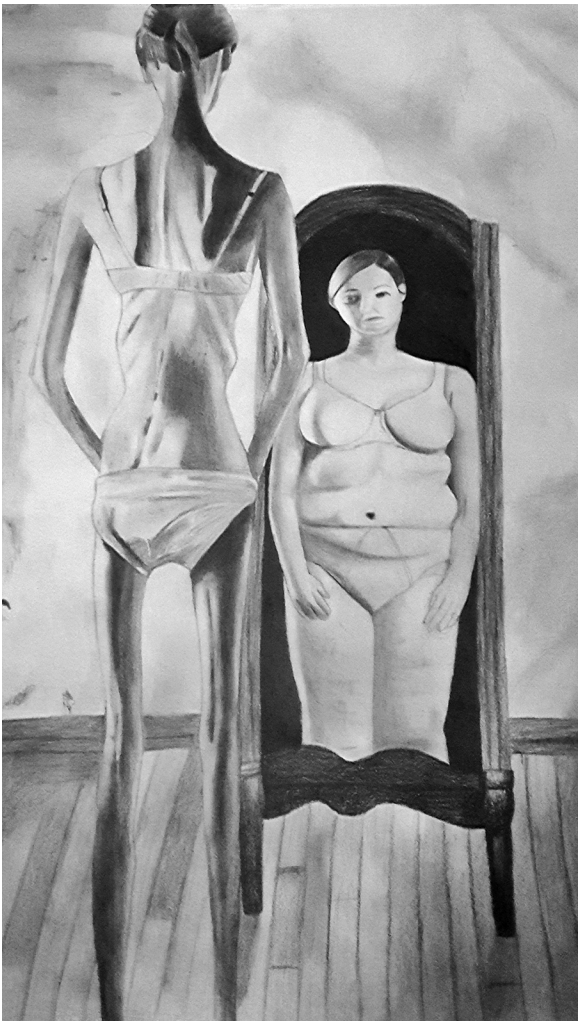

Figure 1.3. A high school student's struggle with perceptual distortion

disorder, so eating disorders are best understood as a spectrum disorder, including AN, BN, and BED. The ARFID category—avoidant/restrictive food intake disorder—remains apart in most ways.

Not everyone is equally vulnerable to developing an illness on the eating disorders spectrum. Some of the risk factors predisposing a person to the development of an eating disorder are summarized in table 1.2 (Gonçalves et al., 2014; Hilbert, Pike, Goldschmidt, et al., 2014; Machado

TABLE 1.2. Risk factors that increase the probability of developing an eating disorder

Risk factor	Comments
Gender	More females than males, but not as large a discrepancy as commonly believed (10:1 in clinical referrals, 2–3:1 in community sample), meaning males are underdiagnosed.
Age	Individuals in preteen, teen, and early 20s age groups most common, with peaks at ages 12–16 for AN and 17–21 for BN; cases noted from 7 to 77 years old.
Geography	Highest incidence in westernized societies and upper socioeconomic classes in developing countries.
Personality (typical, though not required, features)	*Anorexia nervosa:* sensitive, perfectionist, persevering, obsessional, self-critical features of temperament. *Bulimia nervosa:* impulsivity, emotional intensity, mood lability, dramatic features. *Binge eating disorder:* high harm avoidance, low self-directedness, conflict avoidant.
Family history	Increased in families with history of depressive/anxiety spectrum disorder, obesity.
Heritability/genetics	Estimated 40%–60% of risk factors are heritable. Concordance in monozygotic twins 3–4 times as high as in dizygotic twins. Elevated in utero testosterone.
Interest groups	Students in strict dance schools, bodybuilders, models, wrestlers, participants in sports with high performance and appearance demands (girls' gymnastics, figure skating), cross-country runners, media professionals, actors.
Sexual orientation	All individuals in the LGBTQ+ community are at increased risk for eating disorders.
Age of onset of dieting behavior	Girls starting to worry about weight between ages 6 and 10; by age 14, 60%–70% trying to reduce weight.
Racial and ethnic group	Largely independent of racial and ethnic designation if westernized body ideals for thinness are adopted; increased in African American families of higher socioeconomic status. Highly variable according to cultural norms and socioeconomic factors.
Predisposing medical disorders	Type 1 diabetes mellitus, cystic fibrosis, medical prescription of corticosteroids, atypical antipsychotics, unexplained gastrointestinal complaints.
Predisposing psychiatric disorders	Depressive disorders, anxiety disorders, attention deficit / hyperactivity disorder, post-traumatic stress disorder, obsessive-compulsive disorder, autism spectrum disorders.
Location	More common in urban areas than in rural.
Influence of media	Decreased self-esteem, more self-critical body image, after viewing impossible-to-achieve body size/shape ideals in media; onset of eating disorders with introduction of TV and other media, e.g., in South Sea Islands; thinspiration internet sites.

et al., 2014; Meier et al., 2015; Steinhausen et al., 2015). Actors and performing artists have been recognized as also being vulnerable (Szabó et al., 2019), in addition to people working in television. The greater the number and intensity of risk factors, the more likely they will be present during vulnerable windows in development, with fewer compensating resilience factors, resulting in a greater likelihood of developing an eating disorder.

Risk factors are therefore probabilistic, not deterministic. They are generally culture bound, occurring primarily in industrialized cultures that value thinness and have sufficient availability of food so that being overweight is a realistic possibility, but with considerable variation. "Social norms vary massively within cultures, social strata, ethnicity and gender . . . Understanding eating disorders requires integration of psychological factors into a cultural and epidemiological context" (Stern, 2018). Extreme slimness as a goal in societies experiencing widespread starvation is rare, except in the governing or wealthy classes of developing countries that have adopted westernized body images.

Only in 1979 did BN come out of hiding in plain sight; BED has even more recently been recognized as a true eating disorder. Less clearly related to AN, BN, and BED, which is a more heterogeneous diagnostic grouping, is ARFID, which has become widely applied to patients who would formerly have been left out of a diagnosis qualifying them for treatment within the category of an eating disorder. The ARFID diagnosis most often combines disorders with varied disturbances in eating or feeding from undefined causes, most often in younger individuals, but not excluding older individuals.

Throughout this chapter, the term *patient*, derived from the Latin and Greek words for a person who suffers, is used in preference to *client*, which is a term focused on an economic relationship with a person seeking services. The term *client* may also have as a goal the establishment of an equality of power dynamics, with a sense of partnership between a health care provider and the person requesting services, but its use may be an overreaction against perceived paternalism in past medical treatments. Client falls short of conveying the suffering of those afflicted with medical and psychiatric disorders, in this case, eating disorders.

A strong sense of respect for the autonomy of the patient, with a goal of beneficence, not control, is intrinsic to the historical role of the provider to the patient.

Epidemiology

The probability of accurately and validly identifying the incidence, prevalence, and characteristics of a disorder X depends on (1) how an investigator defines disorder X, (2) the specific population in which disorder X is looked for (total population, gender specific, age specific, clinical vs. community samples, health insurance statistics, national databases, death statistics), and (3) the methods of diagnosis that are used (standardized clinical interviews, questionnaires, self-diagnosis). So it is with eating disorders. Among the factors that contribute to differing estimates of the incidence and prevalence (point prevalence and lifetime prevalence) of eating disorders are changes in diagnostic criteria from *DSM-IV* to *DSM-5* (*Diagnostic and Statistical Manual of Mental Disorders*, 5th edition) and from ICD-10 to ICD-11 (International Statistical Classification of Diseases and Related Health Problems, 11th revision); differences in the location of case finding (clinical admissions vs. community samples); and variations in the methods of case finding (new screening instruments, clinic discharge diagnoses, or full individual diagnostic evaluations).

Recent studies have confirmed a general finding that a high percentage of teens and young adults in total meet criteria for an eating disorder. Using the *DSM-5* criteria in a Finnish study (Silén et al., 2020), the prevalence of lifetime eating disorders was found to be 17.9% for females and 2.4% for males (pooled across genders, 10.5%). The estimated lifetime prevalence for females and males, respectively, were 6.2% and 0.3% for AN, 2.4% and 0.16% for BN, and 0.6% and 0.3% for BED. These estimates vary considerably in other studies. A study of eating disorders in European women (Keski-Rahkonen and Mustelin, 2016) found <1%–4% of women met criteria for AN; <1%–2% suffered from BN; BED was present in <1%–4%; and subthreshold eating disorders were found in 2%–3% of women. The authors found that 70% of the individuals reported comorbid

disorders and that only one-third of eating disorders were detected by health care providers, a finding that has been repeated over and over again.

The specificity or breadth of the diagnostic criteria contributes to the variation in epidemiological estimates. What is clear is that in specific subgroups (ballet dancers, actors, models, weight- and appearance-related sports), the prevalence of an eating disorder is high. Past estimates of the female-to-male ratio have varied widely according to testing location and diagnostic criteria used. Males with full or subthreshold AN, for example, present to hospital clinics in a ratio of 10:1 females to males, but more accurately, a 3:1 female-to-male ratio probably exists in community-based samples.

Aggregating results from multiple sources, BN appears to be present in 0.3%–7% of adolescents and young adults, with an estimate of 1.5%–2% as an approximate figure, and with a ratio of 3:1 females to males. BED is the most gender-neutral eating disorder, being present in 2%–3% of the vulnerable population. The prevalence of subsyndromal AN is considerably higher than that of the full diagnostic syndrome, underlining the fact that weight loss and frequency of disordered eating in the population are *dimensional* constructs, with weight loss arrayed on a spectrum from least to greatest weight loss.

Controversy remains as to whether the incidence and prevalence of eating disorders are increasing, decreasing, or remaining stable. Studies finding an increased prevalence may reflect the possibility that these disorders are truly increasing or may simply mirror the wider use of better diagnostic screening tools as well as enhanced clinician awareness. Or patients are possibly now more willing to seek help. The preponderance of evidence supports an actual increase in cases of eating disorders in westernized societies, especially BN and BED.

Differences in the prevalence of eating disorders depends on the criteria used for diagnosis, using broad or narrow criteria, and point in time. Flament et al. (2015) found that the prevalence of full-threshold eating disorders increased from 1.8% using *DSM-IV* diagnostic criteria to 3.7% with *DSM-5*. A large study of 2,977 participants in Australia (Bagaric et al., 2020) using *DSM-5* criteria found a lifetime prevalence of BN to be 1.21% for males and 2.59% for females, and the lifetime prevalence

of BED (broad diagnosis) was 0.74% for males and 1.85% for females, higher than previously reported. Considerable variation exists in different studies based on different populations and sampling methods. Nevertheless, whatever the specific results of various epidemiological studies, eating disorders by any method of sampling remain a substantial problem in public health, nationally and internationally.

The concern for weight reduction may be provoked by psychosocial factors out of proportion to actual medical benefit, as is often the case with anorexia nervosa. But recently documented studies clearly indicate that overweight is a public health priority for clinical providers as well. Ogden et al. (2020) have studied more than 92,000 noninstitutionalized individuals over age 17 regarding their weight changes between the two study periods of 1999–2000 and 2017–2018. Among men, obesity increased overall from 27.5% to 43.0%, and severe obesity from 3.1% to 6.9%. Among women overall, obesity increased from 33.4% to 41.9%, and severe obesity from 6.2% to 11.5%.

Diagnosis: General Features

The first key concept in making an accurate diagnosis of eating disorders is to think of an eating disorder as a diagnostic possibility in any patient presenting for care. In an adult emergency department, for example, a locale in which eating disorders are not often considered a diagnostic possibility, nearly 16% of all patients screened positive for one (Dooley-Hash et al., 2019). A disorder that is not considered a possibility cannot be diagnosed. The second guideline in diagnosing an eating disorder is to first *rule in* the eating disorder by a screening tool, by a set of questions found to be personally useful, or by a longer, more formal evaluation. Avoid first attempting to *rule out* all possible medical or psychiatric causes of the presenting symptom or sign before considering that an eating disorder may be present. A delay in diagnosis may increase the severity of the disorder, cause iatrogenic morbidity through excessive medical testing, and make treatment more difficult. A systematic psychiatric history and mental status examination will reveal the true picture of the disorder underlying the medical presentation. A study of

patients in a gastrointestinal clinic who defied a medical diagnosis—patients who did not respond to the treatments offered—were found to meet criteria for an eating disorder. With appropriate treatment, they showed improvement (Emmanuel et al., 2004; Andersen, 2004).

When a young patient denies a fear of becoming fat but has self-induced weight loss, or desires excessive slimness or shape change, the clinician may have to depend on the account of parents, a peer, or a significant other. AN in a patient who meets full diagnostic criteria presents as a relatively "public" disorder, usually with obvious thinness, often in otherwise energetic young individuals. It may be marked by proud assertions of thinness. BN, however, is most commonly a private, secretive disorder, marked by shame and disclosure avoidance. BN generally involves planned or impulsive exits from functions involving food. A spouse, parent, or significant other living with someone struggling with bulimia nervosa may not be aware of the condition despite the covert presence of years of illness.

AN has been recognized for several centuries, while BN was given a diagnostic term as recently as 1979 (Russell). These two disorders are two sides of the same coin: they both generally begin with dieting to lose weight, leading in the case of AN to sustained low weight, while in BN, dieting results in less significant or no weight loss, instead provoking a breakthrough of hunger in the form of binge eating. The binge episode is followed by compensatory efforts to avoid weight gain through various means, most often by self-induced vomiting, but also by use of laxatives or diuretics, heroic exercise, or subsequent bouts of intense fasting. Compensation by means of exercise or increased fasting may be present in about 15%–20% of individuals with BN. Bulimia nervosa is in many ways a failed attempt at anorexia nervosa: a dieting effort in a person without the persevering traits of a classic anorexic patient to persist in weight reduction even in the setting of extreme hunger. Individuals with BN appear to lack the physiological or psychological features of AN to allow them to tolerate prolonged semistarvation without eating; the binge episodes may represent weight loss efforts that are disrupted by substance abuse, especially alcohol or marijuana. The binge episodes of BN quickly evolve from being driven by hunger alone to

being triggered by dysphoric moods, as well as becoming a response to autonomic conditioning provoked at certain times and in specific locations.

BED is defined by the behavior of chronic binge eating with *no* compensation for the binges—no purging and minimal subsequent dietary restriction or driven exercise—but with the result of psychologic and medical distress, self-recrimination, guilt, and shame after binge eating. BED is not a hedonic experience. Its definition is even more recent than that of BN (Devlin, Goldfein, and Dobrow, 2003; Latner and Clyne, 2008). The concept of BED was in wide clinical use for at least a decade or more before being included in the *DSM*-5.

Commonly there is migration between these eating disorder subtypes, most often from AN to bulimic symptoms, in the form of either developing binge-purge episodes while maintaining low weight (AN-BP) or restoring enough weight to no longer suffer from the medical signs of starvation but experiencing the binge-purge behaviors of BN. Approximately 50% of patients with BN have had a past episode of AN, either a full or partial syndrome. Some attempts at rapid, severe weight loss may quickly develop into typical BN; others may first lead to a period of typical, low-weight anorexia nervosa, and then develop into the binge-purge form of AN. Only later, in a period from weeks to years, BN develops in up to 50% of individuals beginning with AN.

The most recent eating disorder to have formal agreed-on diagnostic criteria, but probably not the last one to be formalized as research continues, is ARFID, avoidant/restrictive food intake disorder, a more heterogeneous disorder with several subcategories (Sharp and Stubbs, 2019; Bryant-Waugh, 2019; Keery et al., 2019).

There is concern that prevalence rates for AN, BN, and BED are significantly higher among sexual minorities than heterosexual respondents (Kamody, Grillo, and Udo, 2020), and that members of this minority are underdiagnosed. Sexual minorities reporting experiences with perceived discrimination had significantly higher prevalence of AN than those not endorsing perceived discrimination: 3.78% versus 0.82%, AOR 5.06. Additionally, victims of sexual assault have been documented to experience a substantially higher prevalence of eating disorders (26.7%), as

well as depressive symptoms and sleep disorders (Schou-Bredal et al., 2020). These sufferers often are hesitant to report their assaults.

Diagnostic Criteria in *DSM-5* and ICD-11

The criteria for a formal diagnosis of an eating disorder have changed from *DSM-IV* to *DSM-5*, and from ICD-10 to ICD-11, although the mental and behavioral features of patients with these disorders have not changed. The criteria in *DSM-5* are roughly convertible to ICD-11 diagnoses in most cases but with some moderately important differences. Fairburn and Cooper (2011) have summarized a view I share that "the *DSM-IV* scheme for classifying eating disorders is a poor reflection of clinical reality . . . *DSM-5* will only partially succeed in correcting this shortcoming." With that admonition in mind, a discussion of current diagnoses of eating disorders by *DSM-5* follows.

The most admirable contribution of *DSM-5* has been to reduce the previous greatly overused and clinically confusing diagnosis of eating disorders not otherwise specified (EDNOS) in *DSM-IV*, a diagnosis not often used by experienced clinicians employing more enduring criteria. This older EDNOS term was previously applied to 50% or more of patients clearly suffering from anorexia nervosa, confusing for clinicians and for health insurance reimbursement. Fairburn (2008) has promoted the helpful suggestion that eating disorders may be best understood by linking them through a transdiagnostic conceptualization. This transdiagnostic conceptual integration applies best to AN, BN, and BED, but less so to ARFID.

DSM-5: Pros and Cons

DSM-5 is a widely used updated diagnostic instrument that is overall a step in the right direction and helpful for diagnostic coding and for insurance reimbursement. It is not synonymous with revealed truth, however. Making a diagnosis by using only the *DSM-5* criteria as a checklist is forcing patients into a procrustean bed. A diagnosis always needs to be expanded into a summary formulation in the record, including

pertinent features of the history, mental status examination, proposed treatment, relevant unique characteristics of the patient, comorbid disorders, and tentative prognosis. The strengths and weaknesses of *DSM-5* both deserve recognition. Several positive changes from *DSM-IV* to *DSM-5* are worth noting.

For a diagnosis of AN (table 1.3), a required weight criterion is now met by a general term, restriction of energy intake relative to requirements that results in "significant" voluntary weight loss. This requirement contrasts with the previously suggested, but often falsely interpreted, final weight of "less than 85%" of healthy weight (*DSM-IV*). The "less than 85%" of healthy weight was meant as an example (*e.g.* means "exempli gratia,"

TABLE 1.3. *DSM-5* criteria for Anorexia Nervosa (AN)

A. Restriction of energy intake relative to requirements leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health. Significantly low weight is defined as a weight that is less than minimally normal or, for children and adolescents, less than that minimally expected.

B. Intense fear of gaining weight or becoming fat, or persistent behavior that interferes with weight gain, even though at a significantly low weight.

C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight.

Specify whether:
Restricting Type: no recurrent binge eating or purging during previous three months.
Binge eating/purging Type: recurrent episodes of binge eating or purging activities during the last three months.

Specify if:
In partial remission: After full criteria for anorexia nervosa were previously met, Criterion A has not been met for a sustained period, but either Criterion B or C is still met.
In full remission: After full criteria for anorexia nervosa were previously met, none of the criteria have been met for a sustained period of time.

Specific current severity:
The minimum level of severity is based, for adults, on current body mass index (BMI) (see below) or for children and adolescents, on BMI percentile. The ranges below are derived from World Health Organization categories for thinness in adults; for children and adolescents, corresponding BMI percentiles should be used. The level of severity may be increased to reflect clinical symptoms, the degree of functional disability, and the need for supervision.

Mild: BMI >17 kg/m^2. Moderate: BMI 16–16.99 kg/m^2. Severe: BMI 15–15.99 kg/m^2.
Extreme: BMI <15 kg/m^2.

Source: American Psychiatric Association, 2013.

or "for example") and was specified as such in *DSM-IV*, but it was often read as mandatory. The more general requirement of a significantly low body weight represents an advance. Many of the previously misclassified cases of anorexia nervosa, called EDNOS because of the presence of menstrual activity of some degree, now meet the criteria for AN in *DSM-5*. In addition, the former requirement of amenorrhea is appropriately no longer included, since it ignores women on contraception, as well as postmenopausal women and all males.

Two categories of true anorexia nervosa are still commonly underdiagnosed by the criteria of *DSM-5*: (1) anorexia nervosa at normal BMI, and (2) anorexia nervosa with an inadvertent onset. An important conceptual error in *DSM-5* is the notion that anorexia nervosa means attainment of a low weight by dieting or other means. In fact, and more correctly, AN is present when a (usually) voluntary restriction of energy intake takes place to the extent that a significant *decrement* in weight occurs relative to a previously stable *set point*, causing the individual to manifest the physical, psychological, and social features of starvation whatever the final weight is in BMI or pounds. When symptoms of starvation from voluntary restriction of food intake occur at a seemingly normal final weight and are associated with hypothermia, bradycardia, hypotension, preoccupation with food, depressed mood, social isolation, and fear of weight gain, the diagnosis is anorexia nervosa. Pertinent for diagnosis is the *delta*, or decrement, of weight change from the starting weight to the lowest weight attained, the amount of weight change that is sufficient to produce medical symptoms of starvation, not the final number on the scale. A patient at seemingly normal weight as judged by population average weights may well be suffering from true AN. In the current *DSM-5*, this individual would be diagnosed as meeting criteria for OSFED (other specified feeding or eating disorder), specifically *atypical anorexia nervosa*. On the one hand, from a clinical perspective, nothing is truly atypical about these individuals with AN at a seemingly normal final weight or who have some menstrual activity (Watson and Andersen, 2003). For another perspective on atypical anorexia nervosa, please see chapter 15, which argues for a separate category of atypical anorexia nervosa.

Case Study: Anorexia Nervosa at Normal Weight, aka Atypical Anorexia Nervosa

J.N. is a 17-year-old high school senior referred for evaluation of weight loss, menstrual irregularities, hypothermia, and hypotension. She became self-conscious of her weight at age 14, at 150 pounds, 65 inches tall (BMI 25.7, mild overweight). After several ineffective diets, including the "paleo" diet, her weight decreased to 112 pounds (BMI 19.2, within the normal range). On mental status examination, she expressed fear of becoming fat through loss of control of her eating. She endorsed feeling cold, always hungry, preoccupied with thoughts of food, irritability, weakness, and lightheadedness. In some clinics, she would be diagnosed as having *atypical anorexia nervosa*. The term *atypical* attached to a diagnosis often confuses clinicians and may lead to denial of payment from health care plans. She was correctly diagnosed with AN, mild in severity, with good prognosis, and responded well to a multidisciplinary program in the partial psychiatric hospital. More dramatic examples abound, with patients losing weight from a BMI of 40 or 50, who restrict energy intake to a final BMI of 24 or 25, a seeming victory, but with all the stigmata of otherwise typical anorexia nervosa.

The second type of individuals with anorexia nervosa that may be missed using *DSM-5* criteria are those experiencing an inadvertent onset of weight loss, from various causes, but who then demonstrate a typical course and symptomatology of AN after the initial inadvertent weight loss. Once the weight has been lost from a medical cause (e.g., infectious disease, Crohn's disease, abdominal operation, jaws wired after a crash), the patient rather suddenly has a sense of accomplishment, finds the weight loss desirable, and "takes over the wheel" to drive the weight loss lower or to avoid any weight gain.

Case Study: Inadvertent Weight Loss Leading to Anorexia Nervosa

B.W. is a 40-year-old woman treated as an inpatient on a specialized eating disorders unit. At age 35 she experienced severe abdominal pain and was hospitalized with a diagnosis of Crohn's disease. Before her

hospitalization, at 67 inches tall, she had stably weighed 115 pounds since her midteen years and had never dieted or desired to lose weight. Because of the Crohn's disease, her weight decreased to 105 pounds but on prednisone returned to 110 pounds. During the next several years, she suffered the losses of her sister-in-law, a close friend, and then the death of both parents. These losses resulted in the onset of a depressive illness and weight loss to 92 pounds. She was successfully treated for her depressive disorder but resisted directions to increase her food intake, daily eating only a small amount of tuna fish and carrot sticks. She expressed for the first time a fear of becoming fat and of losing control if her nutritional intake increased as directed. She stated that a weight in the low 90s was all she could tolerate despite being hypothermic and hypotensive. When the psychiatrist treating her depression urged her to restore her lost weight, she began taking diuretics to decrease her scale weights and inducing vomiting after meals.

Another category of AN often overlooked is *sub-subclinical anorexia nervosa*, characterized by a less severe continuing voluntary restriction of food intake to achieve a socially enviable body weight and shape (called "social X-rays" by Tom Wolfe) but medically symptomatic weight loss, accompanied by overvalued beliefs that preoccupy the person's life and direct her behaviors. Even moderate weight loss but not obvious emaciation may have adverse medical consequences. About 8% of failure to conceive in women may be due simply to being about 4 to 7 pounds below the weight for normal gonadotropic functioning.

For BN, the criteria in *DSM-5* (table 1.4) are similar to those in *DSM-IV* except for reducing the required frequency of binge episodes from two to one episode per week for 3 months. This helpful change allows more patients to be diagnosed and treated. Nonetheless, the diagnostic criteria for BN overlook a substantial number of sufferers because of the requirement that there be repeated ingestion of food in amounts larger than those that most *observers* would consider normal ("objective" binges). This raises the question of how to diagnose the disorder in patients who eat amounts larger than *they* consider acceptable according to their rigid internal goals. Even though an observer might not see what would be

TABLE 1.4. *DSM-5* criteria for Bulimia Nervosa (BN)

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by BOTH of the following:
   1. Eating, in a discrete amount of time (e.g., within a 2-hour period) an amount of food that is definitely larger than what most individuals would eat in a similar period of time under similar circumstances.
   2. A sense of lack of control over eating during an episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).

B. Recurrent inappropriate compensatory behavior in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, or other medications; fasting; or excessive exercise.

C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least once a week for three months.

D. Self-evaluation is unduly influenced by body shape and weight.

E. The disturbance does not occur exclusively during episodes of anorexia nervosa.

Specify if:
In partial remission: After full criteria for bulimia nervosa were previously met, some but not all of the criteria have been met for a sustained period of time.
In full remission: After full criteria for bulimia nervosa were previously met, none of the criteria have been met for a sustained period of time.

Specify current severity:
The minimum level of severity is based on the frequency of inappropriate compensatory behaviors (see below). The level of severity may be increased to reflect other symptoms and the degree of functional disability.

Mild: An average of 1–3 episodes of inappropriate compensatory behaviors per week
Moderate: An average of 4–7 episodes of inappropriate compensatory behaviors per week
Severe: An average of 8–13 episodes of inappropriate compensatory behaviors per week
Extreme: An average of 14 or more episodes of inappropriate compensatory behaviors per week.

Source: American Psychiatric Association, 2013.

considered a "binge," it is a binge to the person. This may well be the case in the category of *purging disorder*, still under investigation, which requires no binges—at least, no large observable binges. I have not evaluated any patient who purges after a bout of eating that others might not consider to be large unless the amount eaten is larger than they "should" have eaten. How much food is acceptable is an internal judgment.

BED is an overdue but now universally accepted eating disorder in *DSM-5* (table 1.5), rather than a provisional category under investigation, as in *DSM-IV*. Similar to the *DSM-5* criteria for bulimia nervosa, only one binge per week for 3 months is required. There is specification of the

TABLE 1.5. *DSM-5* criteria for Binge Eating Disorder (BED)

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
   1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than what most people would eat in a similar period of time under similar circumstances.
   2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).

B. The binge eating episodes are associated with three (or more) of the following:
   1. Eating much more rapidly than normal.
   2. Eating until feeling uncomfortably full.
   3. Eating large amounts of food when not feeling physically hungry.
   4. Eating alone be cause feeling embarrassed by how much one is eating.
   5. Feeling disgusted with oneself, depressed, or very guilty afterward.

C. Marked distress regarding binge eating is present.

D. The binge eating occurs, on average, at least once a week for 3 months.

E. The binge eating is not associated with the recurrent use of inappropriate compensatory behavior as in bulimia nervosa and does not occur exclusively during the course of bulimia nervosa or anorexia nervosa.

Specify if:
In partial remission: After full criteria for binge eating disorder were previously met, binge eating occurs at an average frequency of less than one episode per week for a sustained period of time.
In full remission: After full criteria for binge eating disorder were previously met, none of the criteria have been met for a sustained period of time.

Specify current severity:
The minimum level of severity is based on the frequency of episodes of binge eating (see below). The level of severity may be increased to reflect other symptoms and the degree of functional disability.

Mild: 1–3 binge eating episodes per week
Moderate: 4–7 binge eating episodes per week
Severe: 8–13 binge eating episodes per week
Extreme: 14 or more binge eating episodes per week

Source: American Psychiatric Association, 2013.

binge severity to help determine the intensity of the treatment required and to follow the decrease in severity as a measure of treatment response. Bringing the diagnosis of BED squarely into an accepted diagnostic category within the eating disorders spectrum has been somewhat slow in coming about but is clinically of great import and has been well validated as an often overlooked but functionally impairing eating disorder.

Case Study: Typical Binge Eating Disorder (1)

J.A. was a 32-year-old female referred for evaluation by the bariatric surgery team at Johns Hopkins. She was denied bariatric surgery because of her binge eating. Her struggle with weight began in junior high school. When she was active in sports, she maintained her weight in a normal range, but a knee injury sidelined her. When she was 17, her father had severe pneumonia, and she was told he might die; she began to eat excessively. Her mother began an extramarital affair. Her parents were obese. When the patient began a stressful job after high school, her binges increased to four times a week. Triggers for binges included anger, anxiety, loneliness, and fears of failure. She found that binge episodes made her feel calm. She said that binge episodes caused her stomach to feel bloated, but her mouth still felt hungry. Typical binges might include a pint of ice cream, four grilled ham and cheese sandwiches, and several cinnamon rolls. After binge eating, she felt ashamed. Her boyfriend expressed anger at her overeating. She never compensated for her overeating by purging in any form. She estimated that she spent 75% of her day thinking about food, considering what she would eat next. She suffered from GERD (gastroesophageal reflux disease) and obstructive sleep apnea. She confirmed frequent use of marijuana and a history of cocaine use. Her BMI was 51. Her diagnoses included binge eating disorder, generalized anxiety disorder, substance use disorder, and relationship conflict. She began treatment in the partial psychiatric hospital program (day program) with a plan to interrupt her binge episodes; lose a moderate amount of weight; stop substance use; begin couples therapy; engage in cognitive behavioral treatment with components of dialectical behavioral therapy. The overall plan was to reengage with the bariatric surgery program after one year of being free of binge eating.

Case Study: Typical Binge Eating Disorder (2)

Z.P. is a 39-year-old married male car salesman referred for evaluation of disordered eating. He has struggled with excessive eating all through his life. He remembers growing up in a family centered around communal eating. Both parents were overweight but uncon-

cerned about their weight. Grandparents indulged him with extra food. At first his "stockiness" made him a good member of a school football team. In college he oscillated in weight between 180 and 220 pounds from his repeated unsuccessful attempts at dieting. Once he established himself in his job and family, he experienced higher stress, often self-treating his anxiety with binge snacks during breaks at work and after coming home from work.

His binge episodes worsened when he restricted his breakfast to coffee only. He began to steal candy bars from his son's room. He felt he had to eat snacks at work brought in by coworkers and satisfied customers. His coworkers began to joke about having to hide food from him. He was taught to finish everything on his plate as well as the remainder of food left by his children. He never purged in any form. He felt shame after binge eating but also felt ineffective at dealing with emotional distress without it. He had a lifelong history of anxiety but never sought treatment. In the year before evaluation, he began to experience depressive episodes but no suicidality. His BMI increased to 36. He began to need a CPAP device for sleep. He was diagnosed as meeting criteria for binge eating disorder and generalized anxiety, with a history of ADHD. He was started in an outpatient treatment program, including cognitive behavioral psychotherapy and sertraline, and was referred to a ten-session weekly program in mindfulness. He appeared motivated and made good progress.

Intrinsic to all clinically relevant eating disorder diagnoses is *functional impairment* by the person experiencing the symptoms and behaviors. The functional impairment may be medical, psychological, social, or often, all of these. Dieting may be a full-time ongoing activity for some people, or it may be an intermittent practice, as is typical for many people in westernized societies. There is a marked difference between giving lip service to dieting and sustained dieting to the point of producing a functionally impairing eating disorder that becomes out of control. The change from semiserious or casual dieting to an eating disorder reflects the change from a voluntary practice to a largely involuntary behavior, a fact often not appreciated by onlookers, especially fathers.

Avoidant/restrictive food intake disorder (ARFID) is now a generally accepted category, with criteria listed in *DSM-5* (table 1.6). It has taken on a useful role in clinical psychiatry despite its outward heterogeneity. ARFID focuses on a combination of eating and feeding disturbances from several sources, often occurring in childhood, but not exclusively so. Eating disorders may be roughly divided into two categories: those that require an overvaluation of the benefits of slimness or shape change, and those that do not. AN and BN fall into the first category. Patients with BED value weight loss but appear unable to pursue effective dieting and avoid purging to compensate for their binges. Patients meeting ARFID criteria fall into a second category, without overvalued beliefs in the benefits of weight loss or shape change.

The overarching question of what constitutes an eating disorder remains in flux, but for the time being, the four major categories of AN, BN, BED, and ARFID cover most clinical cases. For example, in a recent review

TABLE 1.6. *DSM-5* criteria for Avoidant/Restrictive Food Intake Disorder (ARFID)

A. An eating or feeding disturbance (e.g., apparent lack of interest in eating or food; avoidance based on the sensory characteristics of food; concern about aversive consequences of eating) as manifested by persistent failure to meet appropriate nutritional and/or energy needs associated with one (or more) of the following:
   1. Significant weight loss (or failure to achieve expected weight gain or faltering growth in children).
   2. Significant nutritional deficiency.
   3. Dependence on enteral feeding or oral nutritional supplements.
   4. Marked interference with psychosocial functioning.

B. The disturbance is not better explained by lack of available food or by associated culturally sanctioned practice.

C. The eating disturbance does not occur exclusively during the course of anorexia nervosa or bulimia nervosa, and there is no evidence of a disturbance in the way in which one's body weight or shape is experienced.

D. The eating disturbance is not attributable to a concurrent medical condition or not better explained by another mental disorder. When the eating disturbance occurs in the context of another condition or disorder, the severity of the eating disturbance exceeds that routinely associated with the condition or disorder and warrants additional clinical attention.

Specify if:

In remission: After full criteria for avoidant/restrictive food intake disorder were previously met the criteria have not been met for a sustained period of time.

Source: American Psychiatric Association, 2013.

of cases referred for evaluation in an eating and weight disorders clinic, out of 27 patients seen, the following diagnoses were made: 3 with AN-R; 5 with AN-BP; 7 with BN; 5 with BED; 5 meeting criteria for ARFID; 1 with OCD (obsessive-compulsive disorder); and one with a delusional disorder. This is a typical combination of diagnoses seen in many eating and weight disorders clinics over a short period and represents the variety of eating disordered patients that a clinician could anticipate evaluating.

Case Study: Avoidant/Restrictive Food Intake Disorder (1)

E.B. was a 40-year-old bus driver who was referred to an eating and weight disorders clinic for severe weight loss of unknown origin. Although he had some difficulty in giving a pertinent longitudinal history of his difficulty in eating, he thought he remembered having choked at age 8, requiring vigorous slaps on his back to dislodge the food stuck in the trachea. This event happened on two more occasions and then did not reoccur. When he attended a dinner at age 39 for a friend at work, which featured a thick steak, he said he lost his appetite after starting to chew a piece of the steak and then spitting it out, but he did not know why he did that. He gradually became fearful of other foods, as well even softer foods, until he experienced a sense of panic at virtually any attempts to chew and swallow. His weight decreased from a BMI in the mid-20s to a BMI of 16. He expressed perplexity at his inability to eat. Endoscopy revealed no abnormalities. He was given a diagnosis of ARFID based on his concern about the aversive consequences of eating with an avoidance due to the sensory characteristics of food. He was admitted to the Eating and Weight Disorders Unit, where he gradually recovered by a program of desensitization concerning his phobic avoidance of food in his mouth. He became able in the CBT (cognitive behavioral therapy) groups to give a name to his fear and to make connections between his childhood choking experiences and his more recent weight loss. He returned to his job at a healthy weight.

Case Study: Avoidant/Restrictive Food Intake Disorder (2)

J.Q. was a 25-year-old woman who was referred to the eating and weight disorders clinic from the Department of Internal Medicine for

severe weight loss and inability to discontinue her nasogastric (NG) feeding. About two years prior to referral, the patient had found her work to be very stressful and time consuming, causing her to neglect her eating. Additionally, she had decided to become vegan because of ethical concerns. She began to experience abdominal pain, bloating, and decreased appetite. Her weight decreased from 105 pounds to 75 pounds. Her internist carried out an extensive work-up, including a CT of the abdomen, all with negative findings. Superior mesenteric artery (SMA) syndrome was ruled out on fluoroscopy. She was started on NG feeding. She had no childhood or teenage history of gastrointestinal disorders. On mental status examination, she expressed discouragement but denied any fear of becoming fat. She denied body image distortion, saying she was too thin. She never purged. She never experienced binge eating, even though she often thought about food. She was cold intolerant and often felt fatigued. A diagnosis of ARFID was made, and she was referred to the inpatient Eating and Weight Disorders Unit. Her nasogastric tube was discontinued. She gradually restored weight by nursing-supervised refeeding, coming close to her previous weight. She came to appreciate applying the principles of CBT to her life. While no definitive diagnosis was made besides ARFID, that diagnosis allowed her to recover and maintain her weight.

When a thorough medical evaluation does not reveal the cause of significant weight loss, and the patient does not meet criteria for other psychiatric disorders, a diagnosis of ARFID should be considered by both medical and psychiatric providers. Treating ARFID patients in psychiatric facilities experienced with eating disorders, including inpatient units, partial hospital, or outpatient clinics, may offer the best hope for improvement. Experienced eating disorders facilities at all levels of intensity can generally evolve an individualized program for nutritional rehabilitation even in the absence of classic symptoms of other eating disorders or a definitive cause. These patients often fall between the cracks of medical care and psychiatric care but are well served on an eating disorders unit, with good liaison between psychiatry and internal medicine always being a goal.

Pica and Rumination Disorder

Two other specific categories of eating disorders are presented in *DSM-5*, *pica* and *rumination disorder*. Pica often has a medical origin, arising from a deficiency in micronutrients, and can become a method to placate the hunger of starvation; it is seldom seen in psychiatric clinics but is a syndrome to be recognized when present.

Rumination disorder is occasionally seen and needs to be considered as a diagnostic possibility. Rumination disorder, when not of medical causation, represents a kind of behavioral approach-avoidance relationship to food. Food is eaten, usually from normal hunger or from emotional distress, and then either repeatedly chewed and spit out, or swallowed and regurgitated, to be reswallowed or spit out, in some ways representing a greatly abbreviated version of bulimia nervosa. The *DSM-5* criteria do not require a morbid fear of becoming fat for a diagnosis of rumination disorder, but in my experience, the combination of a drive for thinness, hunger from food restriction, and the inability to resist the ingestion of food as far as the mouth is generally present. When the primary intent of avoiding food has been overwhelmed by hunger or other needs, the next step is chewing and spitting out the food, or bringing up food from the stomach if swallowing could not be avoided.

Other Specified Feeding or Eating Disorder

The last category within *DSM-5*, other specified feeding or eating disorder (OSFED), includes subclinical forms of AN, BN, and BED, which are self-explanatory (table 1.7). Purging disorder remains for evaluation by further studies as to clinical utility. In my experience, individuals who appear to only purge and do not have obvious "objective" binge episodes have generally eaten more than their internal standards have allowed a person to eat, whether in the form of a subjective binge or even of seemingly normal amounts of food that registered mentally as being excessive or forbidden after being ingested.

A form of eating disorder found within the OSFED category that deserves wider recognition is night eating syndrome, a not uncommon

TABLE 1.7. *DSM-5* criteria for Other Specified Feeding or Eating Disorder (OSFED)

This category applies to presentation in which symptoms characteristic of a feeding and eating disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the feeding and eating disorders diagnostic class. The other specified feeding or eating disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific feeding and eating disorder. This is done by recording "other specified feeding or eating disorder" followed by the specific reason (e.g., bulimia nervosa of low frequency).

Examples of presentations that can be specified using the "other specified" designation include the following:

1. Atypical anorexia nervosa: All of the criteria for anorexia nervosa are met, except that despite significant weight loss, the individual's weight is within or above the normal range.

2. Bulimia nervosa (of low frequency and/or limited duration): All of the criteria for bulimia nervosa are met, except that the binge eating and inappropriate compensatory behaviors occur, on average, less than once a week and/or for less than 3 months.

3. Binge eating disorder (of low frequency and/or limited duration): All of the criteria for binge eating disorder are met, except the binge eating occurs, on average, less than once a week and/or for less than 3 months.

4. Purging disorder: Recurrent purging behavior to influence weight or shape (e.g., self-induced vomiting, misuse of laxatives, diuretics, or other medications) in the absence of binge eating.

5. Night eating syndrome: Recurrent episodes of night eating, as manifested by eating after awakening from sleep or by excessive food consumption after the evening meal. There is awareness and recall of the eating. The night eating is not better explained by external influences such as changes in the individual's sleep-wake cycle or by local social norms. The night eating causes significant distress and/or impairment in functioning. The disordered pattern of eating is not better explained by binge eating disorder or another mental disorder, including substance use, and is not attributable to another medical disorder or to an effect of medication.

Source: American Psychiatric Association, 2013.

syndrome that may be of either psychiatric or medical origin. Night eating syndrome, according to the criteria in *DSM-5* and in my experience, many times represents a form of unwanted overeating resulting from unappreciated daytime restriction of food leading to hunger after the dinner meal. One subtype of night eating syndrome noted in this *DSM-5* category is "excessive consumption after the evening meal." When excessive eating occurs after the evening meal but before sleep, the causes are most often a combination of hunger from earlier daytime food restriction combined with some emotional distress. The unslaked hunger from food

restriction during the daytime is only partly assuaged by the evening meal. Relaxation resulting from daytime activity, combined with the postingestive drowsiness of the evening meal, and at times sedation from drink, together may contribute to letting down one's guard after supper and eating out of control. The second subtype of night eating syndrome, when a person arises after truly falling asleep and then eats excessively, causing emotional and physical distress, most often arises from one of two causes: first, shortchanging the body of nutritional needs through daytime semistarvation causes a resurgence of hunger in sleep and may be enhanced by dreaming about food. Semistarvation does change dream life, often resulting in dreams about food and eating, which combine with hunger to be a powerful force to eat unwanted, often high-density calories in a partially awake state. The second type, which is being increasingly recognized by clinicians, is characterized by involuntary eating during arousal from sleep due to the effect of nonbenzodiazepine sleep medications such as zolpidem, zaleplon, and eszopiclone. The acronym for this disorder is SRED, sleep-related eating disorder. Sleep parasomnias include sleep walking, sleep driving, and unremembered sexual activity; and of forensic interest, violent or destructive activity may rarely occur. Unfortunately, sleep does not always knit up the ravell'd sleeve of care, and the potions to relieve insomnia may be worse than the condition they are designed to treat. Night eating resulting from an adverse side effect of sleep medication in some individuals is technically excluded from the night eating syndrome criteria within OSFED but, nonetheless, needs to be recognized by clinicians prescribing these medications.

ICD-11: Pros and Cons

ICD-11 has been presented to member states in 2019 for review and, if accepted, will come into effect on January 1, 2022. Most of the world uses the ICD (International Classification of Diseases) for diagnostic guidelines to physical, mental, and behavioral disorders, and therefore it needs to be recognized and respected by US clinicians. Regarding eating disorders, it appears from preliminary studies that ICD-11 outperforms ICD-10 (Claudino et al., 2019). Although both ICD-11 and *DSM-5*

represent the results of serious prolonged evaluation and field studies, they differ in a few regards. The ICD-11 uses the term *guidelines* rather than *criteria* but does have specific diagnostic requirements.

For AN, ICD-11 requires significant underweight from restrictive eating that is not due to unavailability of food or another medical condition, listing a body mass index of less than 18.5 kg/m^2 in adults and a BMI-for-age under the 5th percentile in children and adolescents as a guide. It requires an overvaluation of low body weight along with preoccupation about weight loss; it also includes the restricting and binge-purge subtypes. It differs from *DSM-5* by requiring a specific body weight that is indicative of anorexia nervosa, with some excluding caveats. The amenorrhea requirement has been eliminated. ICD-11 would exclude from a diagnosis of AN patients with a BMI greater than 18.5 in adults or greater than the 5th percentile of weight in children and adolescents, even if they manifested clear medical and psychological symptoms typical of classic AN. If a specific lowest acceptable weight is required, then it too would miss diagnosing cases of so-called atypical anorexia nervosa as true AN because the patient's weight after dieting falls in the so-called normal range.

The guidelines for BN in ICD-11 are similar to those in *DSM-5*. Both require, in addition to binge eating, repeated inappropriate compensatory behaviors to prevent weight gain as well as excessive preoccupation with body weight and shape. ICD-11 does not specifically include subjective binge eating as a form of BN, even though studies (Watson, Fursland, et al., 2012) have found that subjective BN could not be meaningfully distinguished from so-called objective binge eating in the more commonly diagnosed presentation of BN. After all, the rapid ingestion of large "objective" amounts of food may be hedonic to some. Conversely, the eating of small amounts of food beyond the limit set by internal controls may be dysphoria producing. The desirability or unwantedness of the food ingested lies in the mental life of the individual.

BED has now been standardized in ICD-11 as a recognized and diagnosable clinical entity and is no longer a diagnosis only for study. The criteria are similar to those of *DSM-5* and specifically distinguish BED from medical causations, identifying Prader-Willi syndrome as one medical condition to be excluded.

Also brought into the fold of major eating disorder diagnoses in ICD-11 is the category of ARFID, with features similar to those of *DSM*-5. All eating disorder diagnoses in ICD-11 require functional impairment as a result of the disorder. The ARFID diagnoses in ICD-11 specifically exclude motivation by preoccupation with body weight or shape or the presence of body image distortion. BED excludes individuals who behaviorally compensate for the unwanted food ingested but does not exclude psychological distress, albeit more in passive resignation rather than active compensation.

Underdiagnosed and Overlooked Groups

The most commonly overlooked or underdiagnosed patient populations suffering from AN include males, minorities, older persons, and those with mental challenges, including those with autism spectrum disorder or intellectual disability. Diagnoses of eating disorders in males appears at times to be excluded by popular stereotypes in inexperienced clinicians. Additionally, eating disorders in males may be statistically inconvenient for researchers, who often use only females in their reports. Males are discussed in detail in a later chapter.

Recent studies have documented the prevalence of AN, BN, and BED in the minority groups of African American and Latinx individuals (Udo and Grilo, 2018). AN is less common in minorities, but this may reflect socioeconomic differences. Likewise, older women are increasingly found to suffer from eating disorders, either continued from younger years or of recent onset, even of postmenopausal origin (Beck, Casper, and Andersen, 1996). Eating disorders in different generations within the same family are not uncommon. Finally, individuals with intellectual disabilities may suffer from an eating disorder (Galluci et al., 2003). The intellectual level required to develop a desire for thinness includes individuals with IQs in the category of mild intellectual disability as well as those within the autism spectrum. The mentally challenged may achieve increased self-esteem and social acceptance from weight loss.

A category that may not be considered enough includes military service personnel. The armed forces have strict annual weight requirements for career service that may provoke excessive rapid dieting in

both men and women that lead to a continuing eating disorder (Masheb et al., 2019). Additionally, those experiencing PTSD (post-traumatic stress disorder) or sexual trauma appear to be at increased likelihood of developing eating disorders in addition to other symptoms (Blais et al., 2017). Breland et al. (2017) document the ways in which military environments affect eating habits. Medical and psychological help for eating disorders in military personnel is made difficult by the dual roles of military providers, especially psychiatrists, who are responsible for giving expert care, but also are often expected to relay information about illnesses in personnel they treat to higher ranking officers, sometimes resulting in restricting promotion in these individuals.

A Transdiagnostic Approach to the Eating Disorders Spectrum

At times investigators into the diagnosis of medical and psychiatric disorders face the need to decide between a grouping or a splitting approach to the classification of a complex group of disorders. Both approaches have their virtues and drawbacks. Fairburn (2008) has promoted the helpful suggestion that eating disorders may be best understood by linking them through a transdiagnostic conceptualization (table 1.8). This transdiagnostic conceptual integration applies best to AN, BN, and BED, but less so to ARFID.

Time and clinical perspicacity may uncover more forms of eating disorders that are clinically important and will be included in future iterations of *DSM* or ICD. In the meantime, the potential benefits of a transdiagnostic approach to the identification and treatment of currently recognized eating disorders deserves serious consideration.

A transdiagnostic approach includes those individuals who meet full criteria for a specific eating disorder as well as those who are on the path to developing an eating disorder but who are subsyndromic, or atypical, at presentation and might be otherwise excluded from treatment. Milder or more recent cases are more prevalent than those that meet full criteria. Eating disorders of all current diagnostic subtypes and degrees of severity in illness share several commonalities:

TABLE 1.8. Transdiagnostic features of all eating disorders

1.	All patients with an eating disorder have an abnormal relationship to food.
2.	Most patients with an eating disorder experience medical abnormalities and require at a minimum medical evaluation.
3.	All patients suffering from an eating disorder suffer from some degree of psychological distress.
4.	Eating disorders result in functional impairment in one or more areas of living.
5.	Most patients with eating disorders meet criteria for one or more comorbid psychiatric disorders, which need to be recognized and included in treatment planning.
6.	Eating disordered patients suffer from some degree of social dislocation.
7.	Treatment generally must recognize the existential consequences of the eating disorder regarding a sense of identity and meaning. Their existence is not synonymous with their eating disorder.
8.	Patients benefit from understanding that eating disorders are true illnesses and not simply bad choices or flaws in living.
9.	Families, partners, and other carers generally play a role in the recognition and treatment of the eating disorder.
10.	Patients benefit from understanding that eating disorders are most commonly the result of genetic vulnerabilities interacting with environmental and developmental forces.
11.	Some eating disorders are ego-syntonic, but the majority are ego-dystonic.
12.	Eating disorders present in many hidden forms and disguises.
13.	Eating disorders may transition from one diagnosis to another, of from one subtype within a diagnosis to another subtype.
14.	Most eating disorders, especially AN, BN, and BED, share common modes of treatment, especially in the use of evidence-based psychotherapies.

1. *Patients with these disorders all have an abnormal relationship to food*. An appreciation of hunger and satiety are essential regulatory drives to sustain life. The experience of normal hunger and satiety requires a complex neuroendocrine system for regulation of eating and maintenance of weight. When medical disorders, such as cancer, and other psychiatric disorders, such as major depressive disorders, are eliminated as causes for the abnormal eating behavior of a patient, and the patient meets criteria for an eating disorder, the patient then falls within the

transdiagnostic category of eating disorders. If a patient migrates from one subtype of eating disorder to another (for example, AN-R often transitions to AN-BP or to BN), the patient remains within the category of suffering from an eating disorder. The treatment in any eating disorder subtype will necessarily involve shared therapeutic strategies for restoring normal eating patterns and weight in regard to age, weight, height, gender, and genetic history.

2. AN, BN, and ARFID, as well as the diagnostic subtypes within ARFID, generally *experience medical abnormalities* and require medical evaluation at a minimum, but frequently medical interventions as well.
3. *All patients within the eating disorders diagnostic subtypes experience various forms of psychological distress*. Treatment for the psychological distress may include a variety of methods, including evidence-based psychotherapies, behavioral relearning, and psychopharmacological medications, to improve demoralization, overvalued beliefs, depression, anxiety, and behavioral abnormalities. In previous diagnostic approaches (*DSM-IV* and ICD-10), the eating disorder categories then recognized, AN and BN, were considered to share overvalued beliefs, such as a morbid fear of becoming fat, a relentless drive for thinness, and body image distortion. While anorexia nervosa and bulimia nervosa do share these overvalued beliefs, BED does so less often, and subtypes within ARFID generally do not. Hence, a more overarching approach to the forms of psychological distress associated with any eating disorder is now required.
4. *All patients with eating disorders meeting diagnostic categories share the feature of functional impairment*, mild to severe, with impairments in the physical, psychological, social, and spiritual realms. It is doubtful that a person who suffers from an eating disorder but who asserts complete comfort with their abnormal eating and thinking is free of any functional impairment.
5. *Most patients with eating disorders, with some exceptions for milder and briefer illnesses, meet criteria for comorbid conditions*. Eating

disorders are seldom solitary diagnoses. This fact means that the treatment approach by health care providers must be integrative; a strategy for simultaneous or sequential treatment of comorbid conditions needs to be planned.

6. *All patients with eating disorders suffer from social dislocation in some form* or another; strategies are required to develop patterns for the development of, or return to, normal social interactions.
7. *An existential component of treatment is often well warranted* to assist patients in developing a sense of personal wholeness, integrity, and worthwhileness, becoming people who suffer from eating disorders, not people who are nothing more than their disorders. They learn that they have an eating disorder, as a person has the flu, and that who they are is not synonymous with their disorder.
8. *All patients will benefit from learning that eating disorders are illnesses and not flaws*; there are no "bad" reasons for developing eating disorders. When patients have experienced an eating disorder from late childhood onward, they appreciate learning in treatment that eating disorders are *strategies* to deal with multiple issues, including issues in development, challenges in mood management, ways to manage relationship distress, and methods to respond to cultural pressures that may be overwhelming in intensity and duration. Accepting that eating disorders are illnesses is not synonymous with abdication of personal responsibility as a partner in treatment.
9. *Families, partners, and other carers are generally involved*, whether enabling and accommodating, or helping set kind but realistic healthy standards, showing nonpossessive warmth and healthy role modeling. An increasing number of studies have found that many cases of eating disorders in adolescents may be successfully treated by a family-based approach, especially bulimia nervosa and anorexia nervosa with a BMI greater than about 14 or 15.
10. *Most eating disorders can be best understood to result from a combination of genetic vulnerabilities interacting with environmental forces*; epigenetic factors may up-regulate or down-regulate

genes. The fact that gene expression may be modified by many factors has revolutionized genetic research and offers contributions to an understanding of the etiology and treatment of eating disorders.

11. Most eating disorders are *ego-dystonic*, although one, anorexia nervosa, may be *ego-syntonic*, not seen as foreign or as a disorder.
12. Eating disorders are *present in many hidden forms* and may be confused with other medical or psychiatric disorders.
13. Eating disorders often *transition* from one type of eating disorder to another, or from one subtype to another subtype within the same type of eating disorder.
14. Most eating disorders share common modes of treatment, especially AN, BN, and BED, and to some extent ARFID.

AN is generally experienced as an ego-syntonic disorder, not considered foreign or as something requiring treatment. To patients with AN, their physical and psychological burdens are often considered an acceptable price for achieving their relentlessly overvalued goal of extreme thinness. BN and BED, in contrast, are most commonly experienced as ego-dystonic. Patients with BN or BED are seldom content with their disorder for long periods, being distressed by their inability to simply restrict food intake to lose weight; they most often feel shame and a sense of inefficacy regarding their patterns of binge eating, and in the case of BN, their purging behaviors. Some college students with short-lived hedonic binge symptoms fueled by alcohol or purging after socially required large meals may be the exception to having feelings of shame or guilt. The ego-syntonic versus ego-alien experience of ARFID diagnoses remains to be understood. It is possible that children who are very picky eaters, causing weight to fall below the 5th percentile, may consider their choices not to be ego-dystonic and of more concern to their parents than to themselves.

Screening Questionnaires: SCOFF or SDE?

The major problem in diagnosing an eating disorder in a patient is simply not thinking of an eating disorder as a diagnostic possibility. Once a

clinician diagnoses an eating disorder, multiple rating scales are available for assessing the severity, the comorbidity, and other factors. These more comprehensive tests include the Eating Disorders Examination (EDE-Q, 41 questions), the Eating Attitudes Test (EAT, 26 items), the Eating Disorders Inventory (EDI, 91 items), and others. Most primary care clinicians, however, need simpler but reasonably validated screening instruments for diagnosis in their practice if the possibility of an eating disorder is brought to mind. Universal screening in primary care medicine and OB-GYN for all females from 10 years of age to 50 and males in higher risk categories is strongly urged, a practice greatly assisted by a brief validated instrument.

For screening, the previous recommendation had been to use the SCOFF five-item questionnaire (table 1.9) as a reasonable instrument for use in primary care and OB-GYN as well as for school populations. While the search for a brief 100% sensitive and specific instrument lies in the future, there are now two good practical candidates for screening. Maguen et al. (2018) have documented that a newly devised questionnaire, the Screen for Disordered Eating (SDE, five questions), had a higher sensitivity than the SCOFF in identifying true cases, especially BED. The five SDE questions (table 1.10) ask about emotional eating, feelings of being out of control when eating, inducing purging, being preoccupied with thinness, and believing oneself to be fat when others see thinness. Although the SDE was validated in female VA patients, it appears to be competitive with the SCOFF as a screening instrument for use by primary

TABLE 1.9. The SCOFF questionnaire

1. Do you make yourself Sick because you feel uncomfortably full?
2. Do you worry you have lost Control over how much you eat?
3. Have you recently lost Over 14 pounds ("one stone") in a three-month period?
4. Do you believe yourself to be Fat when others say you are too thin?
5. Would you say that Food dominates your life?

Source: Adapted for the American population from Morgan, Reid, and Lacey (1999). In the original publication, item 3 reads: "One stone (14 lbs)" rather than "Over 14 pounds ('one stone')."

Note: Ask these questions of all patients 10–40 years old or in high-risk groups. Count 1 point for every yes; a score of 2 or more indicates a likely case of anorexia nervosa or bulimia nervosa.

TABLE 1.10. The Screen for Disordered Eating (SDE)

1. Do you often feel the desire to eat when you are emotionally upset or stressed?
2. Do you often feel that you can't control what or how much you eat?
3. Do you sometimes make yourself throw up (vomit) to control your weight?
4. Are you often preoccupied with a desire to be thinner?
5. Do you believe yourself to be fat when others say you are thin?

Source: Maguen et al., 2018.

care clinicians in general populations. Studies to validate its use in a broader population are under way.

The SCOFF questionnaire's criteria were developed in London (Morgan, Reid, and Lacey, 1999), modified for North America by using "14 pounds" rather than the charmingly archaic "one stone" for weight loss. Its virtue is its simplicity and documented history of high utility in identifying probable cases of eating disorders for further, more definitive evaluation. Again, the key concept in making a reliable diagnosis of an eating disorder is that it is made not by ruling out all conceivable medical causation of symptoms but by confidently ruling in the disorder based on a brief history and mental status examination, after use of a screening questionnaire. Both the SCOFF questionnaire and the SDE are now recommended for screening.

ARFID diagnoses do not at this time appear to be detectable by screening instruments but are best made by following up on a high index of suspicion, confirming any of the subtypes within ARFID with clinical questions and ruling out alternative causes.

Some clues to secretive eating disorders are summarized in table 1.11. These are tip-offs that should lead to a systematic history and mental status examination to ascertain whether an eating disorder, albeit often initially denied, is present.

Orthorexia: Valid Diagnosis or Fad?

The term *orthorexia*, occurring most often in the popular literature, is of uncertain clinical significance, but it is a useful term. It generally

TABLE 1.11. Clues to hidden or secretive eating disorders

Anorexia Nervosa
- Unexplained weight loss, especially in adolescents, or failure to gain weight proportional to height in preteens and adolescents
- Secondary amenorrhea in adolescents or preadolescents without obvious medical cause
- Membership in avocational or identity groups promoting or requiring weight loss / shape change (ballet, wrestling, modeling, female gymnastics, cross-country running, gay orientation)
- Preoccupation with need for additional weight loss or body shape change, despite obvious thinness (females) or muscularity (males)
- Frequent mirror gazing
- Frequent "weight loss talk" without a medical basis; negative comparison of self to thinner peers
- Unexplained high TSH, low T3, low LH/FSH resulting from central hypothalamic hypogonadism; feeling cold compared with peers; objective hypothermia; unexplained hair loss; development of lanugo hair (fine, downy hair on face, arms, and back)

Bulimia Nervosa or Binge-Purge Form of Anorexia Nervosa
- Unexplained hypokalemia (low potassium)
- Family or peer reports of patient vomiting without medical illness; finding boxes of laxatives or diuretics; patient regularly disappearing to the bathroom or taking showers after meals
- Swollen or tender parotid glands without viral, bacterial, or other medical cause
- Loss of dental enamel on lingual surface, or large number of new caries
- Gastroesophageal reflux or symptoms of esophageal erosions in young person without medical cause; hoarseness without medical cause
- Yo-yo weight pattern

Binge Eating Disorder
- BMI over 30–35
- Continued unexplained steady weight gain or sudden rapid weight gain
- Shame or guilt in discussing eating patterns
- Hopelessness, helplessness about weight

Avoidant/Restrictive Food Intake Disorder
- Overly picky eating in children leading to significant growth restriction (severe limitation of type of food eating; complaints of taste, texture)
- Onset of fear-based avoidance of eating in adults without known medical cause
- Limitation of food intake to energy drinks; eating food only in liquid form
- Continued gastrointestinal symptomatology without medical cause

means that there is an excessive, obsessional, preoccupying concern about eating only the healthiest foods, combined with a search for dietary purity, for the perfect vitamins and supplements, which causes some degree of functional impairment. While at times orthorexia may rule a patient's life personally and socially, it has no current agreed-on diagnostic criteria for health care providers. Nonetheless, it may be considered one hazard of an overvalued desire for an impossibly perfect pattern of healthy eating. When pervasive, it often leads to adverse

behavioral, psychological, or interpersonal consequences, including loss of weight. When treatment is sought, the patient may benefit from an open-ended exploration of the goals and consequences of the orthorexia. Vulnerable features of personality, inaccurate information about food, or prior sensitizing experiences may underlie the quest for dietary perfection. Cena et al. (2018) found that the common diagnostic features in studies on orthorexia generally included obsessional or pathological preoccupation with healthy nutrition; experience of negative emotional consequences; and psychosocial impairment, leading at times to malnutrition and weight loss.

The Natural History of Anorexia Nervosa and Bulimia Nervosa

The natural history of eating disorders, especially AN and BN, usually begins only after months to years of a socioculturally induced preoccupation with weight and body size, leading to weight comparisons with peers and role models as well as to attempts at weight loss by various dieting regimens. Although AN and BN differ in some aspects of their natural history, they share many features (figure 1.4). The dieting behavior is driven by the internalized, overvalued desire for thinness, beginning in the 7- to 10-year-old age group, usually combined with a fear of becoming fat and furthered by a distorted body image. To most American women, a "normal" weight means achieving 85% or less of the population average weight. Teens and preteens are encouraged by multiple sources to develop unrealistic goals of an idealized thin but fashionable body size combined with an improbable body shape (defined by slender hips, visible ab muscles, absence of a "stomach," and thighs that do not touch).

Nature has created young people with a dimensional variation in weight and shape. Dieting behavior most commonly (but not exclusively) begins during the pubertal years of normal developmental body change (occurring at younger and younger ages) or in later adolescence, times of changes not only in body size and shape but in hormones, mood, social interactions, and family relationships. The attitudes and values learned earlier about the benefits of thinness drive the dieting behavior.

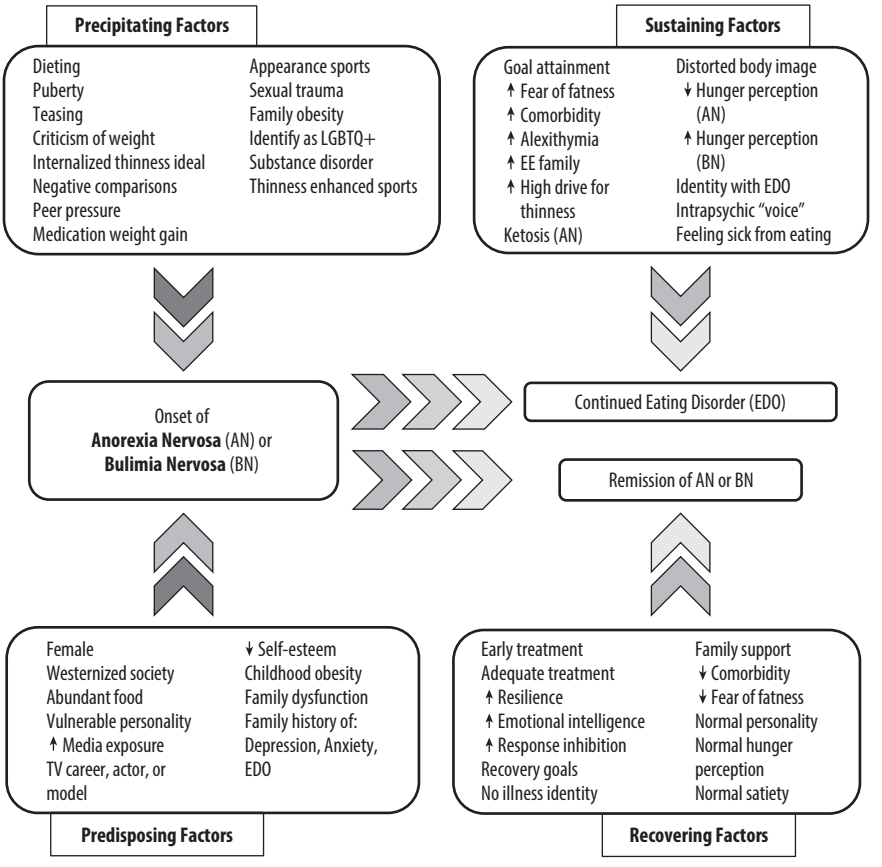

Figure 1.4. Natural history of anorexia nervosa and bulimia nervosa. Written by Arnold E. Andersen and created by Kari Miller.

A specific precipitating factor—such as comments by friends or family, especially mothers or siblings; boyfriends; coaches; teachers; or peers, suggesting that a young person is overweight, is in danger of becoming overweight, or needs to change the shape of a body part—moves the incubating phase of predisposition into the development of an overt clinical eating disorder. Of course, some may dip their toe in the swimming pool of thinness, attempting to lose weight, but never dive in, or may abandon the effort completely. Others may dive in, developing short-term eating disorder symptoms, and then get out quickly. This is especially true for the self-limited binge-purge episodes that often occur

during the college years in a significant number of students. Up to 50% of 9- to 10-year-old girls are already worried enough about becoming fat that they are beginning to diet, even though far fewer than 50% are medically overweight.

The concerns about becoming fat or about the need to reduce weight are catalyzed in part by alarmist announcements about how many children are overweight. T. Moran summarizes the nutritional state of many American children as most typically being "overfed calorically, underfed nutritionally" (personal communication, June 2010). A lack of fitness completes the vicious circle of an increasing desire for more and more thinness combined with a desire for an unattainable, perfect body shape. The real-life context of this preoccupation is the fact that an increase in the actual average body weight of teens is occurring along with an earlier developmental increase in the percentage of body fat, causing earlier attainment of the critical amount of body fat for the onset of menses. Some contributions to the vicious circle of increased weight and decreased fitness include excessive time spent with media and video games, decreased athletic programs in schools, and the overabundance of highly processed foods. Many northern European countries, in contrast, especially the Scandinavian countries, cultivate a "virtuous circle," encouraging eating a healthy, palatable lunch in schools and teaching lifetime carry-over athletic skills for all students rather than only for the athletically elite.

The natural history of AN and BN suggests an ideational schematic: a cluster of *precipitating factors* act on a person made vulnerable through various *predisposing risk factors*, leading to the onset of an eating disorder that may then be consolidated into an enduring illness by *sustaining factors*. Counterbalancing these illness-related variables, *therapeutic and resilience factors* have also been increasingly identified as paving the way to recovery (see figure 1.4).

The sustaining factors for AN and BN appear to be the result of a combination of psychosocial as well as biomedical elements. Comments praising weight loss are like manna from heaven for a girl or young woman who seeks approval not granted by herself due to her sensitive personality, or who lives in the shadow of a more extroverted sibling.

Patients who have lost considerable weight will generally feel sick if made to eat large amounts of food by families, especially foods high in fats, confirming their beliefs that they are meant to be thin. Temporary partial lactose intolerance may be present. Putative changes in serotoninergic functioning as sustaining factors have been suggested but not yet fully proved. There are clearly secondary serotoninergic changes.

The classic account of men who were conscientious objectors during World War II and who voluntarily agreed to lose substantial amounts of weight to provide clinical information on better ways to refeed returning starved prisoners of war remains essential reading for understanding how weight loss changes thinking and behavior (Keys et al., 1950). Many of the symptoms of anorexia nervosa are symptoms of medical starvation, independent of cause. The difference between conscientious objectors and patients with AN is not their amount of weight loss, but the absence of overvalued beliefs about the benefits of slimness in the former, and the presence of these beliefs in patients with AN.

Once dieting has started, several outcomes are possible. A frequent result is the development of the most common eating disorder: a subsyndromal disorder characterized by chronic dieting, with or without binges, not fulfilling strict criteria for any diagnosable eating disorder yet a burden and a source of distress during critical developmental years. In a smaller number of these dieting individuals, the process may then go on until they develop a full eating disorder that meets diagnostic criteria, either for anorexia nervosa or bulimia nervosa.

AN may present as a pure food-restricting subtype (AN-R) or as a binge-purge subtype (AN-BP). The first binge of BN is usually triggered by dieting-induced hunger, provoking binge eating and then compensatory measures to get rid of the unwanted calories because of the fear of fatness. Soon, however, the binges of BN generalize to become a way to deal with any distressed mood, including depression, anxiety, anger, or feeling stuck or bored. Autonomic patterning (Haedt-Matt et al., 2014) helps to lock in the syndrome behaviors. Finally, AN and BN go on to give a personal identity in which the individual cannot imagine life without either self-starving or binge-purge behavior or both. Eating disorders too often become an identity and a vocation, which helps to explain their

frequent intransigence. Although the ages for onset are most often between 11 and 22 years, we have evaluated patients with a spectrum of ages of onset from between 7 and 72 years of age. Dieting should be considered a four-letter word, in contrast to a stable lifetime pattern of balanced nutritional intake, such as following the Mediterranean diet, well validated by multiple studies.

Some males may suffer from reverse anorexia nervosa, a syndrome occurring in bodybuilders and weightlifters, all of whom fear they are never big enough or never have a low enough percentage of body fat to reveal well-defined muscles. Reverse anorexia nervosa is more of a concept than a well-defined diagnosis. The goal is often attainment of less than 5% body fat. A more recent term sometimes applied to this syndrome is *muscle dysmorphia*, but reverse anorexia nervosa does not overlap completely with either muscle dysmorphia or body dysmorphia. Reverse anorexia nervosa is seldom seen in women.

The continuation of the nonadaptive state of starvation in patients with AN presents some enigmas, as detailed by Walsh (2013). Multiple neural mechanisms are embedded in the limbic system that interact with endocrine feedback systems to provoke the onset of essential, motivated, life-sustaining behaviors, including feeding and fluid homeostasis. Questions arise about why this state of starvation is maintained in anorexia nervosa patients despite the force of these mechanisms. For a person with AN, attaining and then maintaining a state of extreme thinness may logically be understood as rewarding in itself, of having accomplished an intrapsychic goal. But some recent studies suggest that some physiological mechanisms may help to perpetuate the state of desired starvation separate from sheer willpower. Neural insensitivity to the effects of hunger in women remitted from AN has been reported by Kaye et al. (2020). Javaras and Pizzagalli (2020) summarize the findings of the Kaye group: "Women with remitted anorexia nervosa were generally characterized by lower activation and less positive functional connectivity in response to tastants in the striatal regions." Previously, Oberndorfer et al. (2013) reported studies suggesting that "restricted eating and weight loss occur in AN because of a failure to accurately recognize hunger signals." They posit that in BN, the opposite situation may

prevail. "Overeating in bulimia nervosa could represent an exaggerated perception of hunger signals."

Natural History of Binge Eating Disorder

BED usually occurs at an older age, in the thirties to the fifties (but not excluding younger ages), thus having modest but not inconsequential overlap with AN and more likely BN. Its natural history is less well studied and less well defined than that of AN or BN. Its onset may be insidious, or it may be the consequence of a lifelong struggle with being overweight (figure 1.5). At times BED is the adverse outcome of many

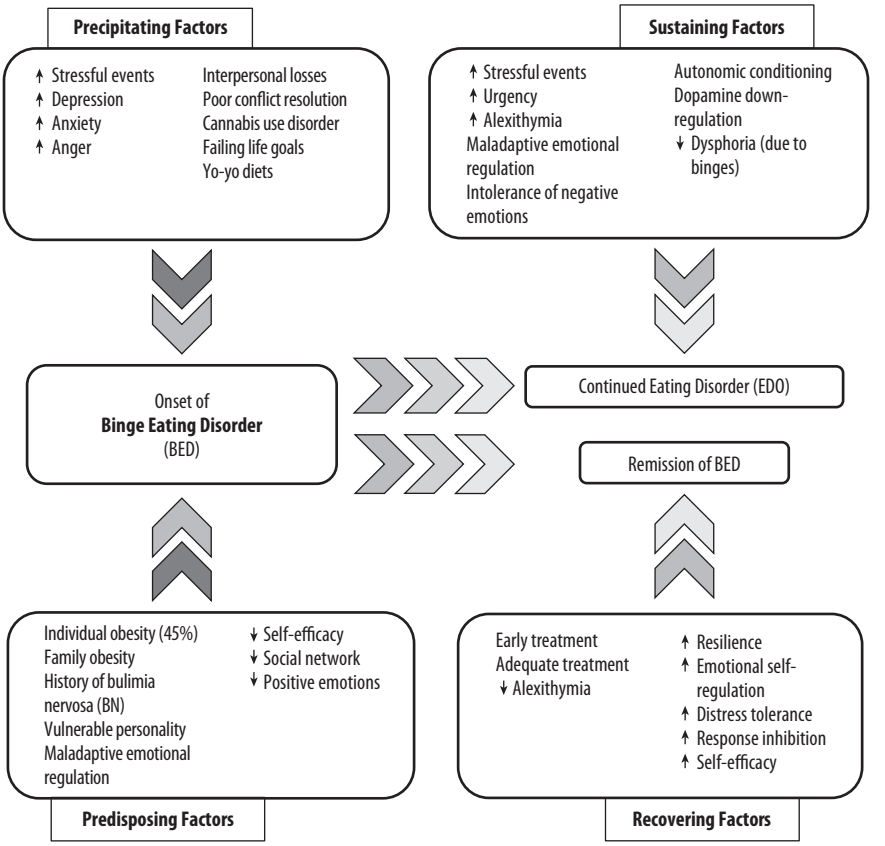

Figure 1.5. Natural history of binge eating disorder. Written by Arnold E. Andersen and created by Kari Miller.

roller-coaster efforts at weight loss through ineffective diets, ending in a demoralized state, which is further worsened by the increasing burden of larger binge episodes, or by the start of new binge episodes.

Some predisposing factors include a family or personal history of medical obesity, and a history of BN. While not meant to be rigidly precise, the generally accepted categories of weights within the body mass index (BMI, kg/m^2) are useful: normal is considered to be roughly from 18.5 to 25; overweight is 25–30; Obese Class I, moderately obese: 30–35; Obese Class II, severely obese: 35–40; Obese Class III, very severely obese: >40. Morbid obesity has a variable definition but generally is considered present when a BMI is 40 or greater. The category of BMI 25–30 has multiple exceptions to being considered significantly overweight. Individuals with a BMI between 25 and 30 with a high percentage of lean muscle mass and low body fat are not truly "overweight." Also, there is some evidence that in otherwise healthy, fit individuals above age 60, the category of being mildly overweight may be somewhat protective. Further, a BMI in the "normal" range but associated with chain smoking, stimulant substance use, or a high percentage of body fat from inactivity is not by itself an indication of being truly healthy.

BED is now the most common eating disorder in some studies, with approximate gender neutrality, estimated to occur in 2% to 4% of the population. It is less obvious than BN, being without purging or any other method of regular compensation to avoid the weight gain from the binge episodes. It tends to present less dramatically than BN and is often not diagnosed until midlife. BED may contribute to approximately 25% of cases of medical obesity. Unfortunately, its diagnosis is overlooked about two-thirds of the time by health care providers because they do not ask certain simple questions, and patients hesitate to bring up their behavioral pattern out of shame and guilt. At times, binge eating precedes the obesity, but the opposite is common as well. Binge eating behavior and, more visibly, obesity are often dismissed by patients, families, and doctors as resulting simply from a lack of willpower.

While binge episodes may be initially calming, they quickly become sources of shame, guilt, and feelings of failure and hopelessness. The binges in BED are generally objectively discernible and not usually sub-

jective, that is, noticeable only to the individual as exceeding their internalized predetermined acceptable amounts of food. The binges in BED may be temporarily concealed in social settings by act of will, but doing so becomes more difficult as time goes on, when the urge to binge or graze becomes relentless. An underlying familial history of obesity often contributes to the initial weight gain over decades.

In one study, 344 of 22,387 survey respondents (1.5%) met *DSM-5* criteria for BED, but only 11 out of 344 (3%) had ever been diagnosed with BED by a health care provider (Citrome, 2019). Primary care clinicians and surgeons with expertise in bariatric surgery should definitely care about making an accurate diagnosis of BED.

Diagnosis of Eating Disorders: Guidelines

A *rule out* approach to the diagnosis of AN or BN by focusing on all possible medical causes leads to delay in treatment and iatrogenic morbidity, as noted previously. Of the several thousand patients with AN that we have treated, in not a single case did we find that a patient with the core psychopathological cognitive distortions and morbid fear of becoming fat had a medical disorder later found to be the true cause of the illness. Medical illnesses by themselves do not cause patients to have a morbid fear of fatness or a relentless drive for thinness. Of course, in less typical patients, such as when a fear of fatness follows weight loss from a medical cause, diagnosis may be more challenging. The diagnosis of BED is becoming more standardized in clinicians treating obesity. The diagnosis of ARFID, in contrast, requires a combination of ruling out certain common causes of these subtypes, as well as a rule-in approach according to the *DSM-5* or ICD-11 criteria. An ARFID diagnosis requires clinical acumen and an awareness of the features of the ARFID categories. An ARFID diagnosis at times will ensure that a patient with complex and unclear origins of abnormal eating patterns, often referred by medical clinicians who have given up on coming to diagnostic certainty, gets appropriate treatment by a psychiatric team. Good interpersonal relations with family physicians and nurse practitioners, and with pediatricians, will facilitate making the correct diagnosis. Pediatric-age cases are common in the ARFID category. My experience has

primarily been with adults from age 18 upward, although cases beginning at age 7 have been evaluated. Therefore, our examples of patients with an ARFID diagnoses are patients of teenage or adult ages.

When extensive medical evaluations do not reveal the cause of significant weight loss, and the patient does not meet criteria for other psychiatric disorders, a diagnosis of ARFID may be considered. An eating and weight disorders inpatient unit can generally evolve an individualized program for nutritional rehabilitation even in the absence of classic symptoms of other eating disorders. These patients frequently fall between the cracks of medical care and psychiatric care but are well served on an eating disorders unit, with good liaison between psychiatry and internal medicine always a goal. Some clinicians might prefer to use the diagnostic categories of other specified feeding or eating disorder (OSFED) or unspecified feeding or eating disorder (UFED) for these enigmatic patients who experience severe weight loss of unknown origin, unexplained vomiting, or only some of the symptoms of more typical cases. Occasionally, we have evaluated and treated patients with unexplained vomiting, checking carefully to rule on emesis gravidarum in pregnant people. Emesis gravidarum may coexist with self-induced vomiting, however, increasing possible adverse consequences to the pregnancy and the mother by the combination of two sources of vomiting. Some challenges to making the diagnosis of an eating disorder are listed in table 1.12.

Comorbid Psychiatric Disorders

Once a diagnosis of an eating disorder has been made, further diagnostic work is essential. Eating disorders typically have one to four co-occurring psychiatric disorders. Comorbid psychiatric disorders may occur either before an eating disorder or as a consequence of an eating disorder. This difference carries importance in planning treatment (table 1.13). The presence of comorbid psychiatric disorders often makes treatment of the eating disorder more complex and may have a substantial influence on the outcome of treatment (Woodside and Staab, 2006). Conversely, some comorbid conditions may improve with intensive treatment of the eating disorder itself.

TABLE 1.12. Challenges to the diagnosis and treatment of an eating disorder

1. Failure to consider that an eating disorder may be present in any patient in primary care.
2. Patient's denial of symptoms.
3. Unawareness or denial in males that an eating disorder may be present.
4. Lack of response of complex medical disorders, especially gastrointestinal disorders, to multiple standard treatment efforts.
5. Concealment of eating disorder–related practices when the eating disorder serves a hidden purpose, such as insulin abuse to counteract binge eating.
6. Lack of motivation in symptomatic patients to seeking evaluation.
7. Using only a rule-out approach to medical symptomatology that may be caused by eating disorders.
8. Narrow stereotypes of who may be vulnerable to developing an eating disorder.
9. Failure to consider binge eating disorder in prebariatric surgery patients.
10. Practicing "CBT-lite" in treatment of eating disorders.
11. Treatment of eating disorders with medications only.
12. Failure to identify comorbid diagnoses in patients with eating disorders.
13. Failure to plan integrative treatment.
14. Lack of recognition of eating disorders in overlooked categories, such as autism spectrum disorders.
15. Parents who fear blame or shame not seeking evaluation of a child.

The diagnosis of an eating disorder is virtually always an exercise in uncovering multiple diagnoses and attempting to differentiate between predisposing and consequential comorbid disorders. And, not uncommonly, and more subtly, psychiatric disorders that precede the eating disorder may simply be intensified by, rather than caused by, the eating disorder. In any case, unless a comorbid disorder is strongly present and interferes with treatment of the eating disorder, it makes clinical sense to first fully treat the medical, weight normalization, psychological, and behavioral components of the eating disorder, especially the cognitive distortions, before treating the companion comorbid disorders. This allows clinicians to determine whether these co-occurring disorders are remitted

TABLE 1.13. Comorbid psychiatric disorders that commonly accompany eating disorders

- Mood disorders (40%–70%): major depressive disorder, dysthymia (persistent depressive disorder), bipolar II (cycles of depression with mild highs). May have seasonal affective disorder subtype.
- Anxiety disorders: generalized anxiety disorder with or without panic; social anxiety disorders
- Obsessive-compulsive disorders (especially in anorexia nervosa)
- Alcohol, marijuana use, and/or other substance abuse (more common in bulimia nervosa or AN-BP), especially methamphetamine, cocaine, or other stimulant drugs
- Personality disorders (full criteria) or vulnerable traits (<2 SD from norm)
  - Traits common in anorexia nervosa: sensitive, perfectionist, persevering, self-critical, anxious
  - Traits common in bulimia nervosa: impulsivity, unstable moods, dramatic behaviors, emotional intensity
  - Traits common in binge eating disorder: high harm avoidance, low self-directedness, difficulties in emotional regulation, low self-esteem
- Pathological gambling
- Autism spectrum disorders

or improved by full treatment of the eating disorder. AN has a substantially increased probability of suicide (Bulik et al., 2008), as does BN.

In our experience, classic food-restricting AN is accompanied, on average, by two additional associated psychiatric diagnoses; AN-BP typically has four associated diagnoses; and BN typically has three (Margolis et al., 1994). BED is often associated with depressive and anxiety disorders as well as a familial and personal history of obesity. Community-based untreated cases of eating disorder may not have as high an incidence of comorbid disorders as those presenting to a specialized clinic, but most individuals with eating disorders who come for treatment at any level will have some form of mood disorder, anxiety disorder, personality disorder, substance use disorder, or obsessive-compulsive disorder (OCD). It is important to emphasize the assessment of comorbid syndromes immediately after diagnosis of the eating disorder because comprehensive treatment and case management depend on a combined and integrative approach. Vulnerabilities of personality, whether traits or disorders, are heightened in most eating disorders but are not randomly distributed. Patients with anorexia nervosa are more likely to be sensitive, persever-

ing, self-critical, and perfectionistic, while patients with bulimia nervosa are more often (but not always) likely to be impulsive and dramatic and to experience unstable moods, especially in emotional intensity disorder, also called borderline personality disorder, an archaic term.

Udo and Grilo (2018; 2019) have extensively studied the comorbid conditions commonly occurring with eating disorders. They report mood disorders and substance use disorders to be the most common comorbid conditions in BED. Other studies on the comorbid psychiatric disorders associated with a diagnosis of BED report depressive and anxiety disorders as co-occurring more than 50% of the time, followed by substance use disorders, with impulse disorders being common as well. The main takeaway point is that while a diagnostic assessment of a patient with an eating disorder begins with reference to the criteria by *DSM-5* or ICD-11, it only concludes with accurate assessment of the comorbid conditions.

Differential Diagnosis: Psychiatric and Medical

Several other psychiatric syndromes imitate anorexia nervosa, primarily when a pattern of food refusal or unexplained weight loss occurs without the core cognitive distortions of AN or BN, but with the diagnostic features of another disorder. The guideline is to diagnose a symptom (weight loss, food refusal) by the company it keeps (low mood, delusions). Some of the most common differential diagnoses within psychiatry include major depressive disorder, delusional disorders, substance use disorders, phobias, and OCD (table 1.14).

Major depressive illness is frequently accompanied by a weight loss of 15–20 pounds. Appetite returns and weight restoration follows treatment with cognitive behavioral therapy, antidepressants, or both; the continued absence of body image distortion or a morbid fear of fatness confirms the correct diagnosis. Patients with obsessive-compulsive disorders or traits may be unable to eat normally because of obsessional ruminations about the nutritional content of food, making decisions about meals difficult. As noted in the ARFID diagnostic discussion, a prior choking episode may have sensitized an individual so that they avoid swallowing food of any kind, which in some patients has led to

TABLE 1.14. Differential diagnosis of eating disorders

Psychiatric Disorders
- Major depressive disorder: often associated with weigh loss >15 lbs
- Obsessive-compulsive disorder: intrusive rumination about content of food
- Paranoid delusions: fear of poisoning in food
- Behavioral sensitization: prior choking episode
- Stimulant substance use disorder overriding normal hunger
- Cannabis-induced overeating

Medical Disorders
- Insulin-dependent diabetes mellitus
- Cystic fibrosis
- Medication-induced weight increase: corticosteroids, atypical neuroleptics
- Occult malignancy

General rule: Diagnose a symptom by the company it keeps.

weight loss as severe as in anorexia nervosa. This disorder is now recognized to fall within the ARFID criteria.

Paranoid states—especially paranoid schizophrenia or a delusional disorder (including delusional disorders secondary to use of substances of abuse, most commonly methamphetamine) in adolescents and young adults with narrowly focused delusions about food being poisoned—may lead to substantial weight loss but be inscrutable to family physicians, as a patient's suspiciousness prevents them from giving the physician full disclosure of the inner delusional process. Treatment with neuroleptics usually leads to increased eating and increased weight in paranoid states, as well as enabling the patient to become more candid in the follow-up mental status examinations about the real reason for avoiding food.

Case Study: Delusional Disorder Presenting as an Eating Disorder

V.O. was a 55-year-old woman referred for evaluation of weight loss from 115 to 85 pounds over two years. Her primary care clinicians found no medical cause to be present and considered the diagnosis to be a probable eating disorder. She was given repeated directions for a weight restoration diet that she did not follow. In the eating and weight disorders clinic, a history revealed features compatible with a delusional disorder. The patient had become suspicious of neighbors, thinking they were spying on her. She closed all the window blinds

tightly and did not venture out into the yard adjacent to the neighbors. She reported a fairly normal mood during the past two years, with only mild episodes of discouragement. She denied any desire to diet.

She also denied any desire to be thinner. She denied any fear of becoming fat, saying she should be at her previous weight, but would not say why she limited her food intake. She reported that the "atmosphere" around her was suspicious. She began to read food labels closely, examining cans and fresh foods for possible signs of contamination. She began to avoid certain types of food without being able to give a reason. She felt colder in the house and began wearing layers of clothing. A history of an aunt who had been admitted to a long-term psychiatric facility was elicited. The patient never experienced hallucinations in any modality. She became increasingly certain she was at risk for being harmed. Her husband was puzzled. A probable diagnosis of a delusional disorder was made. She was started on a neuroleptic and referred to local care for follow-up.

Occasionally, medical disorders may simulate AN in some ways but without the crucial morbid fear of fatness and drive for thinness and without the typical distortion of body image associated with most cases of AN and BN. For example, consider a case of Crohn's disease in a 25-year-old individual referred to the eating and weight disorders clinic who had obsessive-compulsive personality disorder that was manifested by avoidance of all solid food and limitation of caloric intake to sports drinks. This appeared at first glance to be a case of AN. Crohn's disease was also correctly diagnosed after a positive stool evaluation for occult blood was documented; psychiatric assessment noted the absence of typical mental symptoms of anorexia nervosa or any other psychiatric disorder. Medical assessment is vital for understanding the possible causes of any disorder with unexplained weight loss, as well as for decision making about the location, intensity, and methods of treatment, but not for diagnosis of the eating disorder.

Another general guideline bears up well after extensive clinical practice: *A psychiatric disorder is not necessarily present because an extensive medical history and testing have not uncovered a medical diagnosis*. It is preferable

for the medical provider to say that there is no known medical cause than to assume that this means a psychiatric disorder is present. This principle is applicable for psychiatric diagnoses as well. When no psychiatric or medical diagnosis is clearly found to explain the symptoms and signs of loss of appetite, weight loss, early satiety, food refusal, idiosyncratic food choices, unexplained vomiting, or abdominal pain of unexplained origin associated with weight loss, a move to pragmatic treatment with an ARFID diagnosis or a diagnosis of unspecified feeding or eating disorder on an eating disorders unit may ameliorate the unexplained symptoms. It is prudent to accept that there may be a need for admission to a specialized eating disorders unit without being certain of the diagnosis. Admission to an eating disorders unit with supervised refeeding and experienced nutritional prescription, in addition to psychological treatment and continued diagnostic assessment, may benefit these idiopathic cases, regardless of the lack of diagnosis. Doctors at times are better at determining which disorders are not present than what the correct diagnosis is. Treatment with supervised refeeding, psychotherapy, additional efforts at a more inclusive history from the patient and from significant others, and repeated mental status examinations as weight and eating behaviors improve will often reveal the true diagnosis, whether medical or psychiatric.

Vulnerabilities of personality, whether traits or disorders, are increased in all eating disorders but are not randomly distributed. As noted earlier, patients with anorexia nervosa are more likely to be sensitive, persevering, self-critical, and perfectionistic, valuing order and sameness; in contrast, patients with bulimia nervosa or the binge-purge subtype of anorexia nervosa are more likely to demonstrate impulsive, dramatic features, in addition to intense but unstable moods.

Treatment of Eating Disorders: Sequence

Primary care providers can often diagnose eating disorders and, with modest training, treat many early and moderate cases of eating disorders, sometimes in conjunction with colleagues in psychiatry, psychology, nutrition, and social work. After an eating disorder diagnosis and comorbid

psychiatric diagnoses are made, and any reasonable differential diagnoses are considered, the next step is setting goals for treatment.

Safety First: Medical Evaluation

For all patients with eating disorders, the first approach necessarily involves safety. This means correcting all severe, urgent medical disorders; distinguishing between these and self-improving signs and symptoms of medical starvation; and considering strategies for other long-term medical concerns. The medial consequences of AN, the purging abnormalities from BN, and the suicide potential of both AN and BN are the most immediate considerations. Hypothermia, bradycardia, starvation-based hypothyroidism, decreased cardiac size, and increased brain ventricular size, as well as some abnormal labs, are signs and symptoms that in most cases can be passively followed as they gradually improve with the successful restoration of weight in AN and the interruption of binge-purge behaviors in BN (table 1.15). In contrast, arrhythmias other than stable bradycardia, a prolonged QT interval, unstable vital signs, an abnormal state of consciousness, and significantly lowered potassium are among the most common findings indicating a need for urgent intervention.

TABLE 1.15. Urgent/emergent medical signs and symptoms vs. generally self-improving conditions in patients with eating disorders

Urgent/Emergent	Generally Self-Improving	Long-Term
Unstable vital signs	Stable bradycardia	Osteoporosis/osteopenia
Severe starvation (BMI <15)	Decreased cardiac size	Increased brain ventricles
Significant hypokalemia	Hypothermia	Other starvation-related brain changes
Cardiac arrhythmias except stable bradycardia	Moderate orthostasis	Weight redistribution
Prolonged QT interval	Starvation-related hypothyroidism	Sarcopenia
Altered state of consciousness	Amenorrhea	Height restriction Slow GI tract

Incidentally, the preferred phrase to tell patients regarding weight management (all patients with AN and appropriate patients with BN) is that the treatment team is working to *restore weight* to a healthy range, rather than saying that the goal is gaining weight, the latter phrase being fraught with sociocultural tones of becoming fat. For adolescents who have increased in height, stating that the goal is to "catch up" with normal development for age and height is helpful. Not a major point, but why increase the already high fears of becoming fat when an equally accurate phrase can be used?

Weight Restoration, Stabilization, or Reduction

The methods of treatment available to a primary care clinician are low in technology, modest in cost, and yet can be truly effective; treatment is often best accomplished by an outpatient team approach, working with colleagues, such as a dietician, psychologist, or social worker. Patients with AN need to be restored to a full, healthy, individualized weight for multiple reasons; at a minimum, for women, a healthy weight is a weight range that leads to normal, regular menstrual cycles for patients prior to menopause who do not meet criteria for primary amenorrhea. The first signs of menstrual activity are not synonymous with a final healthy weight range, however. An initial weight goal for a very starved individual, based on an individualized history along with possible contributions from insurance tables or pediatric tables, should be written in pencil, not ink, and not necessarily shared with patients until they are well out of danger. Typically, patients are weighed facing backward on the scale, in only night clothing, after voiding, and before eating. A final *goal range* for each patient needs to be individualized by achievement of a cluster of signs of normality, not only a number on the scale. Typically, the return of menses occurs at an average of 92% of healthy weight, meaning do not stop there. Freedom from feeling colder than peers, increasing heart rate from a bradycardic range to a normal rate, and normalization of T3, luteinizing hormone, and estradiol (testosterone in males) are signs of being close to a healthy weight, even if menses are not yet fully restored. In females within the typical age range

of menstruation, the finding of a dominant ovarian follicle on a sonogram supports the fact that the patient is close to a healthy weight range, but she may need additional weight restoration of 5 to 10 pounds. It is important to remember that patients with AN who do not achieve weight restoration may die while having normal laboratory values.

Male AN patients do not have the informative clinical finding of the presence or absence of menses to use as one criterion for estimating the adequacy of weight restoration. In emaciated males, a dimensional increase in sexual drive and fantasies, along with the metrics of increased testosterone and normalization of other laboratory studies, are signs of an approach to a healthy long-term goal range. When in doubt as to whether weight restoration is adequate, follow-up treatment will necessarily include prescribing additional steps to higher, healthier plateaus in weight restoration if indicated by the global signs and symptoms of the disorders. Most AN patients will profess that they are small boned, come from a family of normally low-weight relatives, and request the lowest possible goal weight range.

BN patients may enter treatment below, at, or above a heathy weight range. A personal and family history of weight will help to avoid the pitfall of using only the goal weight ranges found in insurance tables or in textbooks. The clinical challenge occurs when the patient, often a female, clearly belongs not in the narrow goal range of published tables for average age and height but at a higher, healthier, personalized weight. These patients too will insist that they are small boned and belong in the lowest weight range.

Treatment is not limited to achieving a healthy weight range. If weight is the only goal, as too often occurs when patients with anorexia nervosa are treated on general psychiatric, medical, or pediatric units, a revolving door of recurrent admissions often results. Weight improvement without skilled evidence-based psychotherapy is seldom effective in sustaining long-term improvement. For substantially underweight patients, restoration of healthy weight is best achieved with supervised refeeding in conjunction with evidence-based psychotherapy and occasionally with adjunctive psychopharmacology, administered by an experienced team on a specialized unit or subunit. Evidence is mounting

that the strongest predictor of long-term sustained remission of anorexia nervosa is the degree to which fully adequate weight is restored and maintained, in addition to successfully challenging and changing cognitive distortions, as well as improving comorbidities. It is reasonable for an experienced treatment program to safely increase weight by 3 pounds per week for females with AN, and 3–4 pounds for males. A rapid approach to weight restoration (1.98 kg/week for inpatients; 1.36 kg/week for partial-hospital patients) has been demonstrated to be safe and effective (Haynos et al., 2016; Redgrave et al., 2015). The goals of effective treatment include achieving an adequate normal weight, challenging and changing cognitive distortions by effective psychotherapy, prescribing individualized psychopharmacology as needed, and especially requiring an adequate length of treatment, including careful follow-up for a year or more.

Many patients with BN are in the normal weight range, and they may not require any change in body weight. But a good number of BN patients are mildly or moderately underweight, although not in the anorexic range. For BN patients with some degree of lower than healthy weight, restoration to an adequate weight is an essential part of the initial treatment plan, as with fully diagnosed AN, despite some anticipated resistance to weight restoration. In contrast, BN patients with some degree of overweight may urgently request a dieting program at the beginning of the treatment program, but this request should be resisted for two reasons. First, once binge-purge episodes have been interrupted, weight may decrease on its own by elimination of the extra calories from binges. It would be unclear which factor is causing the weight reduction if even a prudent dieting program is initiated along with the initial treatment of the binge-purge episodes. Second, it takes time to assess what a healthy maintenance weight would be for some patients until it is clear how well the patient defends a weight achieved by remission from any binge-purge episodes.

BED seldom requires inpatient treatment; partial hospital may offer greater benefit than a once-a-week outpatient program. As with overweight or mildly obese BN patients, those with BED are best served by first interrupting binge episodes for an extended period. BED generally

responds to a combination of treatment methods, but the first essential goal is to interrupt the binge eating (Wilson, 2011). At that time, attention may next be paid to teaching about and following progress in a prescribed prudent dieting program, such as the Mediterranean diet. BED patients who have a strong family history of obesity will generally need to have an individualized healthy weight goal determined because their healthy maintenance weight may be in a range above a BMI of 25, the putative top of a healthy range. Going lower than a weight that can be sustained without intolerable hunger, hypothermia, or other signs of starvation is an invitation to the return of binge eating, even if the emotional components of the binge episodes have been identified and diminished. At times, when patients with binge eating disorder who are obese do go on to attain a goal within a BMI range of 19–25, they may suffer from significant symptoms of starvation typical of anorexia nervosa. As little as a 10% loss of weight can substantially improve the medical abnormalities associated with obesity—for example, for some patients, achieving and maintaining a BMI of about 30, by moderate reduction in weight, may be all that is necessary for accomplishing medical and psychiatric goals.

While fewer studies of treatment outcome of ARFID patients are available, one report by Sharp et al. (2020) documents a positive result of treatment in 81 patients, with ages ranging from 10 months to about 19 years. Inclusion criteria required dependence on enteral feeding or inadequate oral intake. Treatment consisted of a combination of behavioral intervention and parent training with nutritional therapy and medical oversight. All patients had complex medical, behavioral, or developmental histories with long-standing feeding problems averaging 33 months in duration. At discharge, oral intake had improved by 70.5%, and 33% were completely weaned from tube feeding. At follow-up, 58 patients (72%) were weaned from tube feeding.

Psychological Treatment: Challenging and Changing Overvalued Beliefs

The most validated and evidence-based psychotherapeutic treatment for AN, BN, and BED is cognitive behavioral therapy (CBT in its multiple

iterations and subtypes), followed by interpersonal therapy and dialectical behavioral therapy, all sharing core features of principles and practices. All attempt to fundamentally change the internal factors perpetuating these eating disorders. In fact, according to the British NICE guidelines for evidence-based treatments (National Institute for Health and Care Excellence), CBT (Dalle Grave et al., 2016; Fairburn, 2008) is the recommended first mode of treatment for patients with bulimia nervosa. Unfortunately, rigorous application of CBT is more often promised than delivered. For both AN and BN, a central goal of CBT is *persuading patients to think differently* about their overvalued desire for extreme thinness, with the consequent need for food restriction, letting go of the desire for achievement of an unrealistic, excessively thin body. A central overarching treatment goal is to help patients find personalized ways other than self-starving or binge-purge behaviors to deal with developmental or psychosocial challenges, dysphoric moods (especially depression and anxiety), unhealthy social and peer norms, crises in relationships, and low self-esteem.

The first component of CBT involves psychoeducation concerning both the medical and the psychosocial hazards of starvation and binge-purge behaviors, as well as an outline about how CBT functions in achieving healthy goals. The patient is invited to become a *coinvestigator* in understanding their eating disorder and learning to challenge fundamental misbeliefs with empirical unbiased evidence. The second goal is identifying, and then challenging and changing, distorted cognitions, especially overvaluing the benefits of thinness; the excessive, or morbid, fear of fatness; the presence of a distorted body image; and the need for purging, as in the binge-purge subtype of AN or in BN. These changes are brought about by guiding the patient in gathering evidence to support or refute the underlying cognitive distortions, which together are called *schema*. Third, as a consequence of replacing cognitive distortions with healthy thinking about weight, shape, food, and exercise, the patient is guided into healthy patterns of behavior, including the capacity to eat in all social situations, shopping for and preparing food, shopping for clothing, and attending social and vocational functions involving food. These final goals are best achieved by hands-on experi-

ential methods, often with the guidance of an activities therapist. The clinician should think of eating disorders not only as an *illness* but also as a *strategy that needs to be replaced by healthy alternatives*.

Studies of the benefits of CBT and weight restoration for patients with severe starvation from anorexia nervosa (in contrast to patients with BN) lack truly randomized controlled trials, given the obvious ethical prohibition of allowing an untreated placebo group of severely starved patients to further lose weight or die to contrast with the active treatment group receiving intensive CBT. Despite the lack of formal studies in severely ill AN patients, the evidence supporting the necessity and benefit of combining weight restoration with CBT for people with AN is persuasive. Randomized controlled trials of mildly ill patients with anorexia nervosa without serious medical complications might be undertaken with groups assigned to CBT plus weight restoration, CBT alone, weight restoration alone, and no treatment (for a limited period). Enhanced CBT has been shown to be effective for outpatients (Fairburn et al., 2013).

Interpersonal psychotherapy (IPT) and dialectical behavioral therapy (DBT) share many features with CBT and are potentially effective agents of change, especially with some subgroups of patients, such as those with comorbid severe personality disorders or suicidality. DBT has emphasized, among its other strategies, the cultivation of distress tolerance followed by response inhibition. Distress tolerance involves being "with" the symptom or urge for long enough to give it a name and decrease its power to drive an immediate response. Response inhibition, abstaining from the usual illness-based abnormal behavior, then gives the patient time to decide which healthier method can be employed to deal with the emotional symptom or urge by choosing a healthy response from options reviewed and decided on in preparatory psychotherapy sessions instead of the previous illness-based response, such as purging, self-cutting, or substance use. IPT is especially effective in eating disordered patients with distressed and dysfunctional relationships.

CBT appears to be moderately effective in the treatment of BED (Ghaderi et al., 2018), with more modest support for IPT. CBT and its analogs may be more useful as well in their application to comorbid disorders or syndromes accompanying AN, BN, and BED.

The choice of a psychotherapeutic method for ARFID patients is necessarily individualized and varied because of the heterogeneous nature of these diagnoses. Behavioral therapies may play a major role, especially in patients who have diminished normal eating behavior because of past experiences causing arousal of fear around the process of normal eating, such as past choking episodes. Patients with aversion to eating because of the sensory qualities of food may also respond to individualized behavioral strategies of desensitization. Behavioral methods combined with age-appropriate psychoeducation and, at times, evidence-based psychotherapies may prove effective for those old enough to take part meaningfully. For younger patients, families are generally included in some or all of the treatment. While the general strategies of CBT or IPT may be helpful and effective in ARFID patients, the specifics of which cognitive distortions are operative will not necessarily fall into the area of those distortions most common with AN and BN patients. An ARFID diagnosis will often allow patients who otherwise might fall between the cracks of medicine and psychiatry to obtain necessary treatment.

Integrative Treatment of Eating Disorders with Comorbid Psychiatric Disorders

Treatment of disorders that are comorbid with eating disorders may be more challenging than treating the eating disorder itself. It is helpful, therefore, to have an overall strategy and viewpoint about the treatment of comorbid psychiatric disorders when treating a well-defined eating disorder. Generally, treatment of eating disorders is a "package deal," meaning treatment must involve an appreciation of these comorbid conditions and requires an integrative plan. An important fact in decision making about comorbidity is to ascertain whether the comorbid condition existed prior to the eating disorder, and if it did, whether the intensity of the comorbid disorder increased as a result of the eating disorder. Psychiatric comorbidity has been shown to be a risk factor for the mortality of people with bulimia nervosa (Himmerich, Hotopf, et al. 2019).

A depressive disorder, for example, may have been clearly present before an eating disorder was consolidated; the depressive disorder may then be intensified by the eating disorder, or it may have only presented itself after the eating disorder was established. In the latter case, the best approach is to fully treat the eating disorder and then reevaluate the degree, if any, to which the depressive disorder persists. When an antidepressant is begun at the start of treatment of the eating disorder, it is not possible to know what has produced remission of the eating disorder. When a depressive disorder was clearly present before the eating disorder, often in the context of a positive family history, then antidepressant therapy early on may be prudent. Some general guidelines follow.

Depressive Disorders

For the treatment of depressive disorders, any SSRI (fluoxetine, sertraline, or escitalopram) appears to be equally effective. Tertiary antidepressants, such as venlafaxine and duloxetine, acting on noradrenergic as well as serotoninergic (and probably some dopaminergic) sites, may be used as a second choice for patients with treatment-resistant bulimia nervosa or for binge eating disorders.

We recommend starting with one-half the usual minimum maintenance dose for 1 or 2 weeks, then increasing to the typical starting dosage for 6 weeks; for example, beginning with 10 mg of fluoxetine, then increasing gradually to 40–60 mg. Typical ranges of other SSRIs are 50–150 mg of sertraline and 10–20 mg of escitalopram, beginning with lower doses. Where a seasonal worsening of depression is evident in the lower-light months of fall and winter (not uncommon in BN patients), the addition of bright light therapy (10,000 lux for one half hour at 18–22 inches each morning) will add incremental benefit. Occasionally, a mood stabilizer is helpful for bipolar II disorders. CBT has the additional virtue of being effective not only for eating disorders but for many mild to moderate depressive illnesses. First-generation antidepressants such as nortriptyline may produce QT prolongation, and bupropion is generally contraindicated because of the report of seizures in patients with bulimia. Recent studies have suggested that if lab values, especially

potassium, are in a normal range, and other factors suggest a beneficial response to bupropion might be anticipated, contraindication may not be necessary.

Anxiety Disorders

Panic disorder responds to many of the same treatments as depressive illnesses—SSRIs or SNRIs plus CBT—with additional psychoeducation about the nature of panic symptoms and generalized anxiety. Generalized anxiety is less responsive to medication but does improve, though more slowly, with CBT, behavioral relaxation techniques, mindfulness, and sometimes cautious prescription of short-term benzodiazepines for individuals without substance abuse histories.

The claim that eating disorders are a form of obsessive-compulsive disorder is an overstatement and may reflect a lack of differentiation between obsessive-compulsive traits and disorders. When true OCD exists after improvement in the eating disorder behaviors and thinking, most SSRIs (sometimes at higher than antidepressant doses) are equally effective, although some clinicians believe fluvoxamine is more specific for OCD. There are some contraindications in using fluvoxamine with concomitant prescription of certain antibiotics or antifungals, or in the presence of prolonged QT intervals. Obsessive-compulsive traits may be heightened by self-starvation; these traits respond more slowly to psychological treatments, without any proven benefit from use of medication alone.

Comorbid Substance Abuse

The first goal is to assess the role of substance abuse in the eating disorder. In perhaps one-third to one-half of teenage girls with comorbid substance abuse, the substance abuse is often closely related either to the onset or to self-treatment of the eating disorder. Cannabis use may provoke binge eating, which, once initiated, may go on to have a life of its own. Alcohol may be used for self-treatment or may be provocative to an eating disorder. Some individuals with AN can allow themselves to eat more than minimal amounts of daytime food only when they have had several drinks just before bedtime. Alcohol may also do the opposite by

provoking binge episodes through a decrease in dietary restraint, followed by purging. If there is no clear amelioration of the substance craving despite treatment of the eating disorder, then referral to inpatient or outpatient drug rehabilitation, after treatment of the eating disorder, is the optimal approach. The initial treatment of this dual diagnosis is usually best accomplished by first engaging in an eating disorder program because of the general capability of such programs to treat combined psychiatric disorders. Substance abuse treatment programs generally do not treat the eating disorder effectively. Nicotine withdrawal can be accomplished skillfully without any feared increase in weight.

Personality Disorders

The guideline here is long-term management according to the type of personality disorder. Patients with sensitive, persevering anxious features (often associated with the restricting subtype of AN) generally respond to support, education, and CBT as part of their eating disorder treatment. Repeated confrontation with feared situations is often effective in social anxiety disorders. A common and sometimes challenging combination is the association of a borderline personality disorder with an eating disorder, statistically more likely to be BN. These individuals respond best to care by skilled, experienced group and individual therapists with evidence-based programs in emotional management, such as DBT or the STEPPS program.

True schizophrenia almost never occurs with a diagnosis of an eating disorder. Too often, soft diagnoses of schizophrenia have incorrectly been suggested as comorbid disorders.

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No medication by itself is necessary or sufficient for the treatment of weight loss in most patients with AN, although some medications may be helpful in selected cases for various reasons. For 50 years, medications to increase appetite in AN patients have been prescribed by psychopharmacologically oriented physicians who act much like the carpenter for whom the whole world is a nail: nail everything. When psychopharmacological agents are prescribed because of the clinician's training

limitations, lack of patience for adequate psychotherapy, unrealistic length-of-stay goals imposed by administrators or health plan bureaucracies, the treatment often fails. Crisp (1995) and others have shown that normal appetite is returned with only a few weeks of simple, balanced nutrition. The only "medication" that truly works is food with supervised refeeding. That being said, a new wave of adjunctive pharmacological methods to improve the low weight in AN has featured second-generation neuroleptics, especially olanzapine. Attia et al. (2019) documented "a modest therapeutic effect of olanzapine compared with placed on weight in adult outpatients with AN, but no significant benefit for psychological symptoms."

Psychopharmacology in BED

The major problem with treatment of BED is the failure to recognize it in patients or to treat it adequately. BED treatment studies have proceeded in various directions, psychotherapeutic and pharmacological, with variable success, mostly confirming the effectiveness of CBT along with cautious endorsement of achieving possible benefit from stimulant medications. Hilbert, Petroff, et al. (2019) summarize recent findings after reviewing 81 studies of 7,515 total individuals with BED. "In RCT's with inactive control groups, psychotherapy, mostly consisting of cognitive behavioral therapy, showed large-size effects for the reduction of binge-eating episodes and abstinence from binge-eating, followed by structured self-help treatment with medium to large effects when compared to wait list. Pharmacotherapy and pharmacological weight loss treatment mostly outperformed pill placebo conditions with small effects on binge-eating outcome." The Swedish Council on Health Technology Assessment (2016) found good results with IPT as well.

Lisdexamfetamine has been approved by the FDA for the treatment of moderate to severe BED. Studies have cautiously endorsed its use in clinical trials for BED adults. Heo and Duggan (2017) showed that lisdexamfetamine 50–70 mg per day was more effective than placebo and generally well tolerated. Dasotraline, a dopamine and norepinephrine

reuptake inhibitor, has shown some promise, but studies need to be continued, and it is not yet approved (Appolinario, Nardi, and McElroy, 2019). The use of stimulant medications for BED raises at least three cautions: (1) the side effects of stimulants are well known but must be carefully followed, including awareness of cardiac arrhythmias, insomnia, and anxiety; (2) if no concurrent effective evidence-based psychotherapy is part of the treatment program, then the implication is that the stimulant must be given for a lifetime; (3) the stimulant is not approved for weight loss but may be misused for that purpose, not only for interruption of binge eating. Nevertheless, in selected patients, lisdexamfetamine may be of use. More studies on dasotraline are needed.

Naltrexone has been found to be safe, well tolerated, and moderately effective in decreasing the binge-purge behavior of adolescent patients, suggesting an overlap with addictive disorders. It appears that blocking opioid receptors diminishes food intake. Studies of the effect of naltrexone on BED have generally combined naltrexone with bupropion. Determination of the most effective long-term psychopharmacological agents for the treatment of BED, with or without adjunctive psychotherapy, are under way, but no medications have been universally accepted as the standard of care, which is not to deny that they may offer some benefit for individual patients. Topiramate has also been advocated.

Behavioral Strategies for Eating Disorders

Healthy lifetime patterns of physical exercise are necessary to get patients who have engaged in driven, compulsive exercise to moderate their regimens. In contrast, for the common "couch potatoes," especially overweight patients with BED, there is great need to initiate and sustain physical activity in a progressive, guided manner. Supervised strength training is of special benefit to males with eating disorders, for whom achieving both muscle mass and muscle definition is important to their return to a more normative male role in society, with increased self-confidence and a healthy body image. Female patients are of course welcomed as well.

Innovative supplemental behavioral measures include directing patients to throw out the bathroom scale at home and to cover mirrors, except for enough visual reflection to check hair and makeup. Mirror gazing and frequent weighing are nearly universal in patients with eating disorders. The figure seen in the mirror is seen through the eyes of body image distortion and seldom corresponds to objective measurements. The overall methods of treatment are summarized in table 1.16. In our inpatient program, patients are escorted to the physical therapy area, where they change into bathing suits to learn to relax in a warm hot tub with gentle water circulation; they then leave to record their experiences, including walking past mirrors, usually with surprisingly positive results. Above all, effective behavioral strategies must be applicable to real-life situations. Real-life normative activities are carried out until proficiency is attained, including shopping for food, preparing food, eating food in groups with social conversation, shopping for clothing, and returning in gradually increasing intervals to school or work. Patients of school age continue their academic work without difficulty through a liaison with schools to provide books and lesson plans. No inpatient of ours has missed advancement in school grade level because of their eating disorder. College-age students are provided with a medical statement of treatment duration (without mention of a diagnosis), which enables the student to withdraw from the semester or get a deferred grade.

Overall Strategies for BED

BED seldom requires inpatient treatment. It generally responds to a combination of treatment methods, but the first essential goal is to interrupt the binge eating (Wilson, 2011). Even an intelligently directed gradual weight loss plan cannot be accomplished in the presence of continued binge eating. Normalization of eating behaviors takes priority over loss of weight.

Along with interrupting binge eating behavior, concurrent CBT (or, if indicated, IPT or DBT) is at the core of the treatment of BED along with

TABLE 1.16. Summary of methods of psychiatric treatment for eating disorders

- *Evidence-based psychotherapies*: CBT is the most demonstrated evidence-based form of psychotherapy for eating disorders, but IPT and DBT may also be considered for selected patients. These methods teach patients to challenge, through evidence gathering, their overvalued beliefs about the benefits of slimness and shape change. Interpersonal therapy (IPT) is slower, but its relationship focus may be effective. Dialectical behavioral therapy (DBT) may be an alternative for bulimia nervosa comorbid with borderline personality disorder, teaching stress tolerance and alternative response. CBT is required in Great Britain as a first treatment for bulimia nervosa.
- *Psychopharmacology*: If no experienced cognitive behavioral therapist is available, SSRIs (e.g., fluoxetine, sertraline, escitalopram) or SNRIs (venlafaxine, duloxetine) may decrease binge-purge symptoms by 50%, but patients often relapse. They are prescribed especially for persistent depressive symptoms as a supplement to CBT. Antidepressants or antibinge medication (lisdexamfetamine) combined with evidence-based therapies may be effective in BED.
- *Individualized psychotherapy*: A key goal is to help the patient understand that the core underlying issues in treatment are not weight loss or food but strategies to manage emotional distress, identity formation, and self-esteem, for which the eating disorder is a pseudo-solution.
- *Education of loved ones*: Parents and significant others should be provided with psychoeducation about the nature of the eating disorder and companion disorders. They need empathy and coping strategies for preventing burnout while caring for individuals with chronic illness and for maintaining healthy family relationships without being monitors or "food police." Resolution of family functioning issues can be identified in family therapy.
- *Premature discharge avoidance*: Discharging a patient below healthy weight or with persisting binge eating for AN and BN is demonstrated to increase relapse and require repeat admissions.
- *Family-based outpatient treatment*: For adolescents with mild or moderate anorexia nervosa (preliminary support for bulimia nervosa), family-based treatment can be effective, as demonstrated by Lock and Le Grange (2013). The family is empowered to act as the treatment team. Inpatient hospitalization may be avoided.
- *Careful treatment sequence*: Consider waiting until after normal weight and cognition are achieved before prescribing separate treatment for comorbid disorders. Comorbid psychiatric diagnoses may remit or diminish with full treatment of the eating disorder.
- *Behavioral strategies*: Shopping for food, preparing food, social eating, shopping for clothing, daily diary for thoughts and mood, graduated return to work or school, mindfulness, relaxation methods.
- *Involuntary treatment*: If vital for life or from inability to care for self, involuntary treatment has been demonstrated to improve patients' condition, generally without subsequent litigation.
- *Adequate follow-up*: Usually for 1–2 years, gradually decreasing in intensity and frequency.
- *Continued interaction*: The patient and treatment team should continue to interact with primary care providers for coordinated care.

individualized selection of appropriate medications. After binge eating has been substantially interrupted, and CBT methods have challenged and changed cognitive distortions, only then, if body weight is associated with medical abnormalities, can safe, slow weight loss be attempted.

At times, when patients with binge eating disorder who are obese go on to attain a goal within a BMI range of 19–25, they may suffer from mild or significant symptoms of starvation. As little as a 10% loss of weight can substantially improve the medical abnormalities associated with obesity—for example, by achieving and maintaining a BMI of about 30. The first component of weight that is lost is usually the excessive weight gained through binge eating. The next weight loss challenge often means an attack on the second source of obesity: the bedrock of familial, genetic obesity that is often present. Where genetically predisposed obesity is present, more complex methods beyond CBT are necessary. Moderate regular exercise to increase muscle mass has a goal of decreasing body fat and increasing cardiopulmonary fitness, not weight loss. Weight loss is achieved at the dining table, while fitness is achieved by increasing physical activities, by both aerobic activities and strength training. Additional supplemental goals in the comprehensive treatment of BED include learning assertiveness, practicing distress tolerance, learning response inhibition, management of stress, and mindfulness.

Levels of Treatment Intensity

Some of the most common reasons for referral to hospital-based treatment (inpatient programs or partial hospital) for an eating disorder are listed in table 1.17. In general, most persons with entrenched severe AN will require some form of hospitalization, either in 24-hour inpatient/residential care or in a partial-hospital (full-day treatment) program. Most patients with BN can generally be treated as outpatients, with exceptions for those with significant medical or psychiatric comorbidity. Medically urgent conditions in BN include very low potassium levels or cardiac arrhythmias. BN complicated with suicidality, self-harm, or substance use disorders may also require admission.

TABLE 1.17. Criteria for inpatient/residential/partial (day) hospitalization for patients with eating disorders

- Moderate to severe anorexia nervosa: some hospitalization (full or partial hospital) is generally required. (Exception: family-based treatment in home for teens with intact families willing to learn Locke and Le Grange family or parent-based methods.) Hospitalization is usually needed for *substantial or rapid* weight loss, especially weight loss below 75% of normal age-appropriate weight; sooner rather than later treatment is needed if weight loss is rapid in adolescents. Also needed if mildly to moderately underweight patients with anorexia nervosa, bulimic subtype, have significant cooccurring medical symptomatology (low potassium, unstable vital signs, uncontrolled insulin abuse).
- Bulimia nervosa: outpatient programs are often adequate, except for patients with severe depression, self-harming behavior, suicide plans, or severe medical complications (hypokalemia, etc.).
- Significant psychiatric comorbidity, especially if impairing outpatient treatment progress, includes suicidality, borderline personality disorder with self-harming behaviors, substance abuse, intrusive comorbid OCD, or severe major depressive disorder.
- Lack of response to outpatient treatment after good effort (continued high levels of binge-purge behaviors, lack of reasonable weekly weight restoration).
- Hostile living situation or lack of experienced local therapists.
- Diagnostic uncertainty, in the presence of probable eating disorder and severe medical symptoms.
- Presence of a life-threatening situation (refusal of treatment despite severe weight loss, hypokalemia, suicidality), in which case involuntary treatment may be necessary. Involuntary treatment is as effective as voluntary treatment; generally, insight returns in about 2–3 weeks.

Note: Treatment on a unit dedicated to eating disorders, with a comprehensive, experienced team-oriented approach, is more effective than treatment on a general unit with inexperienced staff.

Compulsory Treatment

Occasionally, compulsory treatment by court order for AN may be life-saving (Elzakkers et al., 2014). While controversial, evidence and logic clearly support the fact that when patients are severely ill, especially with AN, they often lose any capacity to make decisions in their own best interest. Often, patients who are initially treated by court order within 2 to 3 weeks come to recognize how starved they were and gradually drop their resistance to further treatment.

Comprehensive Treatment vs. Inadequate Treatment

Comprehensive treatment with insistence on attaining a healthy, fully normal, individualized weight range, whether by team-oriented

inpatient treatment or family-based treatment, leads to fewer rehospitalizations (Baran, Weltzin, and Kaye, 1995) and, in the long run, is more cost effective (Crow and Nyman, 2004). Managed care has made ruthless inroads in length of stay for psychiatric disorders in general and eating disorders in particular. Similar decreases in adequate treatment for breast cancer or type 1 diabetes mellitus would lead to an outcry resulting in legislative action.

Once initial safety is assured, diagnostic assessment is completed, and goal setting are accomplished in inpatient programs, planning for aftercare and relapse prevention starts on the day of admission. With hospitalized patients under 18 years of age, treatment of the family unit as a whole during and after discharge has produced more sustained improvement and added to greater prevention of relapse than treatment of the teenager alone. The pathways to choices of treatment intensity based on the severity of illness and age are summarized schematically in figure 1.6.

The addition to the *DSM-5* criteria of weight ranges within a diagnosis of AN offers helpful guidance to the intensity of treatment most typically suited to the patient. When a patient with a BMI <14.0, roughly corresponding to weight that is <70% of ideal body weight (IBW), the patient will ideally be treated first in a Center of Excellence for medical stabilization. Centers of Excellence are facilities with widely recognized and extensive experience in the multidisciplinary treatment of the most severe cases of eating disorders, with published results. While patients with an ARFID diagnosis in *DSM-5* are not stratified into weight categories akin to AN patients, appropriate referrals based on weight ranges similar to those for AN, in conjunction with other clinical factors such as the rapidity of weight loss and psychiatric comorbidity, are reasonable components for decision making about level of care. In future iterations of *DSM*, weight categories associated with the ARFID diagnosis may be found useful.

In general, if patients' weight is between 70%–84% of IBW, they are best served in an inpatient or residential treatment center. For weights that are 85%–95% of the IBW, partial hospitalization programs will often be sufficient. These guidelines to the intensity of treatment are not absolutes. The frequency of purging, psychiatric comorbidity, and other

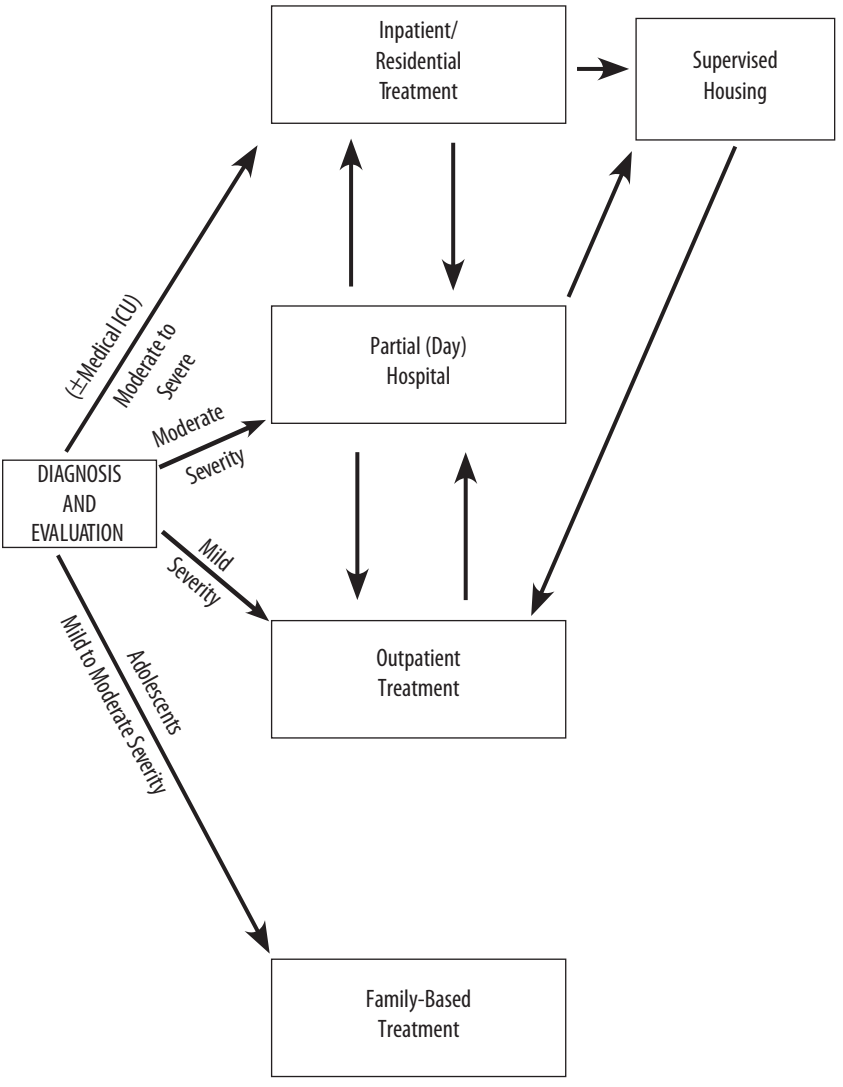

Figure 1.6. Referral to treatment levels after diagnosis

medical and familial factors will further direct the choice of level of care. Garber (2018) notes that "skinniness," aka presentation weight, is not sufficient to assess malnutrition in patients with restrictive eating disorders across a range of body weights. Determining which level of care a patient with an eating disorder needs can be one of the most challenging aspects of referring to treatment.

Between a patient's resistance and the body's ability to feign stability, it is, at times, easiest to refer a patient to the least intensive level of care. In the past, reliance has been placed on the APA guidelines (2006) for decision making about the levels of care between outpatient and inpatient treatment, but these guidelines are deficient in specifying the necessary medical treatment for patients with eating disorders. The severity of the medical disorders of patients at a seemingly normal or higher than normal BMI may be falsely assumed to be lower than they actually are. These patients may present with a falsely elevated weight or be at risk for dangerous complications due to excessive purging behaviors followed by abrupt cessation of purging. Clinical assessment of patients with eating disorders requires an understanding that eating disorders are not only behavioral but also potentially serious medical conditions.

Mortality and Outcome Data

The long-term death rate for patients with eating disorders, especially AN and BN, is one of the highest of all psychiatric disorders, on a par with substance use disorders, especially opioid use. Half of deaths from AN are due to starvation and half to suicide. Deaths are spread out in a continuing pattern from the onset of illness throughout the years of illness, rather than concentrated at one predictable time. Recently, this death rate has decreased, but it is still high in most outcome studies, with death rates gradually accumulating over the years of illness. Deaths from BN are most often associated with cardiovascular or metabolic consequences, as well as suicide (table 1.18). A Danish study has found that the

TABLE 1.18. Causes of death in eating disorders

▪ Anorexia nervosa: medical complications of starvation; hypokalemia in AN-BP subtype; suicide.
▪ Bulimia nervosa: hypokalemia causing cardiac arrhythmia; suicide.
▪ Binge eating disorder: death seldom related to this eating disorder alone.
▪ Mortality in subtypes within ARFID category not well known.

Note: Eating disorders have the highest mortality rate of any psychiatric disorder.

peak hazard for suicide attempts and mortality in AN patients is at age 15 years; for BN, age 22 years (Zerwas et al., 2015).

A few studies, such as that of Strober, Freeman, and Morrell (1997), have reported no deaths in their long-term outcomes for young patients with AN who have been comprehensively treated. In addition, they have documented remission in more than 75% of these patients at 10-year follow-up, demonstrating that full, sustained remission is achievable for patients with AN. Death in patients with BN is generally due to arrhythmias or suicide. Death from the eating disorder is uncommon in BED.

A recent study by Dobrescu et al. (2019) followed patients with AN having an onset during adolescence over a 30-year period, with 96% participation and no deaths. Sixty-four percent had full eating disorder symptom recovery. One out of five had a chronic disorder. Quadflieg and Fichter (2019) assessed the outcome in 1,351 patients with bulimia nervosa. They found a remission of 38% after 11 years and 42% in a subsample after 21 years. Fourteen percent crossed over to another eating disorder diagnosis. Most of the outcome studies of BED have been short term, a year typically. Both evidence-based therapy and pharmacotherapy outperform placebo therapy, but many treatments are of moderate quality. Unfortunately, screening for BED in bariatric clinics varies from 2% to 50%. Studies of bariatric surgery outcomes have uniformly found improvements in hypertension and diabetes, as well as all-cause mortality, with greater improvement in adolescents. Randomized long-term studies of pharmacotherapy alone, CBT alone, or a combination of both versus bariatric surgery in patients with BED in remission are not available.

The goals of treatment for all eating disorders must be reasonable and achievable. A healthy weight range, normal patterns of eating, and healthy thinking about food, weight, and shape are achievable. Being mildly concerned about weight and shape is culturally normative and not inconsistent with otherwise successful treatment. Rodin, Silberstein, and Striegel-Moore (1985) have called this phenomenon "normative discontent," which means having a slight overconcern about dieting and becoming slimmer that does not cause a disorder that meets full or subsyndromal diagnostic criteria, is not causing functional impairment, and has not become significantly preoccupying in the individual's life. Giving lip service to wanting

to be thinner is an almost mandatory part of the female role in western society. There is no guarantee that individuals with bulimia will be free from *urges* to binge or purge even after adequate treatment. What is completely possible is to learn to redirect binge or purge urges so that the individual never carries out binge or purge *behaviors* and chooses healthy alternatives instead. Urges are suggestions, not commands. Patients are on the way to wellness when they realize that the crucial issues in their lives are not food, weight, or shape but important matters in living, relationships, mood regulation, and human development.

Studies have demonstrated that AN is associated with variable changes in the brain. The significance of many of these changes is actively being studied. The brain changes noted in AN are not so clearly correlated with specific symptoms as would be the case, for example, with a stroke causing contralateral paralysis. In severe cases, the brain may appear on magnetic resonance imaging (MRI) to be indistinguishable from the brain of a person with Alzheimer's disease. Ventricles are enlarged, and cortical substance is decreased. While patients with AN often have a surprising degree of accomplishment in school, as weight loss becomes severe, they become increasingly unable to attend and to concentrate on written materials or sustain reasoning. In general, the last functions a patient gives up are school or work. Of concern is the demonstration that weight improvement is not immediately associated with complete restoration of normality in an MRI brain scan, especially in the gray matter. Work is under way in positron emission tomography, tension diffusion studies, and other brain imaging methods to localize the specific brain regions most affected by starvation so as to determine their response to treatment. Defining brain loci that may be the most vulnerable to weight loss, or are predisposing to the development of eating disorders, is a task for continuing research.

Treatment by Primary Care Physicians vs. Treatment at Specialized Programs

Persons with serious, chronic, or complex cases of eating disorders, especially with serious medical symptoms or multiple psychiatric comor-

bidities, usually do best when referred to programs experienced with the diagnosis and treatment of eating disorders, especially programs with a multidisciplinary, unified team approach. The benefits versus risks of referral, however, need to be weighed. Long-term continuity of treatment with an established primary care physician argues for keeping treatment as local as possible. But when weight is not restored steadily (at a rate of more than 1–2 lb/week) in a patient with AN who is receiving outpatient treatment by a primary care provider, or when binge-purge symptoms are not substantially improved in 6 to 8 weeks, then referral to a specialized program, outpatient or inpatient, is strongly suggested.

The success of treatment is maximized by combining the skills of a primary care provider with concurrent psychotherapy by a psychologically trained colleague, whether a psychiatrist, social worker, psychologist, or nurse practitioner. This partnership allows the primary care physician to monitor and treat the medical symptomatology and maintain a long-term relationship with the patient, while the psychologically trained colleague treats the core psychological dysfunction. One study (Volpe et al., 2019) documented that a variety of clinicians typically refer patients to specialist care, including general practitioners (25%), psychiatrists (18%), clinical nutritionists (17%), and others. If motivated and educated in basic psychotherapy methods, the primary care clinician may carry out both the medical management and the psychotherapeutic treatment. This may be especially important when OB-GYN specialists also act as primary care providers, which is often the case for women between the ages of 15 and 50 (Andersen and Ryan, 2009).

Genetics and Neurobiology in Eating Disorders

Multiple studies document that genetics plays a substantial role in the onset and maintenance of eating disorders (Himmerich, Bentley, et al., 2019; Yao et al., 2019). This finding may be of comfort to families who have been accused of causing the eating disorder or have blamed themselves. Evidence that eating disorders include a genetic predisposition may be supportive of reimbursement as well as alleviating family guilt. The genetic studies of AN and BN provide evidence that eating disorders

are serious genetically predisposed disorders, with both psychiatric and medical consequences. The strongest support for genetics playing a role in eating disorders comes from twin studies and family studies. Family studies involve taking an extended family pedigree with identification of all psychiatric disorders suffered by family members, a top-down approach. Heritability of eating disorders along with depressive or anxiety disorders is frequently linked.

Family studies show that an individual with a family history of the mood disorders spectrum, including depressive disorders, bipolar disorders, anxiety disorders, OCD, and substance abuse disorders, has a substantially increased probability of developing an eating disorder. Working from the family history method leaves no doubt that a predisposing genetic vulnerability to developing an eating disorder exists (Brandys et al., 2015; Scott-VanZeeland et al., 2014; Strober, Peris, and Steiger, 2014).

For monozygotic twins, there is a 58% chance of both having an eating disorder if one is afflicted, about four times the probability that a fraternal twin will develop an eating disorder. There is no conclusion yet as to what is being transmitted by this genetic vulnerability—possibly vulnerabilities in personality or mood, such as a predisposition to depressive illness; persevering and sensitive personality traits; and other features that might be related to variations in the central nervous system's serotonin regulation. It has been estimated that genetic vulnerability to an eating disorder ranges from 40% to 70% (Thornton, Mazzeo, and Bulik, 2011).

Working from the other end of genetics research methods (a bottom-up approach) involves identifying a molecular level for the genetic contribution to eating disorders. This method has proved unyielding in producing significant or persuasive results. Energetic studies with as many as 100 coauthors have foundered in their attempts to define specific molecular genetic contributions to these disorders. Eating disorders are undoubtedly polygenetic with variable penetrance. Watson et al. (2019) identified eight significant loci in a recent genome-wide association study.

An epigenetic approach, investigating the interaction of genetic vulnerability with sensitizing life events, holds the most hope for understanding these disorders. Epigenetics attempts to comprehend how environmental factors, especially during critical developmental windows,

interact with genetically vulnerable substrates to make an individual more likely to develop an eating disorder. Gone are the days when genes were believed to act alone in solely determining disorders. The exceptions are rare, such as Huntington's disease. Genes have not changed significantly over recent centuries, but certain disorders such as eating disorders and autism spectrum disorders appear to be on the increase, meaning that environmental factors may play a large role. This is an encouraging finding because, generally, environmental changes respond more easily and quickly to intervention than do genetic vulnerabilities.

Improved diagnosis may play a role in the increased prevalence of eating disorders, but environmental factors should be strongly considered as modifying elements. Michael et al. have (2020) noted, "After imagined experience of genetic counseling participants reported significantly decreased feelings of stigma, shame, and guilt . . . Genetic counseling may benefit individuals with eating disorders by providing accurate recurrence risk." Hübel et al. (2019) have cautiously stated that "the field of epigenetics in eating disorders remains in its infancy." Some of the genetic findings in eating disorders are summarized in table 1.19.

Investigations into understanding the neurobiology of eating disorders have been under way for decades, with discovery of increasingly complex relationships among the functioning of brain systems related to hunger, satiety, eating behaviors, weight regulation, and eating disorders. The goal has been to translate the studies of brain structure and

TABLE 1.19. Genetics of eating disorders

▪ Twin studies: Threefold-to-fivefold increase in identical twins.
▪ Family studies: Substantially increased in families with affective spectrum disorder diagnoses.
▪ Molecular studies: Minimal/modest progress despite intensive effort. Most likely multiple genes with variable penetrance are involved.
▪ Epigenetics: Evidence for an interactive effect of genetic vulnerability with life events during critical windows of development.
▪ Eating disorders "breed true": They are not a *forme fruste* of OCD, depressive illness, or any other psychiatric disorder.
▪ Overall, genetics contribute from 50% to 70% of the vulnerability to eating disorders.

functioning into information to advance the treatment of eating disorders, in both psychological and pharmacological treatments (Bang et al., 2017). Neurobiological findings are generally neutral in perception and not associated with stigma.

Caring for the Caregivers

Studies show that those who care for family members with eating disorders suffer in similar ways to caregivers who struggle to help patients with Alzheimer's disease or schizophrenia—as much or more so. Support, education, relief from blame, and respite care are at the core of helping caregivers of patients with eating disorders (table 1.20). Without education and relief, the objective and subjective burdens of caregiving may become insuperable. Typically, mothers of eating disordered adolescents spend 2.5 hours a day giving care in the form of providing food and emotional support (Rhind et al., 2016). Fathers typically provide 1 hour a day. COVID-19 and the lockdown from the pandemic have substantially increased the burden on the carers for patients with anorexia nervosa (Clark Bryan et al. 2020).

TABLE 1.20. Preventing burnout in families and caregivers

▪ Teach psychoeducational principles about the origin course, diagnosis, treatment, and prognosis of eating disorders.
▪ Take the blame off the shoulders of families.
▪ When appropriate, avoid hospitalization of teens by teaching and implementing family-based or parent-based treatment when family is intact and willing.
▪ Plan respite care for caregivers, as with caregivers of patients with dementia or autism.
▪ Attempt to resolve alexithymia in parents and high EE (high expressed emotion, hostile, critical, intolerant), promote bonding, decrease emotional conflict. Develop a common language of accurate identification of emotional states and shared communication for family members and patients.
▪ Encourage CBT-based learning for parents about eating disorders and management of home life.
▪ Allow parents to set rules for home behavior, including requiring that the patient within the family leaves the eating disordered behaviors at the door.

Families are relieved by understanding that while eating disorders are often serious and sometimes lethal, they also have a documented, proven chance to remit, or at least may be substantially improved with comprehensive treatment—more so than early onset schizophrenia, OCD, or autism spectrum disorders. Yet, the burden of caregiving is higher among caregivers of patients with eating disorders than among caregivers of patients with depression or schizophrenia (Martin et al., 2015). Family members receive additional relief when blame is taken off their shoulders (Salerno et al., 2016). Treasure and Nazar (2016) advocate for an effective approach for carers: "Their role in helping with recovery is to give support and hold a united front themselves and with the professional team to avoid those common interpersonal reactions that adversely impact on outcome such as accommodating to the illness and reacting with high expressed emotion." *High expressed emotion* is a term indicating an adverse family environment with intense emotions of hostility, excessive criticism, intolerance, and overcontrol.

A substantial amount of psychoeducation concerning what is known about the origin, course, and treatment of eating disorders further helps families fear less and hope more. Hope, although not absolute certainty, is reasonable and is evidence based. Understanding that their family member may be genetically vulnerable helps other family members realize that eating disorders are true illnesses, not willful choices or consequences of "refrigerator moms." Respite care may be welcome once caregivers' reluctance to practice self-care is diminished and self-nurturance is prescribed. In addition to improving parent-patient relationships, parents are encouraged to return to a more balanced relationship with the patient's siblings, who may have been neglected in the intense focus on helping the patient.

Finally, parents are empowered to ask patients who are still actively ill to leave their symptoms at the door when they come home. Supportive and educated parents stop being enablers and stop being codependent with patients suffering from eating disorders. The most well-documented patterns typical of ineffective and distressed caregivers include accommodation; enabling behaviors; avoidance of all conflict; overprotection and hostility; and excessive reorganization of family life

around the eating disorder, leading to reinforcement and exacerbation of the disordered behaviors and subsequent family dysfunction (Anderson et al., 2019). When family members in family therapy (especially CBT based) learn to accurately identify and express their feelings, specifically their mood states, so that all members of the family speak the same language of emotional intelligence, then improved bonding and reduced conflict result. As with caregivers of people with schizophrenia, decreasing the intensity of expressed emotion (EE) generates a calmer home environment. In one study (Philipp et al., 2020), up to 47% of carers showed high EE. Predictors of high EE included lower caregiver skills, higher AN symptom impact, and higher levels of depression. A positive experience of caregiving was significantly associated with the use of approach-oriented coping rather than avoidance of issues (Parks et al., 2017).

The Internet, Social Media, and Smartphones in Eating Disorders Treatment and Research

The problematic aspects of the internet and social media have been well documented. Problematic internet use has been associated with the diagnoses of AN and BN, as well as food preoccupation, loss of control of eating, and dieting (Hinojo-Lucena et al., 2019). Participants in the highest quartiles for social media volume and frequency had significantly greater odds of having eating concerns (Sidani et al., 2016). Positive associations emerged between social media use and body dissatisfaction, eating disorder symptoms, and thoughts about using anabolic steroids. Facebook, Instagram, and Snapchat evidence the strongest associations. "The associations of social media use with both muscularity dissatisfaction and eating disorder symptoms were stronger for image-centric social media platforms, such as Instagram, rather than nonimage-centric platforms such as Wordpress" (Griffiths, Murray, et al., 2018). Pornography use, especially in viewers of professional pornography, was associated with use of anabolic steroids. Increased pornography use was also associated with greater dissatisfaction with muscularity, body fat, and height, as well as with greater eating disorder symptoms (Griffiths,

Mitchison, et al., 2018). A study in Singapore found that use of smartphone apps by patients with eating disorders was associated with greater severity of illness (Tan et al., 2016).

The full half of the glass would suggest that the internet, social media, and smartphones may play a healthy role for people with eating disorders, even if that role is somewhat slower to catch up with the unhealthy uses (table 1.21). Preventive intervention is the ideal, and the internet may play a role. Wade and Wilksch (2018) found internet prevention of eating disorders had more impact than control conditions using media literacy, cognitive dissonance, and cognitive behavioral therapy as interventions. The delivery of treatment by the internet may be especially useful in situations in which access to expert face-to-face evaluation and treatment is sparse. Sproch and Anderson (2019) found that videoconferencing-based psychotherapy services for eating disorders is a format that can lead to significant clinical symptom relief and is well accepted by patients. Machado and Rodrigues (2019) note that new methods for the delivery of eating disorders treatment are needed. Internet-based interventions are cost effective and may constitute a viable first line of treatment in a stepped-care model, or as an alternative to a waitlist while treatment is not available. Self-help treatment of

TABLE 1.21. Positive uses of electronic methods for eating disorders: Internet, smartphones, social media

1. Despite multiple adverse uses of social media that promote eating disorders and idealized body images, multiple positive uses for electronic media exist and are increasing.
2. The internet may play a public health role in conveying information about eating disorders to a large number of individuals and can help people to identify the presence of eating disorders in themselves as well as to direct them to resources.
3. Where treatment availability is scarce or costly, the internet may provide self-help treatment, or augment short-term contact with EDO-trained professionals, by follow-up sessions at a distance.
4. Smartphones may be used to give immediate assistance and support to patients in high-stress situations with the quick availability of directed care that is previously agreed on.
5. Smartphones may increase understanding of the natural history of how eating disorders change and evolve in real life by requesting instant responses to mood states, hunger, behaviors, and context.

eating disorders offers promise for the treatment of bulimia nervosa (Yim and Schmidt, 2019). In another area of psychiatric symptomatology, health anxiety, Axelsson et al. (2020) found, "Internet-delivered CBT appeared to be noninferior to face-to-face CBT for health anxiety, while incurring lower net societal costs. The online treatment format has potential to increase access to evidence-based treatment for health anxiety." Multiple studies are under way to increase the potential health benefits of the internet, social media, and smartphones in the areas of prevention, treatment, follow-up, and further understanding of the natural history of eating disorder symptomatology in groups who would otherwise have limited access to face-to-face research studies or treatment. Virtually any medium that can be used for harm may be turned around and used for good purposes.

Hildebrandt et al. (2020) conducted a randomized controlled telemedicine trial of CBT-guided self-help via smartphone for patients with binge eating with or without purging. Patients met criteria for either BN or BED. Each participant received eight CBT-guided self-help sessions over 12 weeks administered by health coaches. They found that CBT-guided self-help along with the Noom Monitor app achieved higher rates of remission compared to standard care (56.7% compared with 30%). Noom Monitor is a tool to track meals, binges, purges, and other activities. This study is a good indicator of how effective treatment for BN and BED can be delivered by a limited number of telemedicine sessions with monitoring for adherence by a smartphone app. While a strength of the study was the large number of participants (225 consents), the weakness was in the fact that the comparison group receiving "standard treatment" in fact received minimal treatment.

Smartphones are an effective means to monitor and assist patients with BN or BED in several ways. Random messages from health care providers can assess the presence and strength of urges to binge by asking the recipient to reply with preset ratings. Additionally, the patient, when faced with stressful situations leading to urges to binge or purge, may access preset support and guidance for choosing alternatives to abnormal behaviors.

Eating Disorders in a Time of the COVID-19 Pandemic

Multiple and complex interactions between eating disorders and the novel coronavirus have occurred throughout 2020 and will most likely continue to occur as long as the pandemic persists. Dietary habits have changed in adverse ways. Children with obesity in Verona, Italy, increased their intake of potato chips, red meat, and sugary drinks (Pietrobelli et al., 2020). An increase in consumption of fruits and vegetables along with decreased fast food has been offset by an increase in processed meats, fried foods, and sweetened beverages (Ruiz-Roso et al., 2020). As a result of disruptions in everyday life, lockdowns, unemployment, and an increase in unstructured time combined with enormous stress, weight gain during the pandemic has been called the "Quarantine-15" (Pearl, 2020), a play on the frequently used "Freshman 15." Exercise has generally decreased as a result of restrictions on outdoor interactions, fears of contagion, masking, and distance requirements.

The consequences of the COVID pandemic on individuals with eating disorders have not been inconsequential. Increased restricting and binge eating behaviors have been reported (Phillipou et al., 2020). A large study in the United States and the Netherlands confirmed the increase in restriction in patients with anorexia nervosa who feared being able to find foods consistent with their meal plans. Branley-Bell and Talbot (2020) have documented the profound negative impact of COVID-19 on individuals with eating disorders. Their findings suggest detrimental impacts on psychological well-being, including decreased feelings of control, increased feelings of social isolation, increased rumination about disordered eating, and low feelings of social support.

Individuals with BN or BED reported an increase in binge eating episodes and urges to binge (Termorshuizen et al., 2020). The National Eating Disorders Association reported an up to 78% increase in calls and online chats compared to a year previously (Flores, 2020). Half of children and adolescents admitted to the hospital for treatment of eating disorders had experienced reactivation of their disorders (Graell et al., 2020). Carers of family members with eating disorders have faced

increased practical demands placed on them and new challenges in managing their role in assisting their ill family member (Clark Bryan et al., 2020).

Treatment interactions have had to be modified. Rapid implementation of telemedicine for adolescents and young adults has taken place. In one study, telemedicine visits increased from zero to 97% of patient encounters in one month (Barney et al., 2020). Increased remote support by eating disorder services has enabled the continuation of treatment as well as self-management resources that promote self-efficacy.

Some of the behavioral and metabolic consequences of decreases in physical activity and increases in food and alcohol consumption during the pandemic as a result of heightened anxiety and decreased social interaction are summarized in figure 1.7. This vicious cycle of adverse

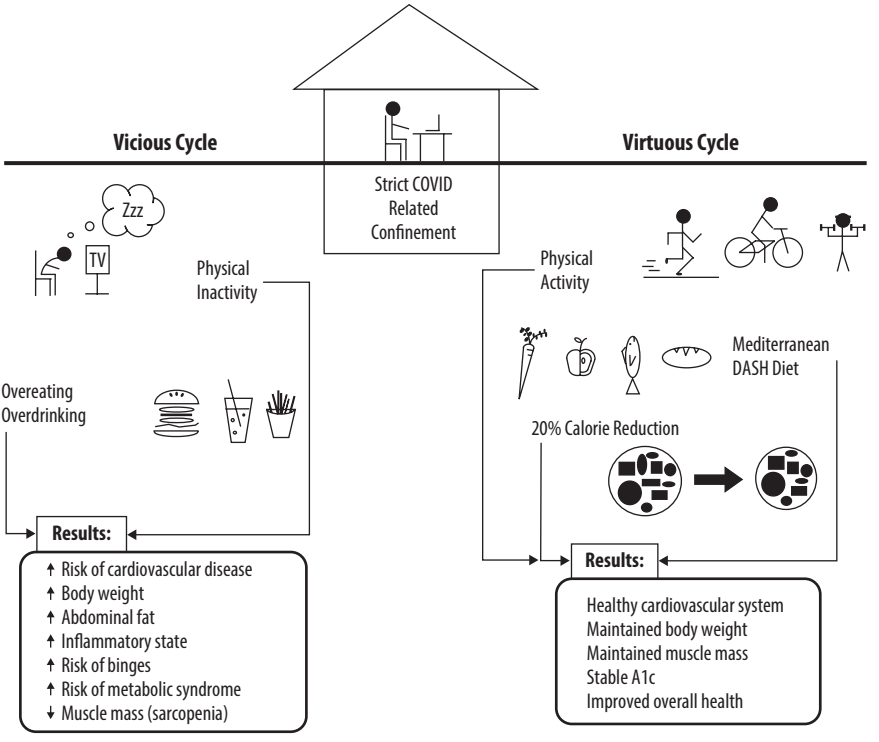

Figure 1.7. Outcomes of strict COVID-related confinement. Written by Arnold E. Andersen and created by Kari Miller.

consequences contrasts with the potential for a virtuous cycle of adaptive, resilient, clinician-guided changes that can lead to stability in health parameters or even an improvement in overall health. Martinez-Ferran et al. (2020) describe some of the adverse metabolic features of forced confinement in detail.

Later studies will document that essentially all of life's activities have been significantly altered by the COVID-19 pandemic, with many of the changes being long lasting, including changes in the behaviors and psychological functioning of patients with eating disorders.

Myths about Eating Disorders

Families with members who have eating disorders, as well as clinicians treating patients with these disorders, benefit from awareness of the myths about eating disorders that they may believe. Such erroneous beliefs will interfere with the process of healing or convey undue pessimism. These myths are summarized in table 1.22.

A Vital Goal: Primary Prevention

The optimum goal of medical practice is primary preventive intervention. Some historical examples are illustrative. Vaccination against common, previously almost universal childhood infectious diseases that resulted in severe morbidity and not insignificant mortality is now taken for granted. Public health methods achieve primary prevention of safe water supplies, free from organisms that cause epidemic infections such as cholera, a high point in the history of medicine. Likewise, childhood immunity to multiple disorders with potential morbidity and mortality is now taken for granted. Phenylketonuria and other aminoacidemias are routinely identified at birth and preventively treated. Pneumonia in the elderly has decreased with vaccination. The number of new smokers of tobacco products in North America, Great Britain, Scandinavia, and some European countries (but not Asia) has declined following a combination of financial disincentives and psychoeducational efforts, with physicians leading the way in decreased smoking.

TABLE 1.22. Myths about eating disorders

Myth	Fact
Eating disorders are always chronic.	The large majority of cases are acute, and patients improve rapidly with skilled care, remitting in up to 75% of cases for younger patients; about 50% or more of adults may remit without detectable eating disorder symptomatology.
Eating disorders are difficult to treat.	Eating disorders respond well at all levels of severity to an experienced multidisciplinary team approach; in mild cases, patients respond in 8–10 sessions.
Eating disorders always recur or reappear in a different form.	*Symptom substitution* has been disproved; recurrence is low with expert treatment.
"Normal" after treatment means no concerns about body weight, shape, or food, ever.	*Normative cultural distress* is a reasonable goal, with lip service about dieting.
Eating disorders are always severe.	Eating disorders occur on a spectrum of severity, with most cases being mild to moderate, often subclinical.
It always takes a village to treat an eating disorder.	Multidisciplinary team treatment is best for severe or complex cases; a small coordinated team usually suffices for moderate disorders. For early, milder cases, an experienced single therapist is sufficient.
All families of patients with eating disorders are abnormal.	Families vary; most are worn out, worried, and have done their best. Distressed families are usually a consequence, not a cause.
Most males with eating disorders are gay.	Most males with eating disorders are heterosexual; most younger males, asexual.
Eating disorders are a form of depression, OCD, or schizophrenia.	Eating disorders "breed true." They are not an indirect expression of other disorders.
Eating disorders are a result of past trauma.	Past trauma increases the probability of all psychiatric disorders, not specifically eating disorders, but past trauma makes treatment more complex.
Forcing patients with anorexia to eat works well.	Persuasion, by challenging cognitive distortions, supervised eating, healthy role models, is effective. Need for nasogastric tube is uncommon.
Finding the right medication is necessary.	Essential "medication" is normal food eaten normally. No medication has proved effective or necessary for anorexia nervosa; medication may be adjunctive for bulimia nervosa; it may play a larger role in binge eating.

TABLE 1.22. (*continued*)

Myth	Fact
You can scare patients into wellness.	Discovering the benefits of wellness works. Scare tactics are not effective; persuasion about seriousness is helpful.
Eating disorders are addictions.	While some similarities are obvious, addiction to food is not possible; patients may be dependent on disordered eating behaviors for emotional regulation.
Eating disorders are purely psychiatric disorders.	Eating disorders begin with voluntary dieting, overvaluation of thinness; once established, they generally become medical disorders.
Eating disorders are entirely intentional, due only to our cultural and voluntary personal choices.	Eating disorders have a 50%–70% genetic contribution.
Eating disorders occur only in spoiled, white, suburban, upper-class teen girls.	Eating disorders are equal-opportunity illnesses, occurring at all ages (7–77), in both genders, in all ethnic and racial groups, in sexual minorities, in all locations.
Anybody can treat eating disorders.	Persons with eating disorders do better with clinicians trained specifically in eating disorder diagnosis and treatment; general competence is not enough.
Eating disorders involve delusions.	Delusions are uncommon. Most patients have strong overvalued beliefs about the benefits of slimness or shape change.
Slow heart rate, low body temperature, and low blood pressure need immediate correction by specialists.	Many starvation symptoms self-improve with weight restoration and watchful waiting. Other symptoms, such as prolonged QT interval, need immediate attention.
The reproductive system is ruined.	With recovery, patients with anorexia nervosa rarely have permanent problems in gonadotropin functioning; nothing is "broken." The reproductive system is on pause, waiting for weight restoration.

Where primary prevention has not been widely effective, secondary preventive intervention through early diagnosis and treatment is an increasing reality. Many chronic, serious disorders that commonly afflict large numbers of people in westernized countries are yielding to a variety of secondary preventive and therapeutic interventions. Death from coronary artery disease has substantially decreased, as has death

from the silent killer of hypertension, although both are still distressingly high.

The status of primary prevention for eating disorders is controversial, with substantial differences between countries and clinicians (table 1.23). Early intervention and increasingly effective treatments are not in dispute. Norway assigns an experienced nurse to encourage healthy athleticism and eating habits (primary prevention), as well as to identify early cases of eating disorders (secondary intervention). Other countries forgo the opportunity for primary prevention. Two examples in the United States offer hope for primary prevention. Ballet schools that set a lower limit on weight for students to be eligible for continuing their studies appear to decrease the number of cases of AN. Some states and institutions have set lowest acceptable weights, lowest speeds of weight loss, and minimum final percentages of body fat acceptable for students engaging in wrestling. What is clear is that speaking to students about signs and symptoms of eating disorders encourages, rather than discourages, the beginning of eating disorders. Reducing risk factors may lead to benefits in reducing eating disorders (Greif, Becker, and Hildebrandt, 2015). School-age bullying is one specific risk factor that appears to be amenable to reduction (Copeland et al., 2015).

TABLE 1.23. Preventive intervention for eating disorders

▪ Currently, secondary intervention is the most strongly proven approach: early diagnosis, prompt referral, experienced treatment with comprehensive goals.
▪ Primary prevention has some preliminary evidence of effectiveness in sports and vocational groups. Prevention in school-aged children is a major goal.
▪ Setting lowest acceptable weight in dance schools and rate of weight loss in wrestling programs limits severe weight loss because the result of falling below this weight results in removal from the programs.
▪ Families that eat the dinner meal together every day have fewer cases of eating disorder and fewer children with substance abuse.
▪ Teaching signs and symptoms of eating disorders to schoolchildren probably increases the number of cases.
▪ Teaching media skepticism for all and functional use of the body in place of objectification by artificial beauty standards for girls may be helpful.

Information for Patients

It is essential for patients to know that organ functions will usually not be completely normal for 3–6 months even after weight becomes normal. Attaining a healthy weight and resolving abnormal behaviors and cognitions is the *beginning* of comprehensive treatment, not the end. The goal is to instill *flourishing*—language for encouraging, to the maximum degree possible, a humanistic life that is creative, flexible, and resilient. Patients with bulimia nervosa may be reassured to know that urges to binge or purge are not, in themselves, indications of ineffective treatment. Continued reflux in formerly bulimic patients may indicate some incompetence of the gastroesophageal junction.

The goal for dealing with persisting medical symptoms is prompt, effective, complete treatment so that we can help patients achieve changes in thinking and behavior without uncomfortable or serious medical symptomatology interfering with the process of psychological and behavioral change. Severe bloating, painful reflux, or severe hypothermia makes it difficult to concentrate in psychotherapy. Occasionally, a specialist may be needed for assessment and treatment of complex residual medical concerns. There is increasing concern that low bone mineral density may be persistent in patients who have had as little as 6 months of amenorrhea from AN. DEXA scans should be requested for patients with a history of 3 or more months of amenorrhea, 6 months of significant weight loss, or, for males (Mehler et al., 2008), low weight for 6 or more months. This information may be used psychotherapeutically. Other chapters in this book deal in more detail with medical evaluation and treatment of endocrine, gastrointestinal, cardiac, bone, and other ailments related to eating disorders.

Summary of the Diagnosis and Treatment of Eating Disorders by Primary Care Providers

Two excellent overviews of the field of eating disorders appeared in 2020: (1) "Current Approach to Eating Disorders: A Clinical Update" (Hay,

2020); and (2) "Anorexia Nervosa" (Mitchell and Peterson, 2020). Both publications offer much up-to-date information.

Primary care providers can confidently diagnose eating disorders with a brief set of screening questions and a mental status examination, and they can treat these disorders with clear, proven, relatively straightforward treatment methods. Recent cases of mild to moderate severity can be treated comprehensively by the primary care physician alone or in conjunction with psychologically trained colleagues. Medical assessment is important for establishing the severity of illness and treating significant medical complications, but not for diagnosis. The guideline is to *rule in* eating disorders, not to *rule out* all possible causes of weight loss or binge-purge. Families respond well to a combination of education, support, and inclusion in the treatment process, according to the age of the patient and the needs of the family. Realistic optimism based on comprehensive treatment is entirely warranted. Every aspect of most eating disorders can be improved, especially with the up-to-date diagnostic and therapeutic skills of motivated medical and psychological clinicians.

Some takeaway "pearls" regarding the diagnosis and treatment of eating disorders are listed in table 1.24.

Hopes and Goals for the Near Future: Attaining Ideal Treatment and Prevention

Ideal treatment may be difficult to achieve, but there is no reason that it should not be the guiding light for the future. Some patients suffering from eating disorders may be detected early, receive appropriate comprehensive treatment, and benefit from patient-specific, personalized follow-up until full health is attained, emotionally, behaviorally, and socially. For many patients with eating disorders, perhaps the majority of those seriously ill, adequate treatment requires provisions for a long journey, provisions that are only sparingly available in many cases, in many health care systems.

TABLE 1.24. "Pearls" concerning diagnosis and treatment of eating disorders

- Large community studies indicate a female-to-male ratio for eating disorders of 1:2–4; the ratio of 1:10 in clinic reports is a false ratio. Implication: many males are undiagnosed or avoidant of diagnosis and/or treatment. Anorexia nervosa in males is less ego-syntonic than in females. Screening criteria are often female oriented.
- Dieting is normative for most females, without specific reasons; dieting in males usually begins for specific reasons; the desire for lean muscularity is normative.
- Diagnosis is made by history and mental status examination, not by ruling out all possible medical causes.
- At least two rapid, easily administered screening instruments are available: the SCOFF and the SDE.
- Although most common in the teens and twenties, eating disorders can occur from 7 to 77 years of age. Remember to consider eating disorders in older women, males, racial and sexual minorities, and the mentally challenged.
- Females with anorexia are often proud of weight loss (ego-syntonic), but bulimic behavior is usually shameful or guilt producing (ego-dystonic). The patient may test how judgmental you are before disclosing bulimia nervosa symptoms.
- The *DSM-5* and ICD-11 diagnoses are categorical diagnoses by checklists. In real life, amount of weight loss, drive for thinness, body image distortion, and fear of fatness are dimensional variables. There are many more subclinical cases of anorexia nervosa than cases meeting full categorical diagnosis. Compare asthma, high blood pressure, and hyperglycemia: all are treated short of severe symptomatology.
- Binge eating disorder (binge eating without any compensation) occurs in about 25% of medically obese patients as a contributing factor to total weight burden. Assess symptoms of eating disordered thinking and behavior in all obese patients. Increase awareness of BED in bariatric surgery clinics.
- Patients with binge eating disorder most typically are older (30s–40s). Occurrence is about equal in males and females; may involve compulsive grazing as well as binges.
- Eating disorders in older patients are often complicated by concurrent medical or depressive symptoms as well as a failure to consider an eating disorder as diagnosis.
- Eating disorders are almost never solitary disorders. Once an eating disorder diagnosis is made, assess for comorbid psychiatric disorders and medical consequences. Decide which are predisposing factors and which are secondary to the eating disorder.
- The most common contributing factor to AN, BN, and, to some extent, BED is dieting. *Diet* is a four-letter word.
- Approximately 8% of anorexia nervosa cases begin with involuntary weight loss; additional, voluntary weight loss is practiced only after initial weight loss from a medical or surgical cause inspires the idea and benefits of losing weight.
- Younger patients with anorexia nervosa may try to fool the physician about prescribed weight restoration by carrying secret weights in their pockets or drinking noncaloric beverages before examination. An outside informant is essential.

(*continued*)

TABLE 1.24. (*continued*)

- Primary care physicians can be fully competent to treat many cases of eating disorders, especially the earlier and milder cases that are most common.
- Males are usually preoccupied with attaining lean muscularity, and especially concerned with body shape from the waist up. Females are most commonly concerned with body shape from the waist down. In males, desire for shape change exceeds desire for weight change; in females, desire for weight change exceeds desire for shape change. Body image distortion is more complex in males than in females; males may desire (a) extreme thinness, (b) normal weight with extreme muscular definition, or (c) high weight with extreme muscular definition.
- An increased probability of gay orientation exists (18%–20%) in males; sexual minorities appear to be at increased risk.
- Males usually perceive fatness and a need to start dieting at higher relative weights than females, who feel fat starting at 15%–17% below normal weight.
- Males may fear being told they have a girl's disease or a gay person's disease. They often delay coming in for diagnostic evaluation. Anorexia nervosa is generally ego-alien in males, often ego-syntonic in females. BN and BED often induce strong feelings of shame.
- Males are sometimes stigmatized by female patients with eating disorders as typical of problematic males in their past.
- Many health professionals are uneducated about eating disorders in males and often do not consider a diagnosis of eating disorders in male patients, or programs may not accept males—a form of gender discrimination.
- The most important factor in identifying eating disorders by primary care providers is to think of it as a possible diagnosis in all patients.

Some realistic goals for the near future (the next decade) include:

1. Establishing more eating disorder treatment centers of excellence, which can comprehensively treat all moderately to severely ill patients, employing a multidisciplinary *continuum of care*, including intensive medical services, specialized inpatient units, a partial psychiatric hospital (day) program, outpatient clinics for diagnosis and treatment, and aftercare in residential settings or other locales that offer decreasing supervision as the transition to independent living is accomplished.
2. Establishing collaborative bridges of liaison between primary care providers, schools, families, treatment centers of excellence, outpatient providers, coaches, and others so that care is carried out in appreciation of the long arc of these disorders, with inte-

grated treatment toward the goal of attaining a life of robust flourishing in both general society and the micro-societies in which we all live. All components of the caregiving health community must be interactive and responsible to each other as partners. The treatment of eating disorders is often a decades long process.

3. Increasing funding for research in the field of eating disorders in all aspects, with integration between research findings and improved treatment methods; funding in health care systems to provide respite care for families of children with eating disorders.
4. Closing the gap between molecular genetics and family genetics; understanding the role of epigenetics for improving our understanding of the mechanisms involved in the predisposing and sustaining factors for eating disorders; advocating for altering life situations that may increase the impact of genetic predispositions, such as prevention of bullying and sexual abuse.
5. Increasing the evidence and follow-through for primary preventive intervention.
6. Wider teaching of simple methods for routine screening for eating disorders in primary care and OB-GYN practices.
7. Advocacy for legislation to establish true parity for health plan funding for the comprehensive, long-term treatment of eating disorders, providing for treatment needs until maximum improvement is both attained and maintained.
8. Increased training of clinicians to develop competence in evidence-based psychotherapies, including CBT, IPT, or DBT, for the treatment of eating disorders.
9. Improved understanding of how males with eating disorders may benefit from different diagnostic methods and treatment strategies than those used for females.
10. Increased use of innovative electronic methods for the diagnosis, treatment, and follow-up of patients with eating disorders, such as the internet and smartphones.
11. Developing for all school-age children a virtuous cycle of lifestyle practices, including healthy nutritional patterns, lifetime athletic skills, and media skepticism about weight and dieting, as well as

empowering young girls with the skills to use their bodies functionally rather than as objects and teaching respect for persons of all ages and all body types, using methods found to be effective in Nordic countries.

12. Identifying additional subpopulations highly vulnerable to eating disorders, such as children and adolescents with insulin-dependent diabetes mellitus, sexual minorities, children with early obesity, and other individuals for whom primary prevention is both necessary and possible.
13. Long-term studies to identify which methods of treatment have the most enduring and comprehensive results.
14. Increasing referral to family-based or parental-based treatments of selected adolescent patients who meet criteria of a BMI greater than 15 or bulimia nervosa teens without life-threatening medical or psychiatric comorbidities.
15. Increased outreach to Latinx and Black populations with the diagnostic and treatment methods proved most effective for minority populations.
16. Recognition that eating and weight disorders constitute a growing public health concern in developing countries.

In summary, the reasonable goals for the near future can be condensed to preventive intervention when possible; early recognition always; comprehensive, personalized, effective treatment by networks of shared concern, over decades if needed, with links between research and treatment; and adequate funding.

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2

The Multidisciplinary Team and Levels of Care in the Treatment of Eating Disorders

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COMMON QUESTIONS

Which major disciplines do members of the treatment team for eating disorders typically represent?

What common psychological therapies are evidence-based treatments for eating disorders?

Is there any way to know whether an eating disorder professional possesses the competencies necessary to be effective?

What is the core psychopathology of eating disorders?

Which medications are FDA approved for the treatment of eating disorders?

Which medications may also be considered in the treatment of patients with eating disorders?

Eating disorders (anorexia nervosa, bulimia nervosa, binge eating disorder, avoidant/restrictive food intake disorder, other specified feeding or eating disorder) are complex illnesses affecting both the body and the mind. Biogenetic, psychological, and sociocultural factors all contribute to the onset and maintenance of these disorders. The complex interactions of these biopsychosocial factors speaks to the need for multidisciplinary teams to provide treatment. In fact, guidelines developed by the American Psychiatric Association (APA, 2006), American

Academy of Pediatrics (AAP, 2013), American Academy of Child and Adolescent Psychiatry (Lock and La Via, 2015), Society for Adolescent Health and Medicine (SAHM) (Golden et al., 2015), Academy for Eating Disorders (AED, 2016), American Dietetic Association (Ozier and Henry, 2011), and Residential Eating Disorder Consortium all emphatically recommend a multidisciplinary team approach to the treatment of eating disorders. The Joint Commission (JC, 2016) and the Commission on Accreditation of Rehabilitation Facilities (CARF, 2013) have recently established disease-specific standards for inpatient/residential treatment for eating disorders that mandate the use of a multidisciplinary treatment team (physicians, psychotherapists, dieticians, and nurses). In this chapter we review the composition, roles, and responsibilities of the multidisciplinary team, present the recommended criteria for different levels of care, and discuss how these affect standards of care for the various disciplines.

Case Study

Consider the number and types of disciplines involved in the care of the patient in this case.

R.G., a 27-year-old married female with a 15-year history of anorexia nervosa and multiple prior treatment stays in residential units, was admitted to treatment in an inpatient eating disorder program at a major medical center. She presented with marked restriction of food and fluids, self-induced vomiting when she ate normal meals, extreme fear of weight gain, and the belief that she already looked too big, at 5 feet 6 inches and 75 pounds (58% of ideal body weight [IBW]; body mass index [BMI] of 12.1). R.G. also struggled with a comorbid anxiety disorder. She described conflict with her husband, stemming in part from her difficulty discussing her thoughts and feelings with him. Admission EKG indicated marked sinus bradycardia (heart rate in the high 30s), prompting a brief transfer to a medical floor for cardiac telemetry monitoring and initiation of nutrition rehabilitation, with consultation from the eating disorder treatment team.

Once R.G.'s heart rate had increased into the 40s, she returned to the inpatient eating disorder unit. There, under the guidance of a

registered dietician, she began following a prescribed meal plan with regular increases in caloric intake to support a weight restoration of 2–4 pounds per week. In the first few weeks of her stay, R.G. had repeated episodes of hypoglycemia; she initially refused to accept glucose tablets or IV dextrose, citing fear of additional calories, but ultimately, after an endocrine consult, she was willing to comply with the IV until her blood sugars had stabilized. She was followed closely by the internist on the eating disorders unit, who also monitored the patient's labs biweekly to help avoid refeeding syndrome.

Throughout her stay in the inpatient eating disorder program, R.G. met with an individual therapist twice a week as well as her dietician and participated in several therapy groups per day. Therapy focused on helping R.G. develop and practice skills for tolerating the distress she experienced as she restored her weight, as well as skills for managing her anxiety more generally. A family therapist met weekly with R.G. and her husband, focusing on improving the couple's communication skills, and the husband attended weekly family education and support groups. Her psychiatrist saw her twice weekly for medication management.

After 4 weeks of inpatient treatment and 6 weeks of residential-level treatment, R.G. stepped down to the partial hospitalization level of care in the same eating disorder program, at a weight of 104 pounds (80% of IBW; BMI 16.8). There she continued the weight restoration process, with support from the registered dietician; began practicing eating snacks and then meals, at restaurants with staff supervision; and participated in a weekly cooking group. Her psychotherapy shifted to include work on long-standing body image concerns, helping R.G. strengthen her sense of self and identity separate from her eating disorder, as well as relapse prevention work.

Diagnostic Categories for Eating Disorders

The most recent *Diagnostic and Statistical Manual of Mental Disorders, DSM-5* (APA, 2013), includes five diagnostic categories of eating disorders (table 2.1): anorexia nervosa, bulimia nervosa, binge eating disorder,

TABLE 2.1. Eating disorder diagnostic categories (*DSM-5*)

Anorexia Nervosa

A. Restriction of energy intake relative to requirements, leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health. *Significantly low weight* is defined as a weight that is less than minimally normal or, for children and adolescents, less than that minimally expected.

B. Intense fear of gaining weight or of becoming fat, or persistent behavior that interferes with weight gain, even though at a significantly low weight.

C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight.

Bulimia Nervosa

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
   1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than what most individuals would eat in a similar period of time under similar circumstances.
   2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).

B. Recurrent inappropriate compensatory behaviors in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, or other medications; fasting; or excessive exercise.

C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least once a week for 3 months.

D. Self-evaluation is unduly influenced by body shape and weight.

E. The disturbance does not occur exclusively during episodes of anorexia nervosa.

Binge Eating Disorder

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
   1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than what most people would eat in a similar period of time under similar circumstances.
   2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).

B. The binge eating episodes are associated with three (or more) of the following:
   1. Eating much more rapidly than normal.
   2. Eating until feeling uncomfortably full.
   3. Eating large amounts of food when not feeling physically hungry.
   4. Eating alone because of feeling embarrassed by how much one is eating.
   5. Feeling disgusted with oneself, depressed, or very guilty afterward.

C. Marked distress regarding binge eating is present.

D. The binge eating occurs, on average, at least once a week for 3 months.

E. The binge eating is not associated with the recurrent use of inappropriate compensatory behavior as in bulimia nervosa and does not occur exclusively during the course of bulimia nervosa or anorexia nervosa.

TABLE 2.1. *(continued)*

Avoidant/Restrictive Food Intake Disorder

A. An eating or feeding disturbance (e.g., apparent lack of interest in eating or food; avoidance based on the sensory characteristics of food; concern about aversive consequences of eating) as manifested by persistent failure to meet appropriate nutritional and/or energy needs associated with one (or more) of the following:
   1. Significant weight loss (or failure to achieve expected weight gain or faltering growth in children).
   2. Significant nutritional deficiency.
   3. Dependence on enteral feeding or oral nutritional supplements.
   4. Marked interference with psychosocial functioning.

B. The disturbance is not better explained by lack of available food or by an associated culturally sanctioned practice.

C. The eating disturbance does not occur exclusively during the course of anorexia nervosa or bulimia nervosa, and there is no evidence of a disturbance in the way in which one's body weight or shape is experienced.

D. The eating disturbance is not attributable to a concurrent medical condition or not better explained by another mental disorder. When the eating disturbance occurs in the context of another condition or disorder, the severity of the eating disturbance exceeds that routinely associated with the condition or disorder and warrants additional clinical attention.

Other Specified Feeding or Eating Disorder

This category applies to presentations in which symptoms characteristic of a feeding and eating disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the feeding and eating disorders diagnostic class. The other specified feeding or eating disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific feeding and eating disorder. This is done by recording "other specified feeding or eating disorder" followed by the specific reason (e.g., "bulimia nervosa of low frequency"). Examples of presentations that can be specified using the "other specified" designation include the following:

A. Atypical anorexia nervosa: All of the criteria for anorexia nervosa are met, except that despite significant weight loss, the individual's weight is within or above the normal range.

B. Bulimia nervosa (of low frequency and/or limited duration): All of the criteria for bulimia nervosa are met, except that the binge eating and inappropriate compensatory behaviors occur, on average, less than once a week and/or for less than 3 months.

C. Binge eating disorder (of low frequency and/or limited duration): All of the criteria for binge eating disorder are met, except that the binge eating occurs, on average, less than once a week and/or for less than 3 months.

D. Purging disorder: Recurrent purging behavior to influence weight or shape (e.g., self-induced vomiting; misuse of laxatives, diuretics, or other medications) in the absence of binge eating.

E. Night eating syndrome: Recurrent episodes of night eating, as manifested by eating after awakening from sleep or by excessive food consumption after the evening meal. There is awareness and recall of the eating. The night eating is not better explained by external influences such as changes in the individual's sleep-wake cycle or by local social norms. The night eating causes significant distress and/or impairment in functioning. The disordered pattern of eating is not better explained by binge eating disorder or another mental disorder, including substance use, and is not attributable to another medical disorder or to an effect of medication.

(*continued*)

TABLE 2.1. (*continued*)

Unspecified Feeding or Eating Disorder This category applies to presentations in which symptoms characteristic of a feeding and eating disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the feeding and eating disorders diagnostic class. The unspecified feeding and eating disorder category is used in situations in which the clinician chooses not to specify the reason that the criteria are not met for a specific feeding and eating disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Source: American Psychiatric Association, 2013.

avoidant/restrictive food intake disorder, and other specified feeding or eating disorder. Throughout this chapter, the generic term *eating disorders* refers to all the diagnostic categories listed above.

Roles and Responsibilities of the Treatment Team

Medical physicians, psychiatrists, psychotherapists, dieticians, and nurses are the common core disciplines in the eating disorder treatment team. The level of care prescribed for the patient usually determines the level of involvement of each of these core disciplines. Additionally, the higher the level of care, the more involvement is needed from other disciplines, such as occupational therapists, activity therapists, and educators.

Medical Physicians

Internists, pediatricians, family practice physicians, and adolescent medicine physicians are the providers who assess and manage the medical conditions associated with eating disorders. Increased attention has been placed in recent years on the awareness of eating disorders among physicians (AED, 2016). This is particularly important because early detection and intervention is associated with better outcomes (Forman et al., 2011), and physicians are often the first point of contact for concerned patients and families. The extent of medical involvement by these providers is determined by the nature and extent of the patient's medical condition and the level of care. It is important that all members

of the treatment team understand that psychosomatic complaints are common in this population. Medical tests and interventions must be judicious and be provided in conjunction with psychological interventions for management of physical discomfort and anxiety. Those with the most severe forms of the illness (e.g., those with BMI of <15 or marked electrolyte and acid-base aberrations resulting from excessive purging behaviors) may require daily medical visits early in the course of treatment. Patients with a BMI of <14 are recommended to initiate treatment in a specialized medical unit in a hospital due to their likely unstable state and increased potential need for urgent interventions.

Psychiatrists

Psychiatrists are usually responsible for medication management of the psychiatric conditions associated with eating disorders. Unfortunately, there is only a modest evidence base for the use of psychiatric medications for eating disorders (APA, 2006). Nonetheless, a good evidence base supports the use of medications for treatment of other psychiatric illnesses that often co-occur with eating disorders, such as depression, anxiety, and obsessive-compulsive disorder (National Institute for Health and Care Excellence [NICE], 2017). Consequently, it is recommended that patients with eating disorders receive a psychiatric evaluation early in their treatment. The extent of ongoing contact with the psychiatrist is then determined by the complexity of the medications recommended and the patient's level of care. Some psychiatrists also deliver psychotherapy, so the frequency of contact and length of sessions could be greater, as dictated by the nature of the psychotherapy being provided.

Many geographic areas do not have adequate access to psychiatrists. Family practice physicians, internists, and pediatricians will often partially fill this gap by taking responsibility for managing psychiatric medications. Conversely, some psychiatrists are highly informed about the medical management of patients with eating disorders and are comfortable with assuming that responsibility, except for patients with the most severe forms of the illness. (e.g., BMI <14–15 or marked medical instability).

Psychotherapists

Currently, the primary evidence-based treatments for eating disorders are psychologically based. These include cognitive behavioral therapy (CBT), interpersonal therapy (IPT), dialectical behavioral therapy (DBT) (NICE, 2017), acceptance and commitment therapy (ACT), and family-based therapy (FBT) (APA, 2006; NICE, 2017). These psychotherapies may be delivered by psychiatrists (MD), clinical psychologists (PhD, PsyD), social workers (LCSW, LSW), or master's-level counselors/psychotherapists (LPC, LMFT). As with the other disciplines discussed here, the initial assessment is critical in determining how the psychotherapist proceeds in treating a patient with an eating disorder, including what psychotherapy approaches are to be used and in what modality (individual, family, and/or group therapy). The overall basis for the different psychotherapeutic approaches, however, remains more "expert" opinion than based on randomized controlled trials (Murray, 2019).

Dieticians

The irrefutable need for nutritional rehabilitation of patients with eating disorders and early recognition of eating disorder symptoms has created an important opportunity for registered dieticians (RDs) to become core members of the behavioral health treatment team. Best practices include a multidisciplinary team that must involve an RD, who can serve as a clinical advocate for the nutritional needs of the patient (Hackert, Kniskern, and Beasley, 2020). According to the 2020 AED *Guidebook for Nutrition Treatment of Eating Disorders*, medical stabilization and nutritional restoration are necessary first steps in the treatment of eating disorders. Dieticians use evidence-based guidelines to help support patients in normalizing eating patterns, decreasing eating disorder behaviors, and restoring body weight back to a healthy state (AED, 2020). Nutrition-focused physical examinations, in addition to subjective dietary recalls, help clarify treatment goals by evaluating the current nutritional and physical condition compared to premorbid status (Malone and Hamilton, 2013).

Dieticians are responsible for implementing meal plans, providing medical nutrition therapy, and establishing goal weight ranges. Meal plans are specifically designed and created by the RD, with input from the patient, to ensure patients meet appropriate energy needs, as well as to provide a structured approach to introducing feared or avoided foods. Goal weights are usually monitored and determined by the dietician, based on historical weight development, ethnicity, and a detailed weight assessment. It is important to note that dieticians who specialize in the treatment of eating disorders require significant education, training, and clinical supervision. Because of the unique clinical severity and symptomatology of the illness, continuing education is required and demands maintenance of professional best practices (Hackert, Kniskern, and Beasley, 2020). Since food is often the "centerpiece" of these illnesses both psychologically and with respect to medical complications, RDs should also be familiar with different physiological changes relevant to treatment modalities for eating disorders. As scientific advances emerge, exploration of nutritional deficiencies and the treatment of eating disorders may offer dieticians additional context in providing medical nutrition therapy (Holscher, 2020).

Nurses

Nurses usually become involved in the treatment of patients with eating disorders at the inpatient, residential, and partial hospitalization program (PHP) levels of care. This is because as medical and psychiatric acuity increases, the need for more continuous supervision of the patient also increases. Registered nurses (RNs) and other nursing staff (psychiatric technicians, nurses' aides, childcare workers, and other milieu staff) are responsible for the overall 24/7 supervision and medical safety of patients in 24-hour care (i.e., inpatient and residential care) and, to some extent, patients in PHP care. They are also largely responsible for implementation of the medical and psychiatric treatment plan and the administering of prescribed medications. In many treatment settings, nursing staff also actively participates in delivering group therapies and in meal supervision.

Competency Certification

Currently only one organization provides competency certification for different disciplines in the eating disorders field. The International Association of Eating Disorder Professionals (IAEDP) (2005) offers certified eating disorder specialist (CEDS) designation for physicians, psychotherapists, dieticians, nurses, and art/recreation/movement therapists. Achievement of CEDS designation based on hours of eating disorders practice, course work and successful completion of the certification examination is one indication of the requisite acumen to care for patients with eating disorders. Membership in either the AED or IAEDP would also indicate disease-specific interest, as would attending continuing education workshops and conferences specific to eating disorders.

Levels of Care and Treatment Team Involvement

Following an initial assessment, patients with eating disorders usually receive a recommendation regarding level of care. Determining what level of care a patient with an eating disorder needs can be a challenging task. As mentioned earlier, the roles and responsibilities of the various disciplines are largely determined by the level of care being provided. The APA *Practice Guideline for the Treatment of Patients with Eating Disorders* offers standards of treatment for different disciplines at each level of care (APA 2006). The most common levels of care, and the roles of the various team members at each level, are summarized below.

Outpatient Care

Initial medical and psychiatric medication evaluations are recommended during the first outpatient visit, with follow-up visits with physician and psychiatrist as needed.

For adults with anorexia nervosa, psychotherapy sessions are typically provided once or twice a week. Family or couples therapy may be added if indicated. For children and adolescents with anorexia nervosa,

a high level of family involvement as prescribed in FBT (Lock and Le Grange, 2013) is usually recommended; individual therapy for adolescents may be added as indicated. For bulimia nervosa and binge eating disorder, one or two psychotherapy sessions per week are provided, typically using CBT or IPT manualized treatment (Fairburn, 2008).

Outpatient treatment for adults with an eating disorder may also include weekly visits with a dietician for nutrition counseling. Monitoring of weight, if needed, is typically provided either by the RD or by the medical doctor. Manualized FBT with children and adolescents with anorexia nervosa does not require dietician involvement.

One challenge for most multidisciplinary outpatient teams is maintaining regular communication to ensure coordinated care. Because medical and psychiatric symptoms are often minimal at this level of care, team communication is often coordinated by the psychotherapist.

Intensive Outpatient Program

The patient in an intensive outpatient program lives at home and attends a treatment program for 2–4 hours per day, typically 3 days per week. Programming usually includes individual psychotherapy once a week, nutrition counseling with an RD once a week, various group therapies, and limited meal supervision provided by the therapist, dietician, or ancillary staff (two or three meals per week). Family or couples therapy may also be included. Initial medical and psychiatric medication evaluation are recommended, with follow-up visits with the physician and psychiatrist as needed.

Partial Hospitalization Program

The patient receiving care in a PHP or in supportive housing lives at home and spends 6–12 hours per day, 5–7 days per week, at a clinic. Medical and psychiatric evaluations are initially required. Medical follow-up is provided as needed, including regular blood draws for phosphorus levels, especially early in the refeeding process. Psychiatric follow-up is typically provided once or twice a week. Nursing care is provided as needed.

Programming usually includes individual, group, and family or couples therapy. Meal support (two or three meals and one or two snacks each day) is provided by milieu staff. Dieticians are responsible for evaluating the nutritional status of the patient, recommending a strategy for nutritional rehabilitation, and providing nutrition counseling and guidance, typically meeting with the patient once a week. This level of care can also include participation from other disciplines, such as activity and occupational therapists as well as educators for child/adolescent programs.

The more intensive 7-days-a-week programs are often more effective—particularly in the early stages of treatment—at accomplishing weight gain and interruption of symptoms such as binge eating, purging, and excessive exercise. These more intensive programs will often reduce the days and hours expected of the patient once they demonstrate progress with symptom interruption.

Residential Treatment

In residential care, the patient sleeps in a facility receiving care and supervision 24 hours per day, 7 days per week. The involvement of medical providers, psychiatrists, and nurses is variable across residential facilities, but 24 hours of nursing and daily medical availability are optimal. Programming usually includes individual, group, and family or couples therapy. Support and supervision at meals and snacks is provided by nursing, psychotherapy, and dietary staff. Psychiatrist and physician visits are twice a week. Individual psychotherapy is usually twice a week, and family therapy occurs once per week. Dieticians are responsible for evaluating the nutritional status of the patient, recommending a strategy for nutritional rehabilitation, and providing nutrition counseling and guidance, typically meeting with the patient twice a week to accomplish 2–4 pounds of weight gain per week. Residential treatment may also include participation from other disciplines, such as activity and occupational therapists as well as educators for child/adolescent programs.

Inpatient Care

Nonspecialized Medical Hospitals and Emergency Centers

These are general medical settings with providers who are often inexperienced in the medical and psychiatric management of eating disorders. The treatment team typically consists of nonspecialized hospitalist physicians and medical nurses. The goal is usually medical stabilization then return of the patient to a specialized eating disorders residential or inpatient program.

Nonspecialized Inpatient Psychiatric Hospital

These general psychiatric settings are focused on crisis stabilization for all diagnostic groups, with an average length of stay of 3–7 days. The providers are typically inexperienced in addressing the medical and psychiatric challenges of weight restoration and symptom interruption for patients with eating disorders. The treatment team typically consists of a psychiatrist, psychotherapist, and nursing staff, and sometimes activity and occupational therapists. The goal is usually to quickly stabilize acute psychiatric symptoms and then return the patient to the outpatient team or refer them to a specialty eating disorders inpatient/residential program.

Clearly, medical or psychiatric acuity may require accessing the closest care available. Nonspecialized, poorly informed treatment, however, can result in medical ineffectiveness or even calamity as well as psychological trauma for patients with eating disorders, due to the inexperience factor. Rapid refeeding can be life threatening, and poorly conceived invasive medical procedures (e.g., nasogastric tubes without clear indication, total parenteral nutrition) and behavioral/psychological interventions (such as use of physical restraints and shame-based psychological interactions) can have long-lasting adverse iatrogenic effects. We highly recommend that specialized care be engaged as quickly as possible.

Specialized Eating Disorders Inpatient Psychiatric Hospital

In this specialized setting, the patient resides in the facility and is under the continuous and intensive supervision of staff, 24 hours per day, 7 days

per week. Staff members are experienced in the medical and behavioral challenges of treating this patient population. This level of care is usually indicated when the patient is below 75% of IBW, is acutely medically or psychiatrically ill, or when outpatient care or PHP attempts have failed to interrupt eating disorder symptoms. At this level of care, physicians are responsible for the overall care of the patient and typically lead the treatment team. At the inpatient level of care, a psychiatrist meets with the patient 7 days per week, and a medical provider sees the patient for an initial assessment and then as often as needed. Full-time supervision by nursing staff is also required. The nursing staff is responsible for ensuring the overall safety of the patient, communicates concerns to the medical and psychiatric physicians, and helps ensure that the treatment plan is being implemented. Nursing staff may also participate in providing group therapy and meal supervision. Milieu staff assists with implementing the individual treatment plan for each patient and with supervising patients at meals and in general. Psychotherapists are responsible for the patient's psychological treatment, which usually includes twice-a-week individual therapy, once-a-week family or couples therapy, and group therapy several times a day. Psychotherapists may also participate in meal supervision. Dieticians are responsible for evaluating the patient's nutritional status, recommending a strategy for nutritional rehabilitation, and providing nutrition counseling and guidance, typically meeting with the patient once or twice a week to help achieve the goal of 2–4 pounds of weight gain per week. The dieticians also participate in meal supervision. The inpatient level of care can also include participation from other disciplines, such as activity, art, or movement therapists and occupational therapists. Educators for child/adolescent programs are usually necessary for younger patients to maintain their academic standing. Competent experienced medical physicians are a critical component of the inpatient care process, and having one present or available 7 days a week and 24 hours a day for this level of care is optimal.

Specialized Eating Disorders Medical Inpatient Hospital

The APA guidelines outline five levels of care between outpatient and inpatient but fail to completely outline a sixth level of care—medical treatment. Severe and extreme eating disorders can cause life-threatening medical complications, including dangerous electrolyte imbalances, heightened risk for refeeding syndrome, severe dehydration, edema, gastrointestinal complications, superior mesenteric artery (SMA) syndrome, bradycardia, and organ failure, to name but a few. It is important to recognize that these potential complications frequently exist at lower BMIs, and thus the need to intervene in a timely fashion.

Intervention should include medical stabilization in a highly sophisticated and specialized medical unit, prior to a patient starting traditional eating disorder treatment. Many experts espouse that this should happen when a BMI is <14.0, or when the patient's weight is <70% IBW (Mitchell and Peterson, 2020). Thus, in general, patients with anorexia nervosa (AN) or avoidant/restrictive food intake disorder (ARFID) who are below 70% IBW should first be treated in a center of excellence for the medical stabilization of those patients.

In general, if the patient's weight is between 70% and 84% of IBW, they are best served in an inpatient or residential eating treatment center, and if the weight is 85% to 95% of IBW, a PHP will generally suffice. But nothing is absolute, and thus the frequency of purging behaviors and other physical or psychiatric considerations can further qualify the level of care needed.

Housed within medical hospitals, these units are very rare and specialize in treating the most medically complex eating disorders. The patient is hospitalized, and the treatment is focused on medical stabilization, with some emphasis on psychiatric or psychological symptoms. The treatment team on these units typically includes internists, subspecialist physicians, nursing staff, and RDs who oversee the patient's nutritional rehabilitation. Physicians, nurses, and dieticians have intensive daily contact and have been extensively trained in the intricacies of safely medically caring for this severely ill patient. The goal is to

medically stabilize by beginning safe nutrition rehabilitation, to effectuate weight gain up to 70% of IBW, and then refer the patient to a typical residential eating disorder program for more in-depth psychiatric and psychological treatment, along with continued nutritional rehabilitation and safe weight restoration.

Considerations with Normal to Higher BMIs

More recently it has been recognized that harm may occur by assuming that those with a "normal" or "higher" BMI are stable enough to access lower levels of care. Not only can these patients be presenting with a falsely elevated weight, but they can also be at risk for dangerous complications due to excessive purging behaviors followed by abrupt cessation or significant and rapid "weight disruption" from weight loss. Thus, take into account the severity of purging behaviors (also outlined in *DSM-5*) and the severity of absolute weight loss when choosing an appropriate level of care. Again, consider medical stabilization first for those showing severe weight disruption as well as those needing to safely "detox" from severe substance, laxative, or diuretic abuse. Starting in the appropriate level of care can have a significant impact on the patient's health, success in recovery, and satisfaction with treatment.

Inpatient versus Residential Facilities

Licensed inpatient facilities must meet the more stringent safety and treatment standards required for medical and psychiatric hospitals across the United States. These include standards for construction, infection control, food and medication management, and medical records. Inpatient facilities also require full-time nursing (RN/LPN) supervision and daily physician involvement. Some inpatient units are locked, and some can provide involuntary treatment when that is needed.

The licensure and regulatory requirements for the residential level of care are highly variable across states. Building and other safety requirements can be minimal. Nurse and physician involvement can also vary greatly and may be minimal.

The Joint Commission (2016) and the Commission on Accreditation of Rehabilitation Facilities (CARF, 2013) are the predominant regulatory organizations that accredit inpatient and residential psychiatric programs. Each has recently developed criteria specific to eating disorders that allow programs to achieve disease-specific accreditation. We highly recommend referring patients to programs that have met these standards, given their inherent increased focus on the care being provided.

Psychiatric Medications and Psychotherapy

Comprehensive treatment for patients with eating disorders simultaneously addresses the medical and nutritional components of the illness (e.g., weight restoration, normalization of eating, treatment of medical complications) and the behavioral and emotional aspects of the disorder. In this section we focus on how treatment addresses the behavioral and emotional parts of the illness and expand on the role of the psychotherapist and the psychiatrist within the treatment team.

At the core of the psychopathology of eating disorders are dysfunctional beliefs, emotions, and behaviors centering on food and weight, often including an overvaluation of the benefits of thinness, misinterpretation of normal digestive sensations, and efforts to lose weight or change body shape. Additionally, there is often marked fear and distress related to the prospect of changing these attitudes and behaviors. Surrounding these core symptoms are various maladaptive factors that maintain the illness for the individual (e.g., lack of sense of self, low self-esteem, fears related to taking on adult responsibilities).

The psychiatrist and psychotherapist join efforts to help the patient identify, challenge, and diminish eating disorder symptoms and maintenance factors as well as move toward recovery, with a combination of psychotherapy, experiential relearning, and individualized medications. They also focus on comorbid conditions, with both psychotherapy and the judicious use of medications.

Psychiatric Medications

No medications are approved by the US Food and Drug Administration (FDA) for the treatment of anorexia nervosa. As Mitchell, Roerig, and Steffen (2013) have said, "The treatment literature in anorexia nervosa consists of relatively few controlled pharmacotherapy studies in comparison to bulimia nervosa and binge eating disorder. Controlled studies have generally shown a lack of efficacy for pharmacological interventions." Current practice is therefore to start with nutritional treatment and psychotherapy and to consider the addition of a second-generation antipsychotic if there is a failure of weight gain. Several randomized studies of olanzapine show a modest effectiveness in helping weight gain. The largest of these was a 16-week randomized controlled trial (RCT) comparing olanzapine with placebo in 152 outpatients in which those taking the active drug achieved a gain of one pound per month. Low doses of olanzapine (2.5 to 10 mg daily) are recommended, with awareness of potential pharmacokinetic issues such as poor absorption, decreased volume of distribution, and increased percentage of free drug due to decreased body weight. Other commonly used antipsychotic medications, including risperidone and quetiapine, have shown no benefit in weight gain in small randomized trials. Aripiprazole has not been studied in randomized trials, but a retrospective study in 106 hospitalized adolescents showed greater weight gain in those who received aripiprazole compared with those who did not receive this medication.

Another medication that has shown modest success in achieving weight gain in anorexia nervosa is the synthetic cannabinoid agonist dronabinol. This medication was looked at in an RTC with 25 patients who had AN for at least 5 years. During dronabinol treatment, patients gained 1.6 more pounds than those taking placebo in a 4-week period. This medication was first approved by the FDA in 1985 for the treatment of nausea and vomiting associated with cancer chemotherapy and subsequently has been approved for the anorexia associated with HIV/AIDS.

Neither antipsychotic nor antidepressant medications have shown good efficacy in reducing distorted body image or abnormal thoughts about food and weight. These medications have also not proved helpful

in preventing relapse. The largest maintenance trial of the antidepressant fluoxetine, a selective serotonin reuptake inhibitor (SSRI), for the treatment of anorexia nervosa, reported by Walsh et al. (2006), randomized 93 patients to either 60 mg of fluoxetine or a placebo and found the medication not helpful in improving time to relapse.

At the nadir weights of AN, SSRIs have not been found effective in treating depressive symptoms. There is better evidence that SSRIs and mirtazapine may be helpful in improving depressive and anxious symptoms in patients with anorexia nervosa who have been weight restored. Antipsychotics have not been shown in randomized trials to improve anxiety or depression symptoms in AN. Small doses of anxiolytics, such as lorazepam, are often given to reduce mealtime anxiety, but there is little research evidence that this is helpful. D-cycloserine added to exposure therapy has shown some efficacy in reducing anxiety in several small studies.

Numerous other medications, including cyproheptadine, lithium, and zinc, have been looked at in randomized studies without much benefit seen. Many AN medication studies suffer from low patient enrollment and high dropout rates.

The only FDA-approved medication for bulimia nervosa (BN) is fluoxetine, which is indicated for acute and maintenance treatment of binge eating and vomiting behaviors in adult patients with moderate to severe bulimia nervosa. In three initial short-term studies, fluoxetine at 60 mg, but not 20 mg, was effective in reducing the number of binge eating and vomiting episodes. The largest maintenance trial, reported by Romano et al. (2002), lasted 52 weeks and showed greater efficacy for fluoxetine over placebo. The dropout rates were high for both arms of the study: 83% for the fluoxetine group and 92% for the placebo group. These very high dropout rates were mostly the result of illness relapse and patients' decisions, not adverse events. Also, of note, as with most BN studies, the goal in this study was reduction of binge eating and purging behaviors, not abstinence.

For patients who fail a trial of fluoxetine, one good option might be to try another SSRI, and several small randomized studies have shown efficacy for sertraline, escitalopram, and fluvoxamine. Similar to fluoxetine,

dosing is usually increased to the higher end of the usual dose for major depression. If there is a failure of a second SSRI trial, one option might be the tricyclic antidepressant desipramine, which has shown efficacy in several RCTs for BN with dosing similar to that used in depressive disorders (Crow, 2021). Another good choice could be the antiepileptic drug topiramate, which has shown efficacy in several randomized studies. Caution should be used in those who have a low normal weight or a history of anorexia nervosa given this medication's possible side effect of weight loss. Further options include trazodone and monoamine oxidase inhibitors such as phenelzine, which have shown efficacy in several randomized trials. Phenelzine can cause difficulties in bulimic patients because of the need for a tyramine-free diet, and reversible monoamine oxidase inhibitors such as brofaromine and moclobemide have not shown efficacy in trials. Finally, the antidepressant bupropion should generally be avoided because it can cause seizures in patients with bulimia or anorexia nervosa. There are suggestive data that SSRIs can help with comorbid depression in BN, but at present "there is no placebo-controlled RCT available with a focus on the use of antidepressants to treat depressive symptoms in patients with BN and a comorbid depressive disorder" (Himmerich et al., 2020).

Binge eating disorder (BED) is the most common of the eating disorders, and numerous medications have been examined for efficacy in this patient population. The only medication with FDA approval to treat BED is lisdexamfetamine dimesylate (LDX), approved in 2015. The efficacy of LDX in treating BED was demonstrated in two clinical trials that included 724 adults with moderate to severe BED. Compared with the placebo group, participants taking the active drug showed a decrease in the number of binge eating days per week and had fewer obsessive-compulsive binge eating behaviors. The most common side effects in clinical trials were anxiety, dry mouth, insomnia, constipation, and increased heart rate. Subsequently, two long-term trials have demonstrated maintenance of efficacy and safety. LDX was initially approved in 2007 to treat attention deficit hyperactivity disorder (ADHD) in patients 6 years and older. Serious risks of stimulants like LDX include psychiatric problems such as psychotic or manic symptoms as well as heart problems, includ-

ing sudden death, stroke, and myocardial infarction. Also, of note, in the United States, LDX is a Schedule II controlled substance, showing a high potential for abuse and dependence. Although some patients lose weight when taking LDX, it is not approved or recommended for weight loss. LDX is a *prodrug*, not active until absorption by the intestine and removal of l-lysine, converting the drug to dextroamphetamine.

Several other classes of medication have shown some efficacy in the treatment of BED. Antidepressant medications were among the earliest studied, and Mitchell, Roerig, and Steffen (2003) found that SSRIs "are reasonably effective agents in suppressing, albeit rarely, eliminating binge-eating disorder." SSRIs examined in RCTs have included fluoxetine, fluvoxamine, sertraline, and citalopram, and reductions were seen in binge eating and depressive symptoms. On the whole, however, significant reduction in body weight was not seen. Other antidepressants that have shown reduction in binge eating symptoms in RCTs include imipramine, a tricyclic antidepressant, and duloxetine, a serotonin norepinephrine reuptake inhibitor.

Another group of medications that have shown efficacy in RCTs looking at BED treatment include agents that are used in ADHD treatment. In addition to LDX, these include methylphenidate, a dopamine and norepinephrine reuptake inhibitor and releaser, and atomoxetine, a norepinephrine reuptake inhibitor. These have positive effects on weight loss as well as binge eating symptoms, but methylphenidate, like LDX, is a Schedule II drug with high abuse potential.

A third group of medications that have shown positive results in BED trials are the anticonvulsants topiramate and zonisamide. Both show significant decreases in binge eating and body weight, but zonisamide was felt to be poorly tolerated by patients. Topiramate has shown efficacy in two short-term trials and one long-term open-label study (Safer et al., 2020). Potential side effects such as somnolence, paresthesia, and cognitive impairment can sometimes be managed by starting with a lower initial dosage and only gradually increasing the dose.

Moreover, an additional group of medications being looked at in BED are those used in obesity treatment. A recent trial in 18 BED patients, with the combination of phentermine and topiramate, showed efficacy

in weight loss and reducing binge eating behaviors (Amodeo et al., 2019). This combination was approved by the FDA for the treatment of obesity in 2012. Phentermine is similar to its parent compound, amphetamine, in that both increase synaptic levels of norepinephrine, dopamine, and serotonin, resulting in reduction of hunger perception. Since BED is often associated with overweight and obesity in treatment-seeking individuals, this combination may be useful if it can be replicated in larger studies. Another combination that has been looked at is naltrexone and bupropion. which was approved by the FDA in 2014 for long-term weight management in patients with obesity. A prospective open-label study in 25 women with major depression and overweight/obesity found improvement in weight and depressive symptoms. At baseline, 91% of subjects had moderate or severe binge eating, and significant improvement in this was seen during the 24-week study.

For patients with BED and comorbid depression, evidence suggests that antidepressants may be helpful. A retrospective cohort study looked at 30 patients with BED and depression and found that both bupropion and sertraline reduced anxious-depressive symptoms and binge frequency.

Despite increased prevalence of bipolar disorder (BD) in BN and BED compared to the general population, "there are no studies available on the specific treatment of BD in people with eating disorders" (Himmerich et al., 2020). The following recommendations have been offered as "expert opinion and not based on trials in patients with eating disorders" (Himmerich et al., 2020). In patients with AN and BD/manic episode, a preferred option is olanzapine, with alternatives being risperidone for acute and aripiprazole for long-term treatment. For patients with BN or BED and BD/manic episode, the preferred medication is risperidone, with aripiprazole being an alternative for long-term treatment. In patients with AN and BD/depressive episode, the recommended option is olanzapine plus fluoxetine, with secondary options being aripiprazole or lamotrigine. Finally, in patients with BN or BED and BD/depressive episode, lamotrigine is recommended, with aripiprazole as an alternative.

Psychotherapy

The goals for psychotherapy with persons who have eating disorders typically include helping the patient begin to make shifts in behaviors and attitudes, develop a stronger sense of identify apart from the illness, and engage fully in a meaningful life. Some therapy approaches focus on changing dysfunctional thoughts and beliefs, while others help patients accept difficult thoughts and change how they relate to those thoughts, thereby lessening their impact. The process of psychotherapy for patients with eating disorders involves taking a dual approach to the individual. On the one hand, the psychotherapist must have a clear, objective, evidence-based understanding of eating disorders, their comorbidities, and the treatments suited for this combination of challenges. On the other hand, the clinician must also establish a sensitive engagement with the patient to form a therapeutic alliance that allows the individual to understand the functions that the eating disorder is serving and, ultimately, to let go of the eating disorder and develop new, more adaptive coping mechanisms and skills for navigating difficult emotions and experiences.

The modest evidence base for psychotherapy for patients with eating disorders has been focused on outpatient treatment. Family-based therapy is recommended by the APA, the AAP, and the National Institute for Health and Care Excellence (NICE) for the treatment of mild to moderate anorexia nervosa in children and adolescents. FBT is a 20-session manualized treatment delivered by a psychotherapist with specific family therapy training. The treatment is delivered to the entire family, with sessions once or twice a week. Several RCTs support the efficacy of this treatment for children and teens with anorexia nervosa (Couturier, Kimber, and Szatmari, 2013; Lock, 2015; Lock and Le Grange, 2013).

CBT and IPT have been approved by the APA (2006) and NICE (2017) as recommended treatments for adults with bulimia nervosa or binge eating disorder. These are 20-session manualized treatments delivered by a psychotherapist in individual appointments typically held weekly, each lasting 45–60 minutes. Randomized trials have demonstrated the efficacy of

these approaches compared with other treatments (Murphy et al., 2012; Watson and Bulik, 2013). Several randomized controlled trials using FBT have also shown it to be effective in the treatment of adolescents with BN.

There is also growing evidence for the efficacy of some of the newer "third wave behavioral therapies," such as dialectical behavioral therapy and acceptance and commitment therapy in the treatment of adults with eating disorders (Bankoff et al., 2012; Juarascio, Forman, and Herbert, 2010; Lenz et al., 2014, Walden et al., 2018.) These therapies focus on increasing awareness of one's experiences, shifting the way one relates to difficult experiences, and developing a life with purpose.

Several other adjunct interventions are commonly used in eating disorder programs, although they have not yet been empirically validated. One such approach is sensory modulation, initially developed by occupational therapists and often used with more severe forms of illness to help patients broaden their repertoire of skills for self-regulating in the face of difficult emotions or experiences. Patients with functional gastrointestinal symptoms resulting in food avoidance, rumination, or vomiting can be provided with behavioral interventions and instructed in the use of diaphragmatic breathing for symptom control. Another adjunctive approach is individual or group-based art therapy, in which making art provides an avenue for exploring and managing thoughts, feelings, and difficult experiences.

Group psychotherapy is an important component of most eating disorder treatment programs. Groups offer the experience of peer support and peer challenge, the opportunity to see parallels in the experience of others, and the chance to use peers who are further along in the treatment process as role models. Psychotherapy groups in an eating disorder program may include process groups (in which patients explore and share their thoughts, feelings, and relationships), skills-based groups (typically centered on a specific treatment model such as CBT, DBT, or ACT), body image groups (in which distorted perceptions of the body are explored and challenged), and psychoeducational groups (e.g., focusing on medication, medical issues, or nutrition). Many programs also include experiential groups, such as for supervised grocery shopping, cooking, or restaurant outings. One category of experiential group that warrants

special mention is the expressive arts groups, including art therapy, psychodrama, music, and movement therapy. These allow patients opportunities to access emotions they might otherwise struggle to name and talk about.

Summary

Given the complexity of these illnesses, the multidisciplinary treatment team is the foundation of state-of-the-art care for patients with eating disorders. Providers from numerous disciplines blend their skills to create the optimal recovery environment. The treatment delivered by this group of clinicians involves a combination of standard components (e.g., medical monitoring, nutritional rehabilitation, therapy groups) and individualized components (e.g., addressing the individual's life journey, family environment, and specific medical needs). Patients looking back on their care often cite "the team" as one of the most beneficial aspects of their treatment and one of the key catalysts in the recovery process. In sum, the assembly, integration, and ongoing collaboration of the multidisciplinary team form an invaluable aspect of the treatment for individuals with eating disorders.

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3

Medical Evaluation of Patients with Eating Disorders

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COMMON QUESTIONS

Are all degrees of anorexia nervosa and bulimia nervosa associated with significant medical issues?
Are there specific physical signs, symptoms, or laboratory results that reliably point to a diagnosis of anorexia nervosa or bulimia nervosa?
What questions need to be asked during an initial history and physical examination?
Which procedures are strongly recommended during the initial medical evaluation (e.g., X-ray, EKG)?
Which examinations are optional?
Which laboratory tests are strongly recommended?
How often do these different tests have to be repeated?

Case Study

M.P. was a 22-year-old female admitted to the residential unit of an eating disorder program for weight restoration. She gave a 12-year history of anorexia nervosa, restricting subtype. Her highest adult weight was 125 pounds, and her current weight of 97 pounds was her lowest. She denied all forms of purging and had been subsisting on less than 500 kcal per day. Her review of systems endorsed cold

intolerance, fatigue, constipation, chest pain, dizziness, and low back pain. Her last menstrual period had been 9 months ago. On physical examination, she was noted to be in no distress. At 5 feet 6 inches tall, she was 75% of ideal body weight (IBW) with a body mass index (BMI) of 15.7. Blood pressure was 94/70 mm Hg and pulse was 52. There was lanugo hair growth on the sides of her face and on her back, a normal cardiac exam, hypoactive bowel sounds, and no edema. Laboratory results revealed a normal comprehensive panel. Her white blood cell count was low, at 2.3 k/μL. The EKG showed sinus bradycardia with a rate of 50 and nonspecific ST changes. She had a recent bone density scan (DXA), with a worst Z-score of –3.1 in her lumbar spine, consistent with decreased bone density.

This chapter presents a broad general overview of the medical evaluation that is appropriate for the patient with an eating disorder. Many of the medical issues dealt with here are covered in much greater detail in other chapters, as noted throughout this review.

Most of the medical complications of anorexia nervosa (AN) and bulimia nervosa (BN) result either from starvation and weight loss or from the mode and frequency of specific purging behaviors. *Purging* indicates all forms of self-induced compensation for unwanted calories or methods to lower weight by loss of body fluids. This can be accomplished by (1) self-induced vomiting (which may be induced manually with an object such as a toothbrush, by simply tightening abdominal muscles, or by ingesting ipecac); (2) laxative and enema abuse; (3) diuretic abuse; (4) diet pill abuse; or (5) ingestion of thyroid hormone or stimulant-type medications. The first two methods account for 90% of purging behaviors, followed by the other methods in descending order of frequency. Most of the comments in this chapter regarding patients with anorexia nervosa restricting type (AN-R) are also applicable to patients with avoidant/restrictive food intake disorder (ARFID).

These are dangerous illnesses. Anorexia nervosa is a potentially life-threatening disorder and one of the most lethal psychiatric disorders. In a meta-analysis, the aggregate 10-year mortality rate for patients with AN was more than five times greater than that for age-matched controls

(Arcelus et al., 2011). About 20% of deaths resulted from suicide; the majority resulted from a direct effect of the illness. Bulimia nervosa is medically more benign than anorexia nervosa, but its mortality rate is still twice that of age-matched controls, with most deaths attributable to serious electrolyte abnormalities due to purging behaviors (Crow et al., 2009). Specifically, death is usually caused by a cardiac abnormality resulting from dangerous hypokalemia (low potassium) due to a purging behavior, or by suicide, which is generally a consequence of the common comorbid personality disorder.

Diagnosis

Most individuals who have an eating disorder will not present with symptoms directly attributable to the eating disorder and may attempt to hide evidence of disordered eating from the physician. This is especially true for individuals of normal weight who have bulimia nervosa. Symptoms, signs, and laboratory results, however, may provide valuable clues for the alert clinician.

Symptoms

Patients with eating disorders often present for medical care with nonspecific complaints of feeling cold, tired, dizzy, or with swelling, numbness, and tingling in their extremities. They can have nonspecific cardiopulmonary symptoms such as exertional fatigue, heart palpitations, or syncope (fainting) (table 3.1). Gastrointestinal (GI) complaints are especially frequent and can include postprandial fullness, early satiety, abdominal distention, abdominal pain, bloating, nausea, and constipation (Hetterich et al., 2019; Schalla and Stengel, 2019). If the physician misses these subtle clues, they can easily overlook the correct diagnosis.

Signs

The most common physical signs associated with anorexia nervosa and bulimia nervosa are listed in table 3.2 (Westmoreland, Krantz, and Mehler,

TABLE 3.1. Symptoms in individuals with eating disorders

Anorexia nervosa	Bulimia nervosa
Amenorrhea	Irregular menses
Infertility	Heart palpitations
Irritability	Esophageal burning
Depression	Abdominal pain
Exertional fatigue	Abdominal bloating
Weakness	Fatigue
Headache	Headache
Dizziness	Constipation/diarrhea
Chest pain—palpitations	Rectal bleeding
Faintness	Swelling of feet
Dysphagia (difficulty swallowing)	Frequent sore throat
Constipation	Teeth sensitivity
Abdominal pain	Depression
Heartburn	Swollen cheeks
Feeling of fullness with eating	Numbness and tingling
Polyuria	Nosebleeds
Dry skin	
Intolerance of cold	
Low back pain	
Fragility fractures	

2016). In severe AN, the emaciation of the patient usually alerts the clinician to the diagnosis, but individuals with anorexia often try to conceal their low weight. Common strategies include wearing large, loose or bulky clothing to mask their thinness and carrying hidden objects (like coins in their pockets) or ingesting water to artificially inflate their weight. Not infrequently, probably because of society's emphasis on thinness, health

TABLE 3.2. Physical signs in individuals with eating disorders

Anorexia nervosa	Bulimia nervosa
Emaciation	Calluses on the back of the hand
Hypothermia	Salivary gland hypertrophy (swollen cheeks)
Hyperactivity	Erosion of dental enamel (perimylolysis)
Bradycardia (heart rate <60 beats/minute)	Periodontal disease
Hypotension (blood pressure <90 mm Hg systolic)	Dental caries
Hypoactive bowel sounds	Facial petechiae (red dots on the face)
Dry skin	Perioral irritation
Pressure sores	Mouth ulcer
Brittle hair	Hematemesis (vomiting blood)
Brittle nails	Edema (ankle, periorbital)
Hair loss on scalp	Abdominal bloating
Yellow skin, especially palms	Cardiac arrhythmia
Lanugo hair growth on face	Rectal prolapse
Cyanotic (blue) and cold hands and feet	Hemorrhoids
Leg edema	
Heart murmur (mitral valve prolapse)	

care professionals overlook the marked degree of emaciation and thus may fail to recognize the seriousness of the patient's condition.

People with anorexia nervosa have thinning scalp hair; lanugo hair growth (downy, fine hair), particularly on the face, neck, arms, back, and legs; and sometimes a yellowish tinge to the skin called *carotenemia*. Carotenemia is a benign clinical condition characterized by a yellow pigmentation of the skin due to increased beta-carotene levels in the blood, typically the result of excessive consumption of carotene-rich foods such as sweet potatoes, squash, and carrots.

Vital signs often show bradycardia, hypotension, and hypothermia, which may present as cold intolerance and an inability to compensate

for changes in temperature. The hands and feet are frequently purplish-blue due to cyanosis and abnormalities of temperature regulation (acrocyanosis) (Strumia, 2009).

In contrast to people with anorexia, whose emaciation draws medical attention, people with bulimia nervosa often appear physically healthy. Because binge eating is often done secretively, most patients will not provide this information unless specifically asked. In addition, the associated behaviors of vomiting and abuse of laxatives, diet pills, diuretics, or ipecac are often concealed from others because of guilt or shame and must be specifically inquired about while taking the history.

Certain specific signs are present on physical examination, however, and are of some utility in detecting occult BN (Sachs and Mehler, 2016). A common sign is the erosion of dental enamel in individuals who engage in self-induced vomiting. The erosion is particularly marked on the lingual surface of the upper teeth. This is referred to as *perimylolysis* (see chapter 11). Fillings or amalgams, which are relatively resistant to gastric acid, appear to be raised above the level of the tooth surface. Other types of dental change include increased sensitivity to cold or hot temperatures and a possible increased rate of developing caries (Kisely et al., 2015). A second clinical sign is swelling of the salivary glands (sialadenosis), particularly the parotid glands on the sides of the face, a few days after cessation of purging. This swelling is usually bilateral and painless but can be pronounced (Park and Mandel, 2006). Patients sometimes refer to this as "puffy cheeks" or "chipmunk cheeks." It is seen in people with bulimia who engage in excessive amounts of self-induced vomiting and abruptly cease (see chapter 11). A third sign of diagnostic utility is calluses on the dorsum of the hand as a result of irritation of the fingers by the teeth during repeated self-induced vomiting (Russell's sign). This sign, specific to bulimia, may be more common early in the course of the illness; during later stages, patients often no longer require mechanical stimulation to induce vomiting and can simply turn their head, bend forward, and initiate vomiting.

In some patients, periorbital petechiae (small red dots around the eyes) or subconjunctival hemorrhages (bleeding underneath the conjunctivae) may be seen shortly after a forceful vomiting episode, and

there may be redness of the skin around the corners of the mouth as a result of irritation by acidic stomach contents. This is referred to as *angular cheilosis*. Fluid retention (edema) can be dramatic, particularly in the legs, after withdrawal of laxatives or diuretics or after cessation of excessive vomiting.

In both AN and BN, physical signs of past self-mutilation are not uncommon as part of the psychopathology of eating disorders, especially in BN associated with borderline personality disorder. Inquiry about this and an examination of the skin are important.

Laboratory Results

Unfortunately, objective, accurate, and specific measures of the severity of disordered eating behavior do not exist. Clinicians must therefore rely on the self-reports of their patients, who may not accurately describe the frequency of their disordered eating behaviors. There are not many consistently reliable screening laboratory tools for occult eating disorders. A good patient-doctor relationship, which sets the tone for candid responses to validated screening questions, is key. There are, however, some helpful clues in blood testing that may speak to the presence of an eating disorder (table 3.3).

Abnormally low serum potassium levels are highly specific for covert purging behaviors but are not highly sensitive (see chapter 5) (Mehler and Walsh, 2016). In general, the body has an impressive ability to maintain normal serum levels of potassium in the absence of severe gastrointestinal or renal losses of potassium. Pure dietary deficiencies of potassium are relatively rare in the absence of purging behaviors, which deplete the body's potassium stores. The serum bicarbonate level is also often abnormal in bulimia. High levels (>30 mEq/L) are most consistent with either self-induced vomiting or diuretic abuse; low levels are most consistent with acute diarrhea due to laxative abuse. Patients with purely restrictive anorexia nervosa are rarely at risk for hypokalemia, even if their weight is very low, until they begin to refeed. In fact, generally, their blood chemistry values are remarkably normal, and they may die from AN-induced starvation with normal blood chemistry values, including a

TABLE 3.3. Laboratory results that might point to a diagnosis of eating disorder

Anorexia nervosa (food-restricting subtype)	Bulimia nervosa
Hypercholesterolemia	Hyperamylasemia
Hypoglycemia	Hyponatremia
Elevated enzymes in liver function tests	Hypokalemia
Bradycardia (heart rate <60 beats/min)	Hypomagnesemia
Low white blood cell count (leukopenia)	Metabolic alkalosis
Low red blood cell count (anemia)	Non-gap metabolic acidosis
Low LH, FSH, estradiol, or testosterone levels	
Low thyroxine levels (T4)	
Hypophosphatemia (low phosphorus)	
Hyponatremia (low sodium)	
Osteopenia/osteoporosis	
Low leptin levels	

remarkably preserved serum albumin level. Therefore, the presence of a substantially low albumin level in anorexia nervosa should prompt the clinician to look for independent medical comorbidities. Severe hyponatremia (low sodium level) can result from diuretic abuse in a patient with bulimia who is further volume depleted from vomiting or in a patient with anorexia nervosa who is drinking 5–10 liters of water per day to obtain a full feeling without eating. If therapeutic correction of the serum sodium is too rapid, these patients risk developing the serious neurologic disorder known as *osmotic demyelination syndrome*.

Serum amylase levels are frequently raised above the normal range in people who vomit regularly. The hyperamylasemia is mostly salivary based rather than pancreatic in origin and results from enlargement of salivary glands from overeating and vomiting. As with potassium levels, the role of serum amylase measurements as a diagnostic test is limited. Nevertheless, amylase levels may positively correlate with frequency of binge eating and purging. Because an elevated amylase level

can also be seen with acute pancreatitis, it may be worthwhile to follow up elevated amylase levels with a lipase test. If the lipase level is normal, then one can conclude that the elevated amylase level is likely of salivary rather than pancreatic origin, but again, it may be due to binge eating.

In summary, the presence of an abnormally low potassium value and an abnormally high serum amylase level, together with an elevated bicarbonate level, in an otherwise healthy young woman seems to be specific for frequent purging behavior. Nonetheless, its sensitivity as a screening tool is poor. As with all patients, a careful history directed toward uncovering problematic eating behaviors that might cause serious medical complications is the most important source of information, for which no laboratory tests can substitute—especially for patients in the right clinical setting who are between the ages of 13 and 30, when eating disorders are most prevalent.

Hypercholesterolemia has been noted in up to 50% of patients with anorexia nervosa but is probably clinically insignificant. It is most likely a reflection of an elevated "good" cholesterol subtype (HDL) rather than an elevated "bad" cholesterol subtype (LDL) and does not need medicinal treatment.

Elevations of liver transaminase levels can be seen before refeeding in patients with severe anorexia nervosa as a result of malnutrition or after aggressive refeeding with dietary plans containing high-dextrose caloric sources (Nagata et al., 2015). Also, in anorexia nervosa, there may be abnormalities in the complete blood count (CBC). Leukopenia (low white blood cell count), anemia (low red blood cell count), and thrombocytopenia (low platelet count) may be present, especially as weight loss becomes more severe, with leukopenia being the most frequently low of the three blood cell lines (Walsh, Blalock, and Mehler, 2020). The anemia is mostly normocytic, less often macrocytic, and rarely microcytic. The latter requires an evaluation to rule out iron deficiency.

In addition, anorexia nervosa is associated with global endocrine dysregulation. The numerous endocrine disturbances seen are due to the body's adaptation to the lower energy state seen in chronic starvation and can include low estrogen, low testosterone, high cortisol levels, and

abnormal thyroid function tests (Schorr and Miller, 2017). A rapid, potentially permanent loss of bone mineral density can be seen with decreased sex hormones, elevated cortisol levels, and growth hormone resistance, and this can result in a lifelong increased risk of fracture (see chapter 10). Levels of leptin, a hormone derived from adipocytes that inhibits appetite, are low in anorexia nervosa. This can lead to changes in brain activity in areas involved in emotional and cognitive control of appetite, which may represent a deleterious maintaining factor for eating disorder behaviors (Cassioli et al., 2020; Li et al., 2017).

Differential Diagnosis

In the food-restricting subtype of anorexia nervosa, the symptoms and signs are usually those that occur secondary to caloric restriction and weight loss. Many of the manifestations are similar to the complications observed in patients with starvation due to other causes, including fatigue, dizziness, and cold intolerance. Yet, there are a few important differences. For example, most starving patients will note lethargy and inability to exercise, whereas patients with anorexia may still amazingly continue to exercise excessively in attempts to lose weight. Patients with weight loss from other organic causes will express grave concern about their weight loss, while patients with anorexia will be unconcerned about their marked weight loss. Moreover, common causes of weight loss due to malabsorption will present with diarrheal symptoms, which are generally absent in anorexia nervosa, or with symptoms of adrenergic excess such as sweating and rapid heart rates, also absent in AN. The clinician should also bear in mind that the most common cause of substantial weight loss in young adolescent females in western societies is anorexia nervosa. Patients with AN are often spared the starvation-related complications (kwashiorkor and marasmus) of the inhabitants of developing countries because of their adequate ingestion of vitamins, even though their total caloric intake is very low.

Especially for patients presenting with atypical features, clinicians should make a careful assessment to exclude other organic pathologies

such as occult malignancies, chronic infections, diabetes, or malabsorption syndromes. Selected laboratory tests can facilitate the diagnosis of other illnesses, such as hyperthyroidism (TSH level), which may cause weight loss and simulate anorexia nervosa. Inflammatory bowel disease (IBD) affecting the intestine can cause abdominal pain associated with eating that may lead to food restriction, incorrectly suggesting a diagnosis of anorexia nervosa; however, the central psychopathology of anorexia (see chapter 1) and normal albumin levels are lacking in IBD. Other common psychiatric disorders are also associated with weight loss, such as depression, schizophrenia, substance abuse, somatoform disorders, and dissociative disorder. When starvation begins with self-induced vomiting and the patient endorses a drive for thinness with a morbid fear of weight gain, a purely medical cause is rarely found. Similarly, various other medical conditions may present with symptoms that are difficult to distinguish from those of bulimia nervosa. For example, illnesses such as IBD and connective tissue syndromes (scleroderma) may cause abnormal upper GI motility and result in many of the same symptoms as in people who have bulimia and abuse laxatives or engage in self-induced vomiting.

Generally speaking, these diagnoses can be made by a thorough medical history and physical examination together with laboratory screening tests. Complex and expensive work-ups are rarely indicated. The guiding principle is that a diagnosis of an eating disorder is made not by ruling out all possible medical disorders but by confident determination of the presence of an eating disorder through a combination of focused physical examination, a thoughtful use of laboratory testing, and screening questions (Academy for Eating Disorders, 2016) (table 3.4).

Patients with eating disorders frequently complain about constipation. A detailed history of the patient's perception of constipation is required, however, because constipation is defined differently by patients and physicians. Experts (using Rome IV criteria) define constipation as having fewer than three bowel movements per week. Patients with bulimia often have unrealistic beliefs about the need to have frequent bowel movements. In BN, constipation is usually caused by dependence on stimulant laxatives, which can in the long run, albeit rarely, result in the death of the

TABLE 3.4. Differential diagnosis for anorexia nervosa and bulimia nervosa

Anorexia nervosa	Bulimia nervosa
Hyperthyroidism	Scleroderma or other connective tissue disorders with GI involvement
HIV	Infections
Malabsorption	Inflammatory bowel disease
Addison's disease	Malabsorptive states
Diabetes mellitus	Gastroesophageal disorder (GERD)
Malignancy, especially lymphoma, stomach cancer	Bowel obstruction
Irritable bowel syndrome	Esophageal stricture
Chronic infection, especially tuberculosis, AIDS, fungal disease	Peptic ulcer disease
Hypothalamic lesion or tumor	Gastric outlet syndrome
Cystic fibrosis	Parasitic intestinal infection
Superior mesenteric artery syndrome	Chronic pancreatitis
Inflammatory bowel disease (Crohn's disease, ulcerative colitis)	Diabetes
Parasitic intestinal infection	Hypothalamic lesion or tumor
Chronic pancreatitis	Zenker's diverticulum
Psychiatric disorders associated with weight loss	Brain tumor

colonic wall myenteric nerve plexus and severe constipation. In AN, colonic motility is delayed by the lack of oral intake, even if there has never been abuse of stimulant laxatives (Mitchell and Crow, 2006) (see chapter 6).

Given the frequency of comorbid psychiatric conditions, a complete psychiatric evaluation should be performed to diagnose comorbid depression, substance abuse, and personality disorders. The patient should also be evaluated for impulsivity and suicidality.

Physical Examination

There are two distinct reasons for conducting a physical examination of patients with eating disorders. The first is to elicit the signs related to the medical complications of AN or BN and, in the process, to begin to define a treatment plan to stabilize patients medically (American Psychiatric Association, 2006). The second is to use the examination in a motivational context and to confront patients with objective medical evidence that the disordered eating behavior has adversely affected their physical health (see chapter 17). The information is particularly relevant for physicians in light of the long-term increased mortality rate, especially for patients with severe anorexia nervosa. The facets of such a physical examination are summarized in table 3.5.

Obtaining accurate measures of a patient's height and weight to determine BMI and percentage of ideal body weight, as well as assessing the degree of emaciation, are important. The IBW is easily calculated using the Hamwi formula, starting with 100 pounds for a female 5 feet tall and then adding 5 pounds for each additional inch. Thus, if the patient is 5 feet 5 inches tall and weighs 100 pounds, her IBW is 125 pounds, and she is at 80% of IBW. For males, starting at 106 pounds and adding 6 pounds for each inch above 60 inches is the common approach. Per *DSM-5*, anorexia is categorized as severe anorexia nervosa for a BMI between 15 and 16 and extreme for a BMI <15 (American Psychiatric Association, 2013). The patient's current weight should not be self-reported, because patients with eating disorders are often unreliable in self-estimations of body weight. When able, patients should empty their bladder, change into a gown, and do a jumping jack (to ensure there are no hidden

TABLE 3.5. Physical examination for patients with eating disorders

▪ Weight, height, BMI, % ideal body weight	▪ Dental examination
▪ Pulse and blood pressure	▪ Cardiac examination
▪ State of hydration	▪ Abdominal examination
▪ Skin examination	▪ Extremity examination (edema and cyanosis)
▪ Neurologic examination (strength)	▪ Musculoskeletal examination (weakness)

weights) before being weighed. If an adult, the clinician should attempt to establish the patient's premorbid weight, and for children and adolescents, growth charts should be obtained.

Examination of the mucous membranes and skin turgor as well as measurement of orthostatic blood pressure help to assess the patient's state of hydration. An examination of the abdomen is necessary for patients with AN or BN. Inspection, auscultation, and light palpation are recommended. For most patients, further evaluation will not be necessary unless significant abnormalities are detected or serious complaints have been articulated.

For individuals with bulimia, a careful dental examination for evidence of dental caries and enamel erosion should be performed. The erosion has a characteristic pattern, tending to occur on surfaces that have maximum exposure to the vomitus, particularly the palatal surfaces of the maxillary teeth and the occlusal surfaces of posterior teeth, especially on the lingual sides (see chapter 11).

Pulse and blood pressure should be checked for all patients. Sinus bradycardia of fewer than 60 beats per minute is present in many patients with anorexia nervosa because of an energy-conserving slowing of the metabolic rate and vagal hyperactivity. Hypotension, with a blood pressure of less than 90/60 mm Hg, is often present in these patients, related to chronic volume depletion and cardiac changes, and may result in episodes of dizziness and frank syncope. Orthostatic blood pressure measurements should include supine followed two minutes later by a standing blood pressure for those with excessive purging and for patients with anorexia nervosa (Sachs et al., 2016).

Laboratory Tests

Mandatory Tests

Several screening laboratory tests are indicated for all people who present with an eating disorder, and several specific procedures and ancillary tests are indicated only in certain situations (table 3.6). The choice of laboratory tests at the initial evaluation will be guided by the results of the history and the physical examination. Most people with eating

TABLE 3.6. Laboratory tests for patients with eating disorders

- CBC with differential
- Serum electrolytes
- Calcium, magnesium, phosphorus levels
- Liver function tests and albumin
- Serum salivary amylase level
- Vitamin D (25-hydroxy)
- Prealbumin
- Serum BUN, creatinine levels
- Blood glucose level
- T4, TSH levels
- Testosterone level (males)
- Urinalysis
- Stool examination for occult blood if abdominal complaints or if anemia is present
- EKG
- Bone densitometry (DEXA)

disorders should have a screening laboratory panel of blood tests. A CBC with differential, electrolytes, blood urea nitrogen (BUN), creatinine, blood glucose, calcium, phosphate, liver function tests, thyroid-stimulating hormone (TSH), and 25-hydroxy vitamin D, as well as testosterone in males, and a pregnancy test in females, are indicated for most people with eating disorders, especially those being admitted to a hospital inpatient unit or a residential or partial hospitalization program.

Low white and red blood cell counts can occur with anorexia nervosa, reflecting bone marrow suppression due to malnutrition. An increase in leukocyte counts, signaling an infection, might be overlooked due to low baseline values. Therefore, increased vigilance is necessary with patients who have AN and are being evaluated for possible infections. Also, these patients may not manifest a fever in infectious states, because of the baseline hypothermia associated with anorexia nervosa.

Serum electrolytes should be checked routinely for all patients to detect possible electrolyte imbalances. Specifically, elevated serum bicarbonate levels indicating metabolic alkalosis are frequently seen in patients who vomit regularly. Hypokalemia is often present in patients with bulimia. Because hypokalemia is a potentially life-threatening abnormality, electrolyte function should be evaluated for patients with BN who are actively purging. Furthermore, many patients with electro-

lyte abnormalities are clinically asymptomatic. Hypophosphatemia can lead to serious complications and often develops during the early stages of refeeding of patients who have AN (see chapters 4 and 5).

Liver enzyme abnormalities are relatively uncommon among patients with eating disorders at near-normal weights. Low body weight can cause hepatic damage, however, and hepatic dysfunction (apoptosis) may occur in patients with anorexia nervosa more often than in the general population (Saito et al., 2008). Elevated liver enzymes, aspartate aminotransferase (AST) and alanine aminotransferase (ALT), occur in patients with more severe forms of anorexia nervosa. In particular, AST and ALT, as well as indirect bilirubin, may be increased during refeeding, especially if nutritional intake emphasizes foods with high glucose content (see chapter 4).

A disproportionate rise in BUN compared with creatinine may be caused by dehydration from diuretics, laxative abuse, or decreased fluid intake in patients with anorexia nervosa. Generally, this ratio is less than 20:1. Blood glucose often decreases with more severe weight loss in anorexia nervosa but is generally asymptomatic in the 40–60 mg/dL range in contrast to non-AN patients. Hypoglycemia is a bad prognostic sign in anorexia nervosa, however, because it indicates depleted hepatic glycogen and glucose stores, necessitating urgent follow-up.

Because the symptoms and signs of both hyperthyroidism and hypothyroidism can mimic eating disorders, thyroid function tests are appropriate. Patients with anorexia nervosa might be misdiagnosed as having hypothyroidism. The pattern of thyroid indices in patients with anorexia is a normal or low T4, decreased T3, increased reverse T3, and normal TSH. This represents a physiologic adaptation to starvation that need not be treated with thyroid supplementation. It is referred to as *euthyroid sick syndrome* (see chapter 9). Patients may even abuse thyroid hormone therapy to further lose weight. Occasionally, TSH is mildly elevated, but it generally returns to normal a few weeks after refeeding is under way and thus should be rechecked before committing a patient to thyroid hormone replacement therapy.

Young people with insulin-dependent diabetes mellitus sometimes overdose their insulin to compensate for binges or underdose to create

hyperglycemic-induced urinary frequency and weight loss. This was previously referred to as *diabulimia* but is more recently known as a dual diagnosis of an eating disorder and diabetes mellitus type 1 (ED-DMT1) (see chapter 10).

Given that conception can occur during recovery from anorexia nervosa even before the return of menstruation, a serum pregnancy test is recommended. Women with bulimia nervosa also have an elevated risk of unplanned pregnancy, partly due to menstrual irregularities and partly to more impulsive personality traits seen in those with BN that can lead to risky sexual behaviors.

Optional Tests

The specific items of the history, the complaints of the patient, and the findings on physical examination should direct the physician to the need for further evaluation. This includes selective use of chest or abdominal X-rays, electromyography (EMG), examination of muscle enzymes (creatinine phosphokinase, or CPK), computed tomography (CT), magnetic resonance imaging (MRI) scans of the head, or GI endoscopy if there is evidence of GI blood loss or significant esophageal, gastric, or abdominal pain.

If abuse of diuretics or laxatives is suspected but is denied by the patient, urine samples to detect the surreptitious use of these agents may be considered in rare cases, as well as stool samples for phenolphthalein testing as evidence of laxative use. The presence of possible neurologic dysfunction may necessitate an electroencephalogram (EEG) or a head CT or MRI to rule out seizure disorder or structural brain illness, especially in atypical eating disorders.

Screening for osteoporosis should be a standard part of the management of individuals with anorexia nervosa of more than one year's duration, as low bone mineral density can have long-lasting consequences, including loss of height, stress and fragility fractures, and chronic pain secondary to vertebral compression fractures (Workman, Blalock, and Mehler, 2019). The results can often be used as a motivational tool. Ordering a dual-energy X-ray absorptiometry (DEXA) scan for measure-

ment of bone density of hip and spine for all men and women with a history of 9–12 months or more of AN is good practice (see chapter 10). These findings may be used to guide the physician in giving advice about the level of physical activity suitable for the patient and support recommendations for adequate calcium intake and full restoration of body weight to restore normal menstrual activity.

On the basis of the initial evaluation, judicious use of medical subspecialists to evaluate any identified major medical complications may be indicated. Certainly, at a minimum, a primary care physician who is well versed in the medical issues of these patients should be closely involved in their care.

An initial electrocardiogram (EKG) with a rhythm strip is desirable for any patient who has BN or has AN and is severely emaciated. It is good practice to obtain a baseline EKG for any patient with an eating disorder admitted to an inpatient or residential unit. The EKG commonly shows sinus bradycardia; low-amplitude P waves and QRS voltages, reflecting reduced cardiac size; nonspecific ST-segment and T-wave abnormalities; and occasionally, U waves associated with hypokalemia and hypomagnesemia. Dangerous QTc abnormalities may also be present (see chapter 5). Follow-up EKGs should be obtained when medications associated with prolonged QTc are instituted.

Ongoing Medical Management

Most of the above-mentioned abnormalities and complications are fully reversible with weight restoration and cessation of purging behavior. Osteoporosis and cognitive functions may be the glaring exceptions. Careful, ongoing medical management is an essential component of the overall treatment plan for patients with eating disorders. The frequency and content of medical follow-up visits must be individualized for each patient, depending on the severity and chronicity of the eating disorder and the associated medical complications. Patients who have severe AN or BN need to be stabilized on an inpatient specialized medical unit. Those with mild to moderate disease should be seen a few times per week during the early phases of their treatment plan or weight restoration

program. Those who are stable for outpatient treatment need to have an informed primary care provider as part of their multidisciplinary team.

Daily monitoring of weight, pulse, and blood pressure is strongly advised for all markedly underweight inpatients who have restricting anorexia or are purging. For many patients with eating disorders, especially those who have bulimia, electrolyte abnormalities are the most prominent medical complication. The clinician should closely follow up on these patients to ensure adequate correction of electrolyte values, as well as patients with anorexia nervosa during the early phase of refeeding.

Follow-up evaluations should be performed more frequently if medical complications have been identified, if the patient with AN is being aggressively refed or is more than 20%–25% below IBW, if the patient's condition has deteriorated, or if medical interventions are planned that might affect these parameters. All health care workers caring for these patients must be aware that eating disorders pose serious medical risks and predispose the patient to development of serious medical complications. Thus, it is important to have close medical oversight as part of the multidisciplinary treatment team.

Summary

Eating disorders can have a deleterious effect on many different body systems. It is imperative that patients with eating disorders have a health care provider who is well versed in the medical complications of eating disorders, especially as the prevalence of these disorders increases, and most especially as the severity of the disorder increases.

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4

Nutritional Rehabilitation

Practical Guidelines for Refeeding Patients with Anorexia Nervosa

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COMMON QUESTIONS

How is ideal body weight calculated for adults?
What classification is used to define the severity of anorexia nervosa and the need for inpatient versus outpatient treatment?
What is the *refeeding syndrome*? Which patients are at risk?
Are there general guidelines to follow in formulating a dietary plan when refeeding a patient who has anorexia nervosa?
How has the approach to refeeding evolved in the past few years?
What laboratory tests should be done and how often should they be checked when refeeding a patient who has anorexia nervosa?
How are caloric needs ascertained and calculated?
What physical findings should the clinician follow during the refeeding process?
What alternative nutritional approaches may help reverse the course of refractory, severe anorexia nervosa?
What are *enteral nutrition* and *total parenteral nutrition*? What is their role in anorexia nervosa?
What determines whether adequate calories are being provided for the nutritional rehabilitation of the patient with anorexia nervosa?

How does one approach a patient's lack of expected weight gain on a specified dietary regimen? Refeeding the patient who has anorexia nervosa is essential to successful treatment. Judging by outcome studies from around the world, most experts agree that one cannot effectively treat anorexia nervosa without first restoring body weight. It is also clear that without a concerted refeeding effort, no meaningful psychotherapy can take place, because these patients have starvation-induced cognitive deficits. Weight restoration, however, may be one of the most challenging and frustrating parts of the recovery process for many patients with anorexia nervosa (AN) or avoidant/restrictive food intake disorder (ARFID), given the inherent nature of their illness and their fear of eating.

Traditionally, centers that treat patients with moderate and severe degrees of AN have used a combination of behavioral techniques, cognitive restructuring, and a progressive program of oral caloric intake to achieve the goal of weight restoration. Different types of enteral feeding and central parenteral nutrition (CPN) may be indicated for rare, more refractory cases. Clinicians caring for these patients must be facile in the art of the refeeding process, given the multitude of potential clinical and biochemical caveats. Two cases illustrate some of these points.

Case Study 1

J.W., a 40-year-old female, had a 15-year history of anorexia nervosa. She was transferred to an inpatient eating disorder program after an apparent suicide attempt through self-inflicted stab wounds. On admission, she was found to be almost 40% below her ideal body weight (IBW) (height 5 feet 6 inches, weight 83 pounds). Significant laboratory data included a low hematocrit of 21% (normal range 38%–45%) secondary to recent blood loss, sodium of 127 mg/dL (138–143), and serum albumin of 3.4 g/dL (3.5–5.0). Her dietary history indicated an average intake of 400–600 kcal per day. She reported weekly use of laxatives and daily exercise of 1 to 2 hours. Her predicted resting energy expenditure (REE) was 1,050 kcal per day, based on the Harris-Benedict formula.

J.W. was started on 1,000 kcal per day divided into three meals and progressed to 1,600 kcal per day during the ensuing 7 days. After the first week, she developed slight edema in her legs, which was thought to be secondary to her lacerations rather than a manifestation of the refeeding syndrome. Her liver function tests became slightly elevated (AST and ALT), and she had intermittent problems with asymptomatic hypoglycemia, with a nadir fasting venous blood sugar of 38 mg/dL (normal range 60–100). An evening snack was added.

By the end of the second week, her liver function tests began to improve, and her edema resolved. Blood sugar levels remained low. Calories were progressively increased to 2,000 kcal per day, but her weight remained unchanged. An indirect calorimetry study was obtained. It calculated an REE of 911 kcal per day, which indicated that she should have been gaining weight on her current diet plan. J.W. subsequently admitted to covert excessive exercise during evening hours. Once this was addressed in her treatment plan, her hypoglycemia resolved, and she gained a total of 14 pounds over the next 4 weeks. She subsequently gained an additional 18 pounds after discharge on a dietary plan that provided 3,100 kcal per day, and she has maintained a weight of 105 pounds over the past 3 years. She did not, however, have resumption of her menses.

Case Study 2

S.C., a 41-year-old female, had a 20-year history of anorexia nervosa. She had had multiple unsuccessful inpatient admissions for treatment of this condition. Over the preceding 18 months, she had lost 29 pounds and was infected with a fungal pneumonia. In addition, she complained of cold intolerance, fatigue, and an inability to concentrate. She was therefore readmitted to an inpatient eating disorder unit for the treatment of her anorexia nervosa.

Despite intensive psychotherapy and a defined nutritional plan in the hospital's regimented eating disorder program, S.C., who was 5 feet 10 inches tall, remained at her initial admission weight of 89 pounds. She also developed purulent bronchitis caused by the virulent bacteria *Enterobacter* and *Klebsiella*. Because of her persistent

refractory anorexia nervosa, with a body weight more than 40% below ideal, and her adamant refusal of a gastric tube, the decision was made to judiciously initiate therapy with an alternative mode of nutritional rehabilitation using CPN.

S.C. received a total of 9 weeks of CPN. When CPN was discontinued, she had gained 28 pounds, which she has maintained for the 2 years since the CPN was completed. The CPN, consisting of a solution containing 50% dextrose and an 8.5% amino acids solution, was started at a rate of 25 mL/hour, and the infusion rate was cautiously advanced over the course of 3 weeks to achieve a maximum intake of 3,600 kcal per day. Calories were also partly derived from a 10% intralipid solution for the first week of CPN, which was then advanced to a 20% infusion of 500 mL every other day. Laboratory values were checked every other day for the first 2 weeks, and weekly for the last 2 weeks of therapy. Aside from some minor elevations in serum liver transaminase levels, there were no other significant biochemical changes. During the course of the CPN, S.C. also worked intensely with a dietician to choose oral foods at each meal. By the time the CPN was discontinued, the number of calories she derived from oral feeding had increased from 300 to 2,700 kcal per day. During the last 2 weeks of CPN therapy, the rate of infusion was progressively reduced, and her weight gain was successfully maintained after 6 months of follow-up.

Background

In general, the nutritional needs and goals of individuals with anorexia nervosa are based on attaining their healthy IBW. For a female, IBW may be calculated as 100 pounds for 5 feet of height plus 5 pounds for each additional inch. For a male, the only difference in the calculation is to start at 106 pounds and use 6 pounds per additional inch. Thus, patients' condition can be classified as mild, moderate, severe, or critical based on whether they are 10%, 20%, 30%, or more below IBW. An alternative way of classifying patients is based on body mass index (BMI), which is calculated based on height and weight and is readily available in tables

for an array of heights and weights. Specifically, the BMI value is obtained by dividing an individual's weight in kilograms by height in meters squared. The utility of BMI is somewhat limited, however, in that it does not provide any measure of body composition or nutritional status (Yates, Edman, and Aruguete, 2004), and it overestimates health status in males. In addition, standardized tables of "ideal body weight" may not be appropriate for use with adolescents. Normative data from the National Center for Health Statistics' height and weight tables may be better suited for adolescents 12–18 years of age.

Assessing IBW may also differ for people with a history of constitutional thinness. It is important to be aware of the difference between patients who have constitutional thinness and those struggling with AN, as patients with eating disorders often claim they are just "naturally thin," using this as a barrier to achieving full weight restoration. How do we decipher between those patients who are, in fact, "naturally thin" (or have constitutional thinness) and those who are just trying to avoid fully weight restoration? Constitutional thinness is defined as a BMI <17.5 with an almost normal hormone panel and, for women, normal menses (Estour et al., 2017). People who are constitutionally thin do not have a desire to lose weight, and disordered eating behaviors are not observed during meals. One lab test that could be a helpful tool to help differentiate between constitutionally thin and AN-R patients is a free triiodothyronine (T3), which can be included with blood work without any preparation needed. Free T3 has been shown to be significantly lower in AN-R patients than in constitutionally thin patients. Another lab value that may be helpful to assess constitutional thinness versus AN-R is a leptin level. Leptin levels in constitutionally thin patients are found to be significantly lower than a normal counterpart but higher than an AN-R patient. A final thing to consider is a weight history when it is available. There is evidence to support that most AN-R patients trended near the 50th percentile for BMI for age prior to restricting nutrition, whereas constitutionally thin patients usually trended around the 5th percentile during childhood and adolescence. It is important to differentiate between constitutional thinness and AN-R patients to avoid aggressive or

ineffective treatment and to understand that constitutional thinness should not be considered an exclusion of the AN-R diagnosis (Estour et al., 2017).

According to the American Diabetes Association, certain ethnicities, such as Asian Americans, are at high risk for health conditions, specifically diabetes, at lower BMIs than other Americans (Hsu et al., 2015). This is something to take into consideration when assessing ideal goal weights for patients of this ethnicity who need weight restoration while in treatment. Individualizing care and weight gain goals for patients with constitutional thinness or from varying ethnic backgrounds can help ensure that the patient is weight restored appropriately, ensure an appropriate length of stay to achieve full weight restoration (i.e., not overweight restoration), and help set achievable, manageable, and sustainable weight restoration goals.

When a patient is set to begin the refeeding process, there must be, from the onset, an earnest attempt to achieve agreement among caregiver, dietician, and patient as to what the range for target weight is going to be. Also, spending time educating a patient with anorexia nervosa about metabolism and how it may change during the process of weight restoration may prevent future difficulties and reduce stress for the patient. Generally, weight gain to or very near IBW is an acceptable goal, regardless of the mode of refeeding. Some view a healthy weight as being the weight at which normal menstruation occurred in the past. If amenorrhea persists, however, it may be necessary for the patient to achieve full IBW or even a little above it. Discharging patients before they reach a minimum normal weight is associated with an increased rate of readmission (Castro et al., 2004; Redgrave et al., 2015). Current practice is generally to admit a patient to the hospital for inpatient treatment, medical stabilization, and nutritional restoration when the patient's weight is more than 30% below IBW (American Psychiatric Association, 2006). This is to minimize morbidity during the early stages of refeeding and to avoid severe medical complications, the rate of which increases at these low weights (Gaudiani et al., 2012). It is worth noting that as opposed to most other forms of malnutrition, where low serum

albumin levels correlate with poor nutritional status, albumin is typically within the normal range even in extreme forms of anorexia nervosa (Gaudiani, Sabel, Mehler, 2014).

Refeeding Syndrome

Before understanding the specifics of refeeding, it is important to consider a potentially catastrophic complication that can occur during this process: refeeding syndrome (Solomon and Kirby, 1990). This syndrome occurs in significantly malnourished patients, those who have had marked caloric restriction, during the early phase of nutritional replenishment—whether by the oral, enteral, or parenteral route. The risk of refeeding syndrome is directly correlated with the degree of weight loss resulting from the anorexia nervosa. Thus, patients who are more than 30% below IBW should initially be refed during an inpatient hospitalization (Bermudez and Beightol, 2004).

In the early twentieth century, the heart was believed to be immune to the effects of chronic malnutrition. During World War II, however, experiments were performed using conscientious objectors who voluntarily agreed to lose a certain percentage of their body weight. As a result of this weight loss, they developed low blood pressure and reduced cardiac size, as seen on a chest radiograph. This finding led to a progressive realization that the heart can be adversely affected by weight loss as well as by subsequent refeeding. There is an even earlier reference to refeeding syndrome in the famed novel *The Count of Monte Cristo*, by Alexandre Dumas, first published more than 175 years ago: "He turned his eyes toward the still steamy soup that the jailor had put on the table, raised the bowl and drank. Then he had the courage to stop, for he had heard of shipwrecked survivors found depleted by hunger, dying from eating gluttonously, overly substantial food."

The mechanism of potential cardiovascular collapse in refeeding syndrome is multifactorial (table 4.1). First, the reduced heart mass that accompanies weight loss makes it difficult for the heart to handle the increase in total circulatory blood volume that accompanies refeeding. The result of this can be heart failure. Even though the heart mass does

TABLE 4.1. American Society for Parenteral and Enteral Nutrition (ASPEN) consensus criteria for identifying adult patients at risk for refeeding syndrome

	Moderate Risk: 2 Risk Criteria Needed	Significant Risk: 1 Risk Criteria Needed
BMI	16–18.5 kg/m2	<16 kg/m2
Weight Loss	5% in 1 month	7.5% in 2 months or >10% in 6 months
Caloric Intake	None or negligible oral intake for 5–6 days OR <75% of estimated energy requirement for >7 days during an acute illness or injury OR <75% of estimated energy requirement for >1 month	None or negligible oral intake for >7 days OR <50% of estimated energy requirement for >5 days during an acute illness or injury OR <50% of estimated energy requirement for >1 month
Abnormal prefeeding potassium, phosphorus, or magnesium serum concentrations	Minimally low levels or normal current levels and recent low levels necessitating minimal or single dose supplementation	Moderately/significantly low levels or minimally low or normal levels and recent low levels necessitating significant or multiple dose supplementation
Loss of subcutaneous fat	Evidence of moderate loss	Evidence of severe loss
Loss of muscle mass	Evidence of mild or moderate loss	Evidence of severe loss
Higher-risk comorbidities	Moderate disease	Severe disease

Source: da Silva et al., 2020.

revert toward normal with weight gain, the first few weeks of refeeding require more attention to a patient's cardiovascular status (Lamzabi et al., 2015). Studies in anorexia nervosa have shown diminished cardiac output as a result of heart muscle atrophy that accompanies unhealthy weight loss. (Goldberg, Comerci, and Feldman, 1988).

Second, changes in serum levels of phosphorus as well as potassium and magnesium are key variables in refeeding syndrome. The mechanism by which hypophosphatemia (low phosphorus levels) develops during refeeding is mainly due to the glucose content of the food. The glucose load increases insulin release, which in turn drives phosphorus

and potassium into the intracellular space. There is also an incorporation of phosphorus into newly synthesized tissues and high-energy compounds during refeeding. The resultant low serum phosphorus levels are accompanied by depletion of the high-energy compound adenosine triphosphate (ATP), which impairs the contractile properties of the heart and can evolve into congestive heart failure (figure 4.1). Refeeding-induced hypophosphatemia can also result in diaphragmatic muscle fatigue and respiratory failure. Other sequelae of refeeding-induced hypophosphatemia include red blood cell membrane disruption (hemolysis), skeletal muscle injury (rhabdomyolysis), and seizures. Rhabdomyolysis is diagnosed by the finding of an abnormally high serum level of the muscle enzyme creatinine phosphokinase (CPK). Low levels of serum potassium and magnesium can also cause cardiac irritability and arrhythmias, along with skeletal muscle weakness.

The best predictor of risk for developing refeeding hypophosphatemia is BMI; the lower the BMI, the greater the risk (Brown et al., 2015). This is certainly true as BMI falls to the 10–12 range. It may be less well correlated at higher BMIs in the 14+ range (Richardson, Huniewicz, and

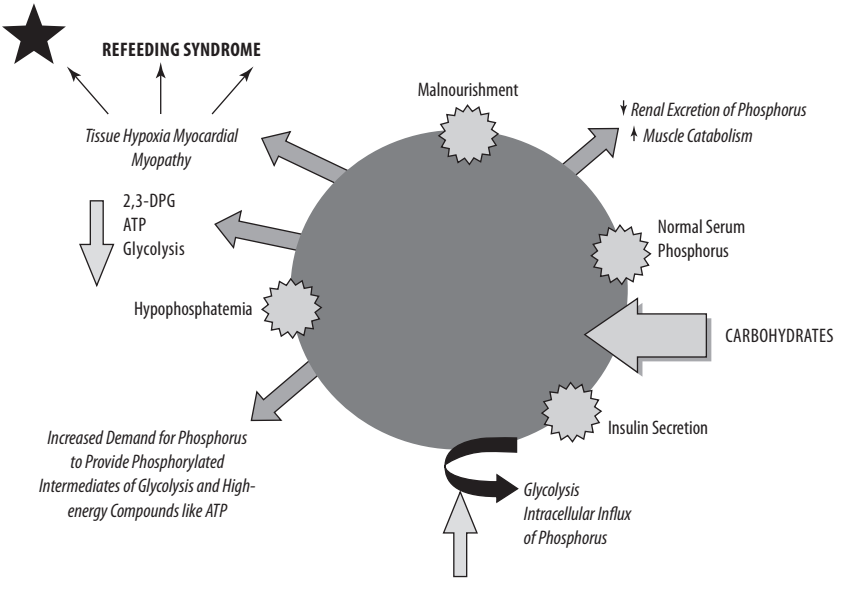

Figure 4.1. Refeeding hypophosphatemia

Paslakis, 2020). Clinicians must regularly check phosphorus levels, perhaps daily, for the first 1–2 weeks of refeeding, and replenish phosphorus levels with either powdered phosphorus or the pill form. There is no need for prophylactic phosphorus. Rather, the more prudent approach is to assiduously monitor blood levels closely and replace phosphorus when low, while increasing calories, especially in the earlier stages of refeeding (Reber et al., 2019).

Practical Tips for Refeeding

The medically worrisome refeeding syndrome is preventable (table 4.2). The older dictum on dietary calorie repletion to "start low, advance slow" has changed, with most studies supporting a more aggressive refeeding plan (Golden et al., 2013; Redgrave et al., 2015; Haas et al., 2020) and the safety and feasibility of a more accelerated renutrition approach. But caution should still be exercised, especially for patients with severely low BMIs. The caloric requirements for the female patient with anorexia nervosa can be accurately calculated with the Harris-Benedict equation for basal energy expenditure (BEE):

$$\text{BEE} = 6.55 + (9.6 \times \text{body weight in kg}) + (1.8 \times \text{height in cm}) - (4.7 \times \text{age in years})$$

Dieticians are familiar with this calculation. Alternatively, BEE can be measured by indirect calorimetry, a relatively simple procedure involving a breathing test, available in many hospitals through the nutrition department. Calorimetry is based on measurement of carbon dioxide production and oxygen consumption. The BEE value basically reflects the energy used when the body is at rest; in clinical practice, the terms BEE and REE are used interchangeably (Cuerda et al., 2007). The BEE must be multiplied by a stress factor (1.2–2.0) to determine total energy expenditure (TEE). In a critically underweight patient, the BEE will likely need to be multiplied by a stress factor of 2.0–2.5 to determine TEE. In general, TEE exceeds BEE or REE by 10%–60%, depending on how active the patient is. The TEE should generally not be the starting point of caloric repletion. Rather, it is a target to achieve days to weeks after the

TABLE 4.2. Strategies to avoid refeeding syndrome

▪ Identify patients at risk (e.g., any patient who is chronically malnourished, is severely underweight, or has not eaten for 7–10 days).
▪ Measure serum electrolyte levels and correct abnormalities *before refeeding.*
▪ Obtain serum phosphorus and electrolyte values every day for the first 5–7 days, or until not falling, and thereafter at a reduced regular interval.
▪ Attempt to slowly increase daily caloric intake by 300–400 kcal every 3–4 days until the level of caloric intake is sufficient to produce adequate weight restoration.
▪ Clinically monitor the patient carefully for the development of complications.

initiation of refeeding. The REE measured by indirect calorimetry gives a slightly lower value for caloric needs than the values estimated by the Harris-Benedict formula. But, in reality, a beginning empirical caloric intake of 1,400–1,800 kcal per day is a reasonable starting point for most patients and obviates the need for calorimetry or other calculations.

Although it is important for patients to experience a significant degree of weight gain to maximize the chances of a successful recovery, the optimal dietary intervention for effectuating this is unknown. There has been a distinct and surprising paucity of research on nutritional interventions in critical illness and in anorexia nervosa, and thus a rigorous evidence base is lacking (Haynos et al., 2016; Taylor et al., 2016). The usual goal is a weight gain of 3–4 pounds per week as an inpatient and 1–2 pounds per week for outpatients. Several approaches have been promoted (Mittnacht and Bulik, 2015). The main tenet is to check blood chemistry values frequently in the early stages of the refeeding process and review those results that same day. Caloric levels usually begin at approximately 1,400–1,800 kcal per day and are increased by 300–400 kcal every 3–4 days. The treatment corollary of this is to individualize intake based on the achieved rate of weight gain. Augmenting the diet with a liquid supplement in the early stages of refeeding to achieve the prescribed daily calorie goal is an effective strategy for weight gain. Most patients require a caloric range that peaks at 3,000–3,600 kcal per day, but some may require upward of 4,000 kcal per day, especially younger males. It may be difficult to define precisely the factors that consistently correlate with the number of calories needed to gain one pound.

In general, starved patients who have anorexia nervosa are metabolically inefficient and may require a little more than the expected 3,500 kcal, beyond the expected caloric needs to restore a pound of body weight.

Some simple general rules to follow (summarized in table 4.3) are:

1. Caloric intake should rarely exceed 70–80 kcal/kg of body weight but can be upwards of 80+ kcal/kg of body weight when a patient is critically underweight (<55% IBW), typically ranging from 80 to 100 kcal/kg.
2. For the patient who has more severe anorexia, begin a diet at 30–40 kcal/kg.
3. Protein intake should generally not exceed 2 g/kg of body weight, but again, at very low body weights, this is often exceeded. Patients who are critically underweight may regularly require closer to 4 grams of protein per kilogram without showing any signs of kidney injury.
4. Meal planning should be individualized.
5. Macronutrient distribution should be approximately 50% carbohydrate and 25% protein and fat, but this may not work for all patients.
6. Weight gain should be in the range of 3–4 pounds per week for inpatient and residential patients.
7. Micronutrient deficiencies most commonly include zinc, vitamin D, copper, selenium, and vitamin B_1 (Hanachi et al., 2019)

Males generally peak at 4,000 kcal per day and females at 3,500 kcal per day. All approaches require vigilant clinical and laboratory monitoring and individualization of the dietary plan depending on the rate of weight gain and the laboratory and clinical course of the patient (table 4.4).

TABLE 4.3. Guidelines for refeeding patients with anorexia nervosa

▪ Nutritional rehabilitation should be individualized.
▪ Start refeeding at 30–45 kcal/kg/day.
▪ Intake should rarely exceed 70–80 kcal/kg/day.
▪ Limit protein intake when possible to less than 2.0 g/kg/day.

TABLE 4.4. Practical guidelines for refeeding

▪ Start at 1,000–1,800 kcal/day.
▪ Advance by 300–400 kcal every 3–4 days.
▪ Maximum is usually around 3,500 kcal/day for females and 4,000 kcal/day for males.
▪ Monitor clinical examination and phosphorus/electrolyte levels closely.
▪ Begin with low-lactose, low-fiber diet.
▪ Consider zinc to improve dysgeusia.

Currently, on most eating disorder units, caloric intake is being increased faster than in years past (Matthews et al., 2018). These increases should always be guided by the patient's actual pattern of weight gain rather than the standard goal of 3–4 pounds per week. It is not uncommon, however, to see no weight gain or only 1–2 pounds of weight gain during the first weeks of nutritional repletion. Drastic increases in dietary calories should not always be instituted in response to this. Rather, judicious consideration of covert purging behaviors or other complications should be undertaken. Similarly, caloric intake must be modified occasionally because of changes in REE during refeeding, and such modifications will affect the rate of weight gain. Change in REE of the patient with anorexia nervosa seems to be a real phenomenon: REE is often lower in these patients early in the refeeding process than at later stages (Forman-Hoffman, Ruffin, and Schultz, 2006). This may be attributable to reversion to a euthyroid state from euthyroid sick syndrome or to increased anxiety. It is possible that these changes contribute to the difficulty of ongoing, sustained weight gain during the latter stages of refeeding (Salisbury et al., 1995). Caloric requirements for weight restoration are best determined by monitoring an individual's rate of weight gain. Given this dynamic process, caloric requirements may have to be recalculated if weight gain is not achieved as expected during the refeeding process, or consideration must be given to potential confounding sources of weight loss, such as covert exercise or other modes of purging.

Potential Complications

From a clinical standpoint, rates of weight gain greater than 3–4 pounds per week are usually not nutritionally sound or sustainable and may represent edematous fluid retention or retained bowel contents due to constipation. Aside from the aforementioned numeric guide to weight gain, some clinical parameters should be closely followed. Specifically, vital signs contribute useful daily information. Individuals with AN generally have bradycardia (<60 beats per minute). Although there are other potential reasons for tachycardia (>100 beats per minute), the presence of an elevated supine heart rate, especially during the early stages of refeeding—even if just in the 80–90 beats per minute range—might be a harbinger of refeeding syndrome and cardiac compromise. Thus, even if patients technically do not have tachycardia, a sudden sustained increase in the pulse to greater than 80–90 beats per minute, in the supine position, deserves evaluation. Daily monitoring of vital signs, daily nursing assessments, and physician rounds are clearly valuable during the first weeks of the refeeding process.

Checking for the presence of edema in the shin area is another worthwhile practice during early refeeding; its presence can be an ominous sign. However, edema may also occur as a minor complication. During the early stages of refeeding, insulin secretion normally increases, which, in turn, induces sodium retention by increasing kidney tubular sodium reabsorption (Yucel et al., 2005). Low sodium diets should not generally be used as part of the nutritional plan, however, as there is emerging evidence that this may have deleterious effects on levels of aldosterone secretion (Doukky et al., 2016). If spot urine sodium level is greater than 80, however, lower dietary salt content might be considered. These clinical findings deserve a thorough medical evaluation, with rare consideration to decreasing the rate of refeeding. In addition, because of the delayed stomach emptying (gastroparesis) and prolonged colonic transit time found in the severe stages of anorexia nervosa, these patients often complain of abdominal bloating and constipation (Trees et al., 2016). A bowel regimen, a minimal amount of

fiber, and adequate hydration may help alleviate these symptoms during the early refeeding process (see chapter 6).

A multitude of potential fluid and electrolyte aberrations can occur during the refeeding process, especially early on and in cases of more severe AN or ARFID. As the body shifts from a catabolic to an anabolic state, potassium, phosphorus, and magnesium are incorporated into the newly synthesized tissues and used for intermediary metabolism; this phenomenon can cause low serum levels of potassium, phosphorus, and magnesium. Phosphorus is the key electrolyte in refeeding syndrome, as mentioned above (O'Connor and Nicholls, 2013). Frequent initial monitoring of the patient's blood chemistry values can avert these problems. Early in the refeeding process, checking serum chemistry values every day or every other day is a reasonable plan. These checks can be reduced to weekly once the patient has consistently gained weight and has a relatively stable blood profile. Ultimately, with continued weight gain and stable laboratory results, monitoring can be even less frequent. The major emphasis, however, should be on serum phosphorus levels because, again, refeeding hypophosphatemia that goes undetected or untreated is the main chemical cause of refeeding syndrome.

Alternative Modes of Refeeding

Although progressive oral refeeding is the basic mode of refeeding for most patients with ARFID or AN, there are alternatives to this traditional mode. The oral refeeding plan, with a strict behavioral protocol, is nearly always the first choice because it provides a less invasive and more therapeutic method of treatment. The dietary goal must be to move from the reduced-calorie approach of anorexia nervosa to a truly balanced nutritional program, with adequate calories to balance energy expenditures and the patient's developmental stage, while avoiding underfeeding.

There are definite indications for enteral feedings and even parenteral feedings in anorexia nervosa (Mehler, 2008; table 4.5). These alternative modes of refeeding, however, are not a panacea for the treatment of this disorder, and they cannot be recommended as routine therapy for these patients. Some potential criteria for their use are (1) persistent

TABLE 4.5. When to use nutrition support

- Oral refeeding should almost always be the number one choice
- Consider use of enteral nutrition if:
  - There is a persistent failure to weight restore with other standard diet therapies
  - Patient has life-threatening weight loss
  - Patient has a worsening psychological state
  - Patient has additional medical comorbidities
- If using enteral nutrition:
  - Attempt to place the tube postpyloric to help reduce fullness
  - Consider use of a calorie-dense formula (1.5 kcal/mL–2.0 kcal/mL)
  - Attempt to use nocturnal tube feeding vs. continuous if using in tandem with oral intake
- Consider parenteral nutrition for severe and refractory cases only
  - Examples: a bowel obstruction, an ileus, severe cases of SMA syndrome
  - Use in a hospital setting only to help monitor lab values and reduce risk of infection

failure to restore weight with other standard dietary therapies, (2) life-threatening weight loss, (3) worsening psychological state despite standard treatments, and (4) medical comorbidities that preclude oral food. A patient's odds of recovery decline as the time spent continuously ill with anorexia nervosa lengthens, and thus, on rare occasions, these alternative modes of weight restoration might be reasonable if judiciously deliberated.

Surprisingly, controlled studies of nutritional treatments for anorexia nervosa are essentially nonexistent. Enteral nasogastric (NG) feedings or percutaneous endoscopic gastrostomy–based (PEG) feedings have both been used for more refractory cases of anorexia. Both enteral modes of nutritional delivery use liquid supplements that contain 1–2.0 kcal/mL. The only difference is whether the tube that delivers the enteral feed is inserted at the bedside through the nose and down the esophagus into the stomach (NG) or inserted surgically through the skin above the stomach and advanced into the stomach and secured (PEG). These two modes of enteral feeding can be used as a continuous flow of supplement, or just nocturnally, or as a bolus feed, which takes only 5–10 minutes to push in, with the daily caloric amount divided into two or three bolus-type feeds (Falcoski et al., 2020).

Dieticians are invaluable in calculating the rates and amounts of enteral feeds necessary to achieve weight gain. Using a more calorie-dense

formula, such as a 2.0 kcal/mL, can reduce the overall volume of tube feed the patient needs to ingest. This, coupled with using a NG tube that is weighted and ideally can be placed or navigated postpyloric, can help negate the feelings of fullness that are commonly associated with gastroparesis. Using a more calorie-dense formula can also help reduce the time needed to run the formula and the amount of time the patient needs to be hooked up to the tube feed, which can help ease psychological distress associated with the ingestion of calories. When possible, it is most accurate to order tube feeds as a total volume for the day versus a rate for a set amount of time. Volume-based feeds tend to be more accurate for many reasons and allow flexibility and consistency for both the nursing staff and the patient. Volume-based tube feed is preferred because it accounts for the patient whose tube may get clogged in the middle of a feed, which could potentially lead to the tube needing to be replaced, which would cause the tube feed to be stopped. If this happens, nursing can assess the total volume ingested and infuse the remaining amount of formula after the tube has been placed. Volume-based feeds can help support patients by not allowing them to engage in behaviors such as kinking or pinching the tube or manipulating the hourly rate; if the patient is participating in these behaviors, volume-based feeds allow the team to have additional time in the day to replace any missed or lost calories and, in turn, ensure that the patient is receiving the full amount of nutrition. When using volume-based feeds, consider running the total volume for <24 hours per day (i.e., 21–22 hours/day) to account for disconnecting the tube for activities of daily living, as well as circumstances that may arise throughout the day. For patients who volitionally vomit their tube feed, or in whom refractory fullness persists despite the use of a prokinetic agent, a naso-jejunal tube, often placed under fluoroscopic guidance, may be a useful, albeit rare, alternative.

In some instances, nutrition support may need to be delivered involuntarily. The *British Journal of Human Nutrition and Dietetics* discusses the importance of using the "least restrictive practice" when involuntarily tube feeding a patient (Resmark et al., 2019). Because restraints are sometimes necessary when involuntarily tube feeding patients, it is important to consider how to keep them restrained for the least amount

of time to preserve their psychological state as much as possible. The journal recommends using a high-calorie, low-volume formula and bolus feeding the patient as few times as possible so that the patient receives the lifesaving nutrition they need in the least restrictive way possible. The study observed eight instances of bolus tube feed combined with water flushes and Calogen (a high-calorie, long chain triglyceride), ranging from 779 mL fluid to 1120 mL fluid/bolus. Patients reported tolerating these bolus feeds with minimal nausea and no reported vomiting. Bolus feeding patients in this manner allowed for the least amount of time necessary being restrained compared with running a cyclic tube feed, which could necessitate a longer amount of time a patient may need to be restrained.

Fullness and discomfort, as well as tube manipulation, are problems less associated with parenteral nutrition, which is nutritionally comparable to enteral feeds. Parenteral nutrition is more expensive, however, and is also fraught with serious catheter-related complications. There are no randomized trials to guide the use of parenteral versus enteral nutrition in anorexia nervosa. These different modes of nutritional support have been compared in other medical patients. Recent evidence indicates that the parenteral route may not be more dangerous than the enteral route (Harvey et al., 2014). Yet, it is best avoided unless there is a compelling reason to use it.

Although standard protocols exist, parenteral nutrition must be viewed as a treatment modality prescribed only for severe and refractory rare cases, with utmost discretion and on an individual basis. It is beyond the scope of this chapter to discuss all of its specifics. Although CPN is a powerful tool and has been used successfully for patients with anorexia nervosa who have comorbid gastrointestinal issues that preclude oral caloric intake (Mehler and Weiner, 2007), its administration may be associated with mechanical, infectious, and metabolic difficulties. It is therefore best reserved for use in medical centers with expertise in dealing with such potential difficulties and for patients whose condition is truly refractory to more traditional refeeding programs.

When administering parenteral nutrition, it is important to be aware of the patient's glucose infusion rate (GIR) as a means to control the

patient's blood sugar. A GIR that is too high can result in hyperglycemia and possibly the need to alter the patient's macronutrient distribution (consider increasing lipid) or to start insulin. Clinicians who decide to alter the macronutrients and increase lipid should be mindful of hyperlipidemia and continue to monitor for this as well. The American Society for Parenteral and Enteral Nutrition (ASPEN) recommends not exceeding a GIR of 4–7 mg/kg/min. In a critically underweight patient, one might see that the GIR regularly exceeds 4–7 mg/kg/min. In these instances, it is recommended that the blood glucose be checked per the facility's protocol (typically every 4–6 hours). If the patient is hyperglycemic and the macronutrient distribution cannot be adjusted, it is appropriate to do a risk/benefit analysis to consider if it is more beneficial to control the hyperglycemia via insulin or to reduce the overall caloric intake and possibly compromise weight restoration to help control the patient's blood glucose. Alternatively, if parenteral nutrition (PN) is stopped too abruptly, a patient could be at risk for rebound hypoglycemia. Therefore, if you need to stop your patient's PN and are not starting oral or enteral nutrition, ASPEN recommends tapering the infusion rate a few hours before discontinuing the PN. If you need to rapidly discontinue the PN, you may need to infuse a dextrose containing fluid 1–2 hours after discontinuing PN to prevent rebound hypoglycemia (da Silva et al., 2020).

Summary

Although nutritional support of the malnourished patient is a most important component of the care plan for individuals with anorexia nervosa, nutrition regimens can cause adverse effects if used injudiciously. Because rapid refeeding can result in a catastrophic sequence of events, caution and restraint must be used when initiating therapy, together with careful early monitoring of blood chemistry values and the patient's pattern and rate of weight gain. A well-balanced, nutritional oral diet plan is generally the optimal approach for refeeding malnourished patients with anorexia nervosa. Clearly, the approach to refeeding has become more aggressive. This is mostly appropriate if tempered with assiduous monitoring and clinical evaluations, especially early in

the refeeding program. Yet, many unresolved questions remain about this critical treatment paradigm for patients with anorexia nervosa. At this time, we must be ever vigilant and circumspect as the tapestry of evidence is woven in this critical field.

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5

Evaluation and Treatment of Electrolyte Abnormalities

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COMMON QUESTIONS

Which patients with eating disorders develop electrolyte abnormalities? How common are these abnormalities?
Who is at risk of developing electrolyte abnormalities?
Which electrolyte abnormalities are most common in patients with eating disorders?
Is a particular pattern of electrolyte abnormalities associated with a specific mode of purging behavior?
What are the available classes of abused diuretics? How do they differ?
What is pseudo-Bartter's syndrome? How is this condition managed?
How is edema avoided or treated during the correction of electrolyte abnormalities in patients with bulimia nervosa?
How should the clinician treat the fluid and electrolyte abnormalities encountered in these patients?
What is the optimal treatment for severe hypokalemia?
What are the critical levels of potassium, sodium, and bicarbonate that necessitate a patient's emergency hospitalization?

Case Study

L.V., a 29-year-old female with a long history of AN-BP (anorexia nervosa, binge-purge subtype), presented to an inpatient eating

disorder unit for medical stabilization and the beginning of weight restoration. She had a long history of anorexia nervosa that began when she was in high school, at the age of 14. She was on the cross-country team, and in an effort to train more aggressively and build her endurance, she began a strict exercise regimen. She concomitantly and gradually restricted her caloric intake to the point of consuming only about 800 kcal per day. Following a traumatic event, her disordered eating behaviors changed, and she began to binge and subsequently purge through self-induced vomiting of all ingested calories. She had been to multiple eating disorder treatment facilities but had relapsed following each treatment course.

L.V.'s illness was further complicated by frequent visits to the emergency department, when not in treatment, because of weakness, dizziness, near syncope, and palpitations. Her work-up consistently revealed hypokalemia, metabolic alkalosis, and hypochloremia. Treatments in emergency departments consisted of aggressive resuscitation with intravenous fluids, electrolyte replacement, and discharge to home. Her outcome after emergency room visits was always the same: she gained 10–20 pounds of edema weight within a few days. This aggravated her body dysmorphia, further traumatized her, and made her very resistant to entering any kind of treatment facility for fear of edema formation and rapid weight gain.

At the time of admission to the inpatient unit, L.V. had been purging 10–15 times daily and had been treated in the emergency department 2 days prior, for palpitations and dizziness. On the day of admission, she was found to be tachycardic to 115 beats per minute and hypotensive to 85/50 mm Hg. She was 5 feet 5 inches tall and weighed 93 pounds. Examination revealed an emaciated woman with enlarged parotids and dry mucous membranes. Results from her cardiac, pulmonary, and abdominal examination were normal, but she had striking lower extremity edema up to the midthighs bilaterally.

Laboratory tests revealed a normal complete blood count and normal liver function. But her potassium level was low, at 2.6 mmol/L (normal 3.5–5.0); her bicarbonate level was high, at 38 mmol/L (normal 22–28); and her chloride was low, at 94 mmol/L (normal 101–112). She

was given gentle intravenous hydration with normal saline at a rate of 50 mL per hour, potassium repletion, and spironolactone at 50 mg per day, and she began a progressive oral nutrition plan of 1,400 kcal.

Within one day of hospitalization, L.V.'s vital signs improved, her electrolytes had normalized, and her IV fluids were stopped. Over the next 2 weeks, she was compliant with treatment, and her kilocalories were gradually increased up to 2,600 daily. The edema almost completely resolved, and the spironolactone was discontinued. This was the first time in her life that after entering treatment, she had experienced gradual resolution of her edema instead of its worsening.

Frequency

Patients with anorexia nervosa of the restricting subtype (AN-R) may have abnormalities in their liver transaminase levels (aspartate aminotransferase, AST; alanine aminotransferase, ALT) and bone marrow dysfunction evident from their blood counts. Laboratory analysis should show normal electrolytes, however, even for patients with extremely low body weight (Mehler, 2001). Conversely, patients who engage in purging behaviors as part of their eating disorder, whether in the form of vomiting, diuretic misuse, or laxative abuse, commonly have electrolyte abnormalities. This is true for patients with the binge-purge subtype of anorexia nervosa (AN-BP) and for those with bulimia nervosa (BN). A study by Mitchell et al. (1983) documented the frequency of fluid, electrolyte, and acid-base disturbances encountered in patients with bulimia. Almost 50% had electrolyte abnormalities. The most common abnormality was an elevated serum bicarbonate (metabolic alkalosis), present in 27.4% of the patients. Other abnormalities observed were hypochloremia (low chloride; 23.8%), hypokalemia (low potassium; 13.7%), decreased serum bicarbonate (8.3%), and hyponatremia (decreased sodium; 5.4%). There are complex interrelationships among electrolytes, and an abnormality in one should prompt further investigation and measurement of other electrolytes (Connan, Lightman, and Treasure, 2000). In Miller et al.'s study (2005) examining the prevalence of common medical findings in an outpatient eating disorders popula-

tion, 24% of the cohort exhibited purging behaviors; overall, the most common electrolyte abnormalities were hypokalemia and hyponatremia. Hypokalemia was present in 20% of the study participants, and 48% of them reported a history of regular purging; hyponatremia was present in 7% of the participants. When examining the electrolyte abnormalities at an inpatient/residential level of care in a population defined by 28% AN-BP and 24% BN, the following abnormalities were found in those engaging in purging: hypokalemia was the most frequent electrolyte abnormality (69%), followed by metabolic alkalosis (57%), and hyponatremia (26%) (Mehler et al., 2018). Thus, despite discordant data on the most frequent metabolic abnormality seen in eating disorders of the purging variety, the top three most common disturbances remain hypokalemia, metabolic alkalosis, and hyponatremia. The normal laboratory ranges for the electrolytes that are most frequently altered by purging behaviors are shown in table 5.1.

Another observed abnormality is hypomagnesemia (low magnesium), which is most commonly seen in individuals who have bulimia and abuse diuretics. Frequency of hypomagnesemia is difficult to discern because of a wide range in the reported incidence (16%–60%) but seems to commonly develop about four to five days into refeeding (Raj, Keane-Miller, and Golden, 2012; Birmingham, Puddicombe, and Hlynsky, 2004). It is generally accompanied by hypokalemia because the two cations are handled in a similar fashion by the kidney.

Electrolyte abnormalities are less frequent in patients who have anorexia nervosa without associated purging behavior, except for hyponatremia. Gentile et al. (2011) evaluated 38 common laboratory tests for

TABLE 5.1. Normal serum electrolyte ranges in laboratory tests

Electrolyte	Concentration range (mmol/L)
Bicarbonate (HCO_3^-)	22–28
Chloride (Cl^-)	101–112
Potassium (K^+)	3.6–5.2
Sodium (Na^+)	138–145

patients with AN, all of whom had a BMI below 17.5. The study found that 60% of the patients indeed had no abnormalities on laboratory chemistry evaluation of their blood. Regarding hyponatremia, one study found an incidence of 16% in those with AN-R at an inpatient/residential level of care (Mehler et al., 2018), and another study found a similar rate (14%) in an inpatient setting in AN-R patients with a mean BMI of 12.9 kg/m^2 and additional clinical and laboratory evidence of volume depletion (Caregaro et al., 2005), similar to the frequency of hyponatremia found in those engaging in purging behaviors. Hyponatremia in AN-R patients, however, likely develops as a result of the kidneys' inability to clear free water, as opposed to the effects of dehydration on the kidneys seen in those engaging in purging. Also, unlike patients with BN, patients with AN-R do not have a significant metabolic alkalosis along with elevated BUN levels. In fact, if significant hypokalemia, hypochloremia, or a metabolic alkalosis is present in a patient who supposedly has anorexia nervosa, it is most likely due to a coexistent covert purging behavior disorder that the patient is attempting to conceal (Wolfe et al., 2001). This is an important clinical fact. The presence of a low albumin level in a patient thought to have pure anorexia nervosa, or the finding of electrolyte abnormalities, should prompt a thorough search for additional causes aside from the AN diagnosis (Krantz et al., 2005).

Clinical Manifestations

The most common electrolyte abnormalities encountered in patients with purging behaviors are, as noted above, hypokalemia, hyponatremia, metabolic alkalosis, and hypochloremia; patients may also present with hypomagnesemia and rarely hypocalcemia (low calcium) (Stheneur, Bergeron, and Lapeyraque, 2014). No specific constellation of presenting symptoms enables the clinician to diagnose such abnormalities based on history and physical examinations alone, given that the clinical manifestations of electrolyte abnormalities are nonspecific and vague. The most common patient complaints accompanying marked aberrations in electrolytes include weakness, fatigue, dizziness, constipation, myalgias, paresthesia, and depression.

Hypokalemia (<3.6 mmol/L) causes generalized muscle weakness, cramping, paresthesia, and fatigue, as well as constipation and, occasionally, heart palpitations. If severe (<2.5 mmol/L), it can also cause rhabdomyolysis (muscle breakdown), significant cardiac arrhythmias, and sudden death. Indeed, hypokalemia is extremely dangerous, especially when potassium is very low, and in females is associated with an elevated risk of death (Jansen et al., 2015). In a study of patients who presented to an emergency department and were diagnosed with hypokalemia, only 49% of those with severe hypokalemia were symptomatic; their most common symptoms were weakness and muscle pain. Typical electrocardiographic (EKG) changes resulting from hypokalemia may include flat or inverted T waves, ST-segment depression, and prominent U waves, which can exceed the amplitude of the T waves. Cardiac complications and EKG abnormalities due to hypokalemia are rare if the potassium level exceeds 3 mmol/L and the decrease has occurred slowly—unless there is concomitant heart disease, in which case complications increase when serum potassium is less than 3.9 mmol/L. The finding of hypokalemia in an otherwise healthy young female is highly specific for the presence of a covert eating disorder. Thus, besides replenishing potassium, there should be inquiry about the potential cause of the hypokalemia.

Hyponatremia in the eating disorders population can be a consequence of dehydration, such as in purging disorders (due to fluid loss in emesis, stool, and diuretic effect); water loading; excessive water intake (free water intake exceeding water excretions); or extreme restriction of water and sodium intake as seen in AN-R. Dehydration is most commonly seen with purging behaviors; water loading is mostly found in cases of restricting anorexia nervosa, as a result of low solute load in the kidney and an inability to clear free water (Russell and Bruce, 1966). Symptoms in mild to moderate hyponatremia (120–135 mmol/L) can be vague, such as nausea, vomiting, headache, and confusion. In severe hyponatremia, when the sodium level is below 120 mmol/L, the symptoms can range from delirium to impaired consciousness, seizures, and cardiopulmonary collapse (Henry, 2015).

Metabolic alkalosis is generally asymptomatic; however, when moderate to severe (serum bicarbonate >35 mmol/L), it may cause compensatory

respiratory adaptations in which patients take fewer and shallower breaths (Feldman et al., 2012). This leads to symptoms of tachypnea and, when prolonged, respiratory insufficiency. Hypomagnesemia may also complicate these symptoms and contribute to arrhythmias and sudden death. Although hypomagnesemia and hypocalcemia are less common in bulimia, they can, when severe, also result in tetany.

Pathophysiology

Metabolic alkalosis is one of the most common metabolic abnormalities in patients with bulimia nervosa. In those who have metabolic alkalosis due to active purging through diuretic abuse or self-induced vomiting, the serum bicarbonate level, which is normally 22–28 mmol/L, ranges between 30 and 40 mmol/L. The more severe the metabolic alkalosis, the more likely it is that the purging behavior is self-induced vomiting.

The pathogenesis of this metabolic alkalosis varies according to the mode of purging behavior. With self-induced vomiting, the metabolic alkalosis develops in part because of the acid lost directly in the vomitus. More importantly, however, vomiting also leads to loss of sodium chloride (NaCl) and water. This results in a state of decreased intravascular volume (hypovolemia) resulting from dehydration. The body's adaptive response to a state of dehydration (to prevent fainting) is to stimulate the kidney's renin-angiotensin-aldosterone axis. This aids in sodium and water retention through the function of aldosterone, in an effort to prevent hypotension and syncope. Aldosterone, a hormone produced in the adrenal glands, acts on the kidneys to increase salt absorption and bicarbonate reabsorption at the expense of potassium and hydrogen. It thus leads to worsening metabolic alkalosis and hypokalemia as the body preferentially reabsorbs salt and water and, in exchange, wastes potassium and acid in the urine. This type of metabolic alkalosis is occasionally referred to as a *contraction alkalosis*, or a sodium chloride–responsive metabolic alkalosis, as it is primarily due to a state of dehydration, or volume *contraction*, and is corrected with an infusion of saline. This entire cascade is the body's normal protective response

to prevent dehydration and its symptoms, such as hypotension, dizziness, and syncope, in the presence of ongoing and frequent purging.

The metabolic alkalosis seen with surreptitious use of diuretics is also the result of intravascular volume depletion. Three main classes of diuretics are available on the market: thiazides, loop, and potassium-sparing diuretics. Thiazide diuretics (such as hydrochlorothiazide and metolazone) inhibit sodium and chloride reabsorption in the distal renal tubule, resulting in natriuresis and kaliuresis (sodium loss and potassium loss in the urine) and thus hypovolemia. Therefore, the typical electrolyte abnormalities are hyponatremia, hypokalemia, and metabolic alkalosis. Loop diuretics (such as furosemide) act in the kidney's loop of Henle to block sodium reabsorption, which also leads to natriuresis, kaliuresis, and hypovolemia. Potassium-sparing diuretics (such as spironolactone) act on sodium channels in the kidney. Thus, although natriuresis and a decrease in vascular volume are achieved, there is no potassium loss in the urine (Mascolo, Chu, and Mehler, 2011). These are all prescription diuretics and most often are abused by health professionals who have access to such medications. Over-the-counter (OTC) diuretics are much less potent and less likely to cause major electrolyte aberrations. The active ingredients in OTC diuretics are caffeine, ammonium chloride, and/or pamabrom. The side effect common to all classes of diuretics abused by patients with eating disorders is hypovolemia. In general, metabolic alkalosis seen with diuretics is milder than that resulting from vomiting, which, again, is associated with the most severe cases of metabolic alkalosis. A good rule to remember is that serum bicarbonate concentrations greater than 38 mmol/L are almost always due to self-induced vomiting. Elevated serum bicarbonate levels can also be a useful covert laboratory finding when a patient is denying purging behaviors, and this finding is almost always indicative of excessive vomiting or abuse of diuretics.

The constellation of laboratory abnormalities found in patients who purge by self-induced vomiting or diuretic abuse—namely, metabolic alkalosis, hypokalemia, and hypochloremia resulting from a state of dehydration—is known as pseudo-Bartter's syndrome (Bahia et al., 2012).

In 1962, Frederic Bartter and colleagues described a syndrome of hypokalemia, hyponatremia, hypochloremia, and metabolic alkalosis with an associated hyperaldosterone state in two adolescents. This syndrome was later discovered to be due to defects in electrolyte transporters that led to hypovolemia and thus upregulation of the renin-angiotensin-aldosterone axis. Although the biochemical changes that result from purging are the same as those described by Bartter, the electrolyte transporter in patients with eating disorders is intact—thus the name *pseudo*-Bartter's syndrome.

Although laxatives are an ineffective means of weight control, their abuse is fairly common, with studies reporting a prevalence of 10%–40% in people with eating disorders (Roerig et al., 2010). Self-induced vomiting and abuse of stimulant laxatives probably account for upward of 80% of all purging behaviors. Whereas purging by means of vomiting or diuretic abuse always leads to metabolic alkalosis, laxative abuse can give the initial picture of either metabolic alkalosis or mild metabolic acidosis. Significant acute diarrhea typically causes a hyperchloremic metabolic acidosis (also called a non-gap acidosis) as a result of the large amount of bicarbonate lost in the stool. The acid-base abnormality seen with chronic laxative abuse, however, is more commonly metabolic alkalosis, with low serum chloride and potassium levels; this is consistent with pseudo-Bartter's syndrome, resulting from volume depletion and dehydration due to loss of diarrheal fluids. The alkalosis tends to be mild, with bicarbonate levels in the range of 30–34 mmol/L, whereas the hypokalemia is usually more marked. The primary reason for hypokalemia is the loss of potassium in the diarrheal stools.

The typical serum and urinary electrolyte findings in the three types of purging behaviors discussed above are summarized in tables 5.2 and 5.3. Engaging in purging once or twice a week may not cause any significant electrolyte disorder, and thus the finding of normal electrolytes does not completely rule out the presence of BN symptoms. The corollary of this is that the finding of an abnormally low potassium level or an elevated bicarbonate level is not consistent with a patient's report that they rarely purge or that the purging behaviors have receded (Mehler, 2001). Therefore, appropriate medical assessment

TABLE 5.2. Serum electrolyte levels in purging disorders

	Serum level				
Purge type	Sodium	Potassium	Chloride	Bicarbonate	pH
Vomiting	High, low, or normal	Low	Low	High	High
Laxatives	High, low, or normal	Low	High or low	High or low	Low or high
Diuretics	Low or normal	Low	Low	High	High

TABLE 5.3. Urinary electrolyte levels in purging disorders

	Urine level		
Purge type	Sodium	Potassium	Chloride
Vomiting	Low	High or low	Low
Laxatives	Low	Low	Normal or low
Diuretics	High	High	High

of a patient with bulimia should always include obtaining a serum electrolyte panel.

Fortunately, the diagnosis of electrolyte abnormalities is usually straightforward. A set of electrolytes with serum magnesium and amylase can be tested in blood drawn as part of the clinical evaluation, and this analysis is readily available in all laboratories. It is always wise to conduct an EKG to look for changes secondary to significant hypokalemia, hypomagnesemia, and hypocalcemia. As mentioned above, the common EKG changes seen with a decreased serum potassium are T-wave flattening, U waves, and ST-segment depression. Hypomagnesemia, hypokalemia, and hypocalcemia can cause a prolonged QT interval and nonspecific T-wave changes. The prolonged QT interval can precipitate serious ventricular arrhythmias, especially as the QTc increases above 480 milliseconds. If there is a need to differentiate between the several types of purging behaviors, measurement of serum and urine electrolytes can be helpful. In general, once again, metabolic alkalosis (elevated bicarbonate level) indicates vomiting or diuretic abuse, and a metabolic acidosis (low bicarbonate level) indicates laxative abuse, although

chronic laxative abuse leads to metabolic alkalosis. It may be necessary in some cases, when there is a suspicion of laxative abuse despite the patient's denial, to check the feces or urine for phenolphthalein, an ingredient in some laxatives. Similarly, when there is suspicion of diuretic abuse, a laboratory can test the urine for the presence of common diuretics.

Treatment

Treatment of the metabolic abnormalities observed in eating disorders requires an understanding of the pathophysiology of these problems, as outlined above. There are two important concepts to keep in mind. First, because potassium is primarily an intracellular cation, the serum measurement can be misleading in estimating the true degree of *total* body potassium depletion. As a general rule, for each drop in serum potassium of 0.3 mmol/L, there is a deficit in total body stores of more than 100 mEq (Asmar, Mohandas, and Wingo, 2012). Rough estimates of total body potassium deficits are shown in table 5.4. A common mistake is to underestimate the amount of potassium necessary to correct hypokalemia, and thus replacement is prolonged and delayed.

The main treatment to correct hypokalemia is replacement of potassium in either of two ways: the intravenous (IV) route or the oral route. There are no clearly defined potassium levels for which IV replacement is indicated, but if the patient is hemodynamically unstable due to hypokalemia or if the serum level is below 2.5 mmol/L, it is generally recommended to replace potassium intravenously. Intravenous replacement requires cardiac monitoring, and the infusion should run no faster than 20 mmol per hour. Cardiac monitoring is required because IV potassium

TABLE 5.4. Total body potassium deficits

Serum potassium concentration (mmol/L)	Potassium deficit (mmol/L)
3–3.5	100–150
2–3	200–300
<1.5	400–600

may lead to overcorrection of the deficit and deadly arrhythmias, and it thus warrants hospital-level care. The most common side effects of IV replacement are overcorrection of hypokalemia and burning at the site of infusion. When potassium is replaced intravenously, repeat serum potassium level checks should follow each 30 mEq infusion.

Three salts are available for oral repletion of potassium: potassium chloride, potassium phosphate, and potassium bicarbonate. Potassium phosphate has minimal amounts of potassium and is mainly used to replenish low phosphorus levels. Potassium bicarbonate is rarely used and usually only in settings of concomitant hypokalemia and metabolic acidosis. Of the three salts, potassium chloride is the most common formulation used to treat hypokalemia; it is available as a tablet and a liquid. The liquid form is less expensive but has an unpleasant taste and may lead to further nausea and emesis. Two slow-release tablets are available, of which one has a wax matrix and the other is a microencapsulated tablet. Each is fairly well tolerated, although the risk of ulceration and bleeding of the gastrointestinal tract is slightly higher with the wax matrix formulation (Gennari, 1998). In general, the rule of thumb is that for each 10 mEq of oral potassium replacement, the serum potassium level should increase by 0.1 mmol/L. Note that the total body store deficit mentioned above (for each drop in serum potassium of 0.3 mmol/L, the deficit in total body store is >100 mEq) should not be used as a guide by which to replace potassium. It does not take account of the complicated intracellular shifts of potassium and would lead to overcorrection of this delicate electrolyte. Rather, replacement should be based on the actual serum level, with the understanding that 10 mEq of any formulation will raise the serum potassium level by about 0.1 mmol/L. Common oral potassium formulations are listed in table 5.5.

The second concept to keep in mind when treating hypokalemia due to purging behaviors is that efforts to replenish potassium are less effective if the coexisting metabolic alkalosis induced by dehydration is not also addressed and treated. This is because dehydration resulting from any purging behavior stimulates aldosterone production, which not only causes a metabolic alkalosis but also leads to ongoing renal potassium excretion. Cessation of purging behavior is the first step in

TABLE 5.5. Oral potassium preparations

Preparation	mEq per tablet (or other preparation)
K-Dur (KCl)	10 or 20
K-Tab (KCl)	10
Micro-K Extencaps (KCl)	8 or 10
Slow-K (KCl)	8
K-Lor powder packets (KCl)	20
Polycitra-K oral solution (K citrate)	10/tsp (5 mL)
Rum-K syrup (KCl)	20/2 tsp (10 mL)

treatment, followed by rehydration with IV or sodium-containing oral solutions to reduce aldosterone secretion and aid potassium repletion. The metabolic alkalosis in patients who abuse diuretics or engage in self-induced vomiting, with resultant volume contraction and elevated bicarbonate levels, is thus termed *chloride responsive*. This means that normal saline, which contains water and sodium chloride, is the appropriate corrective treatment. Intravenous infusions of sodium chloride restore intravascular volume and lead to a reduction in excessive aldosterone secretion. The efficacy of potassium repletion will be abrogated if the dehydration state is not also corrected. Treatment of severe metabolic alkalosis (>33–35 mmol/L) with sodium-containing fluids usually needs to be done intravenously at a *slow* rate of 50–75 mL per hour for 1–2 L of fluid. Milder cases can be successfully treated with aggressive oral hydration using fluids other than plain water, such as bouillon.

A word of caution here: Excessive amounts of IV saline (>2 L per day), given at a rapid rate of infusion, can lead to marked edema formation and volume overload. It takes about 1 to 2 weeks following cessation of purging behaviors and restoration of normal plasma volume for the body to naturally down-regulate the excessive secretion of aldosterone (the hormone driving the reabsorption of sodium and loss of potassium) (Schrier, Masoumi, and Elhassan, 2010). Therefore, with high ambient serum aldosterone levels, the IV saline is avidly reabsorbed by the kidney, a process that can also contribute to the body's rapid retention of

salt and water with resultant edema. In fact, the edema can be severe during the early period after purging behaviors are discontinued, even without the use of IV fluids, because of persistently increased serum aldosterone levels. As mentioned above, this constellation of symptoms is termed pseudo-Bartter's syndrome. Key points and treatment of pseudo-Bartter's syndrome are highlighted in table 5.6.

Some individuals with purging disorders who need to frequent emergency rooms for electrolyte replacement therapy may be reticent to seek additional help if their previous visits were punctuated by development of significant edema due to rapid infusions of IV saline. The formation of edema is distressing, especially for a population of patients that suffer from body dysmorphia. Emergency department staff, who are used to rapid infusion of IV saline to treat adults with other states of dehydration such as gastroenteritis, must be warned about the dangers of this practice when applied to patients with purging behaviors (Trent et al., 2013). In fact, these misguided treatments often prevent patients with purging disorders from seeking timely treatment despite feeling ill from their dehydration and electrolyte derangements. This delay may contribute to the increased standardized mortality ratio for bulimia nervosa—nearly twice that of age-matched controls (Smink, van Hoeken, and Hoek, 2012). Generally, the edema can be minimized, if not prevented, during this period with leg elevation for 10–15 minutes a few times a day and, when IV fluids are necessary, a slowed infusion of saline; a detailed explanation to the patient of the distressful but transient nature of the resultant edema is helpful (Mehler and Linas, 2002).

TABLE 5.6. Pseudo-Bartter's syndrome

▪ Syndrome is characterized by hypokalemia and metabolic alkalosis.
▪ Volume depletion plays a central role.
▪ Efficacy of potassium repletion is abrogated unless volume is restored.
▪ Restore volume with IV normal saline slowly (50–70 mL/hour).
▪ Edema may be alarming to the patient.
▪ Risk dissipates by 2–3 weeks after cessation of purging.

To reiterate, given the sodium-avid state that results from purging behaviors, when IV fluids are necessary, the rate should not exceed 50–75 mL per hour until the serum bicarbonate level is less than 30 mmol/L, at which point the IV infusion can be discontinued.

In addition to cessation of purging behaviors, restoration of volume status, and correction of electrolytes, there is one more important treatment modality that can minimize or prevent the formation of edema in this patient population. Paradoxically, it is through the use of a diuretic, a potassium-sparing one: spironolactone. Spironolactone is an aldosterone antagonist that competitively inhibits the action of aldosterone at the receptor level. It thus can block the action of aldosterone in the initial phases of treatment while the body naturally ceases its overproduction (Lainscak et al., 2015). The action of spironolactone is threefold: it not only blocks the action of aldosterone, but also is potassium sparing, thus aiding in treatment of hypokalemia, and is a mild diuretic, thus aiding with edema formation and treatment (Mascolo, McBride, and Mehler, 2015). A starting dose of 25 mg daily, for 1–2 weeks, can be initiated at the onset of cessation of purging behaviors. This can be increased as needed up to 400–600 mg as a single daily dose if edema worsens or there is rapid weight gain. Note that patients who have any degree of renal compromise should have their electrolytes closely monitored if spironolactone is used, given its potassium-sparing effect and potential for development of hyperkalemia.

A further word of caution when replenishing a dehydrated patient: If the patient also has significant hyponatremia (serum sodium <120 mmol/L), the saline infusion should be slow so that during the initial 24 hours, the serum sodium is corrected to only approximately 125 mmol/L. More aggressive correction of the serum sodium level may result in a devastating neurologic complication termed *osmotic demyelination syndrome*. A targeted rate of correction that does not exceed 4–6 mmol/L in 24 hours is recommended (Sterns, 2015). In addition, although a healthy person with normal kidney function would need to drink a large amount of plain water (25–30 L) to adversely dilute serum sodium and experience marked, symptomatic hyponatremia as a result, patients with anorexia nervosa are different. Their ability to tolerate water is severely limited by their extremely limited solute intake, as seen in older

men and women who live on tea and toast and are thus unable to excrete a diluted urine. For patients with anorexia nervosa, amounts as little as 5 L per day can cause hyponatremia (Caregaro et al., 2005).

In addition to the central role of volume depletion in the treatment of hypokalemia associated with severe self-induced vomiting and diuretic abuse, undetected magnesium deficiency can also cause refractory potassium repletion. Magnesium deficiency should especially be considered in those individuals whose primary means of purging is the use of potent diuretics such as the loop, thiazide, or potassium-sparing diuretics. Most diuretics cause some loss of magnesium in the urine (Tamargo, Segura, and Ruilope, 2014). Since magnesium and potassium hold the same charge, they, in effect, competitively inhibit each other for absorption. Thus, ongoing magnesium deficiency, if not treated, can impair efforts at potassium repletion. The most effective way to replenish magnesium is with magnesium sulfate through the IV route. Oral magnesium oxide is not well absorbed and can lead to diarrhea, which perpetuates the dehydrated state.

Summary

Metabolic abnormalities are common in patients with eating disorders, especially those who engage in purging behaviors, including self-induced vomiting, diuretic abuse, and laxative abuse. Metabolic alkalosis and hypokalemia are the most common abnormalities encountered clinically. The treatment of these metabolic derangements requires cessation of purging behaviors, repletion of electrolytes, and treatment of the volume contraction that causes hyperaldosteronism. A clinician who has the correct skills and a familiarity with treating these potentially dangerous electrolyte disorders in this patient population is essential for all but the mildest abnormalities.

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6

Gastrointestinal Complications

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COMMON QUESTIONS

What clues from a gastrointestinal (GI) history and physical examination are helpful during both early (occult) and later stages of eating disorders?

What GI symptoms occur with early weight loss?

What common abnormalities found on laboratory testing may signal the presence of an eating disorder?

What symptoms and signs develop as weight loss becomes more severe and can frustrate weight restoration?

What treatment strategies can be used for common GI symptoms (bloating, early satiety, fullness) that may interfere with nutritional rehabilitation?

How do GI symptoms improve with time and weight gain? How can this be used to reassure patients?

What can you tell patients about the long-term effects of anorexia nervosa on the GI tract?

When are GI studies, such as upper or lower endoscopy, barium studies, or nuclear medicine testing, indicated?

What role is there for medications to help treat various GI complications of anorexia nervosa and bulimia nervosa?

Case Study 1

W.H., a 19-year-old female, presented with pure restricting anorexia nervosa, having had a 20-pound weight loss (height 5 feet 6 inches; original weight 115 pounds) and now 70% of ideal body weight (IBW). She had no prior history of intestinal disease. At the nadir of her weight loss, she complained of epigastric bloating and constipation. Physical examination revealed a normal abdomen, no evidence of distention, and hemoccult-negative stool.

Inpatient multidisciplinary treatment enabled W.H. to gain 25 pounds over a 10-week period. She was reassured that the bloating and constipation would improve with time and weight restoration. Fiber, which can often increase bloating, was avoided in her diet so her complaint of bloating would not worsen. Her bloating and constipation spontaneously improved without the use of laxatives or the need for any GI studies.

Case Study 2

A.D., a 25-year-old female with bulimia who was undergoing outpatient psychotherapy, continued intermittent self-induced emesis twice a day. She had no weight loss and did not engage in laxative use.

A.D. complained of sore throat, hoarseness, dyspepsia, and intermittent odynophagia. She was empirically treated with omeprazole 20 mg once a day. The hoarseness, dyspeptic symptoms, and sore throat resolved after 4 days, and the odynophagia after 2 weeks. One month later, she denied any swallowing problems. With intensive psychotherapy, her purging ceased, as did all the aforementioned symptoms. She was concerned about the previous difficulty with swallowing and wondered if additional testing was necessary. Her omeprazole was discontinued, and her symptoms did not recur.

Background

People who struggle with disorders commonly complain of gastrointestinal symptoms (Hetterich et al., 2019). Such symptoms are often a consequence of the eating disorder, although GI symptoms can also

predispose to disordered eating (Wiklund et al., 2019). Early in the course of the illness, GI complaints may so preoccupy the patient and the physician that they interfere with and deflect attempts at psychological treatment. There may even be some question as to whether a primary GI disease exists in addition to the eating disorder. Moreover, an eating disorder may exacerbate a preexisting intestinal disease, especially irritable bowel syndrome (IBS), one of the most encountered GI disorders in the general population that may overlap with eating disorders (Perkins et al., 2005).

The primary care physician and the primary therapist are often confronted with a wide spectrum of GI complaints in patients with eating disorders. To care for these patients, clinicians should be somewhat familiar with several germane GI topics:

1. The prevalence of GI symptoms in eating disorders
2. The time course of these GI symptoms' evolution during illness and recovery
3. The effect of GI symptoms on treatment
4. Possible long-term GI sequelae for individuals who recover from eating disorders
5. Therapeutic modalities available to treat these GI issues

Both anorexia nervosa (AN) and bulimia nervosa (BN) are associated with specific GI symptoms that are uniquely attributable to the food restriction and weight loss or the purging behaviors that characterize these two eating disorders. Fear of GI symptoms is also a major contributor to disordered eating in those with avoidant/restrictive food intake disorder (ARFID) (Murray et al., 2020). An initial complete medical history and physical examination are part of the evaluation for patients with eating disorders. It is important to elicit a thorough history of relevant GI disease and current symptoms. A history of gastroesophageal reflux (acid reflux), for example, may be exacerbated by even a small amount of weight gain (Jacobson et al., 2006), or the self-induced vomiting of bulimia may require aggressive treatment. Similarly, patients with preexisting IBS may develop worsening symptoms of pain or altered bowel patterns during the refeeding process. Patients with irritable

bowel syndrome, already predisposed to focus on intestinal complaints, may develop slowed GI motility due to ongoing weight loss. This can lead to more severe bloating or constipation, which further frustrates weight restoration.

Anorexia Nervosa

Common GI symptoms associated with the weight loss in anorexia nervosa that generally improve with weight gain are listed in table 6.1 (Norris et al., 2016). Patients with AN who present to gastroenterologists are often heavy consumers of health information because they are hoping to diagnose organic causes for their litany of somatic complaints (Emmanuel et al., 2004). It is important to neither summarily dismiss nor repeatedly evaluate a patient's GI complaints, as both approaches, if injudiciously taken, can be laden with deleterious risks for the recovery of these patients. There is a complex interplay between the GI system and eating disorders, given the inextricable connection between food and the body system that digests the food. Indeed, an increasing amount of research is beginning to focus on the role of this "gut-brain axis" as it relates to the development and maintenance of eating disorders (Seitz, Trinh, and Herpertz-Dahlmann, 2019).

Gastroparesis

With pure food restriction, once a weight loss of approximately 10%–20% occurs, there is potential for the development of gastroparesis (Weterle-Smolińska et al., 2015). *Gastroparesis* refers to delayed emptying of the stomach due to impairment of antral stomach contractions that impedes

TABLE 6.1. Common gastrointestinal symptoms with anorexia nervosa

▪ Bloating	▪ Nausea	▪ Abdominal pain
▪ Fullness	▪ Constipation	▪ Distention
▪ Early satiety		

grinding of the food. The threshold for weight loss needed to cause gastroparesis is unknown. A shorter duration of AN seems to portend less likelihood of developing gastroparesis. Bloating and early satiety are the main symptoms and may be severe. The bloating can be exacerbated by a high-fiber diet, such as the vegetarian diet these patients often adopt. Once gastroparesis develops, early satiety, nausea, bloating, and even acid reflux and vomiting that is no longer self-induced may occur in patients with anorexia nervosa (Perez et al., 2013). This may hamper attempts at weight restoration. Although the bloating and early satiety generally improve with weight restoration, improvement often takes many weeks to months, and its natural history is not well defined in AN. Therefore, it is crucial to offer reassurance to these patients that their symptoms are indeed inherent to their weight loss and the refeeding process, and to instruct them to avoid legume-type foods and excessive fiber, which promote gas and distention. This will enable them to understand that their symptoms will pass with time and that they are not "causing" the pain by eating. Likewise, patients should be informed that refusal to eat and regain weight will only delay the improvement and ultimate resolution of their symptoms. Working with a registered dietician experienced with patients who have anorexia nervosa is important to effectively deal with these refeeding GI issues (Trees et al., 2016).

Useful approaches to the problem of gastroparesis-induced bloating in patients with anorexia include the following:

1. Use of liquid food supplements as a portion of the daily calories (one can of supplement three times per day for a total of 750–1,000 calories) for the first week or two of refeeding, since gastric emptying of liquids is generally normal even in severe anorexia nervosa.
2. Ingestion of liquid components rather than solids earlier in the meal, which generally results in less bloating, so that the patient can continue eating.
3. Division of the daily caloric intake into two or three snacks and three smaller meals per day, so that meal-induced bloating will

not result in termination of a meal and loss of a major portion of the daily caloric intake.
4. Use of particle-sized foods (Olausson et al., 2014).

Symptoms of gastroparesis induced by weight loss generally improve with partial weight restoration. Thus, the severe bloating initially complained of, which often prevents patients from eating adequate calories with their meal, progressively improves with weight gain for most individuals. For patients with residual psychopathologic distress, however, symptoms of bloating may remain despite refeeding (Benini et al., 2004). Thus, it may rarely be necessary to obtain a nuclear medicine gastric-emptying study to formally investigate prolonged gastroparesis-type symptomatology after refeeding and weight restoration.

Metoclopramide is a medication that stimulates stomach contraction and hastens emptying of the stomach. Doses of 2.5–5 mg, 30 minutes before meals and perhaps also at bedtime, are clinically useful in treating bloating and early satiety secondary to delayed gastric emptying in anorexia nervosa (Camilleri et al., 2013). Metoclopramide is also a well-established antiemetic medication. Caution must be exercised when using the drug, however, because it can exacerbate neck cervical muscle spasm (torticollis) and, rarely, can cause tardive dyskinesia, especially at higher doses and in older patients. Macrolide antibiotics such as erythromycin or azithromycin are occasionally added to or used in place of metoclopramide. Dual use may be of added benefit for refractory symptoms (Moshiree et al., 2010), although close cardiac monitoring is required as both macrolide antibiotics and metoclopramide can cause QT prolongation. Macrolides can also cause tinnitus.

Gastroesophageal Reflux

Gastroesophageal reflux (heartburn, acid reflux) often begins in patients with anorexia nervosa, perhaps secondary to gastroparesis. It also generally improves with time as weight is restored. In contrast to the gastric-emptying abnormalities noted above, however, esophageal emptying has been found to be normal in anorexia nervosa. Therefore, esophageal

complaints in patients with restricting AN are often of a functional nature (Boyd, Abraham, and Kellow, 2010). Gastroesophageal reflux usually requires histamine-2 antagonists (such as famotidine, 20 mg twice a day) or a proton-pump inhibitor (omeprazole, 20–40 mg per day) (Mittal and Vaezi, 2020). The maintenance dosage usually used for peptic ulcer disease (only a nighttime dose) is often inadequate to control symptoms secondary to reflux. Any patient with persistent vomiting, severe reflux symptoms, or dysphagia that does not improve after 3 weeks of treatment should probably undergo an upper endoscopy procedure to evaluate for peptic ulcer disease or for ongoing severe esophageal inflammation. Inflammation from gastric acid exposure can progress over years to Barrett's esophagus, a precancerous transformation of the lining mucosa of the esophagus (Pacciardi, Cargioli, and Mauri, 2015). Although Barrett's esophagus can progress to esophageal adenocarcinoma, research instead suggests an increased risk for esophageal squamous cell carcinoma, more likely related to alcohol and smoking, in those with anorexia nervosa (Brewster, Nowell, and Clark, 2015).

One additional concern, especially in patients with more severe forms of AN, is the possibility of *dysphagia* (difficulty swallowing), frustrating patients' ability to complete their meal plan. The pharyngeal muscles can atrophy with weight loss and result in the development of dysphagia. Consultation with a speech therapist may be of help (Holmes et al., 2012).

Constipation

Constipation invariably accompanies the weight loss of anorexia nervosa, even in patients who have never abused stimulant laxatives. Persons with weight loss are expected to have fewer bowel movements due to an overall slowed GI transit time. They may incorrectly respond to their perceived problem with constipation by starting to use bulking, fiber-containing laxatives or dangerous stimulant laxatives. This may worsen the constipation by inducing bloating and distention, through diarrheal losses of potassium, or by making the bowel dependent on stimulant laxatives. Because individuals often take laxatives in the mistaken belief that they will produce substantial weight loss, it is useful to educate patients

that laxatives act in the colon, after caloric absorption has already occurred, and are thus ineffective as a means of weight loss (Bo-Linn et al., 1983). Others may take laxatives because they mistakenly believe that their abdominal pain is due to decreased bowel movements. Patients may need frequent reassurance that there is nothing fundamentally wrong with their bowels other than a need for weight restoration. They must be encouraged to focus on the expected return, over weeks, to their prior bowel pattern as weight is restored. Generally, with weight restoration and refeeding, normal bowel transit time resumes in less than one month; however, symptoms may not fully resolve with normalization of slow colonic transit as there is seemingly a lack of correlation between bowel transit time and reported symptoms (Chun et al., 1997).

Anorectal dysfunction and anal muscle weakness documented by manometry are sometimes seen in patients with weight loss and can contribute to the bowel complaints. Rectal prolapse has also been described in anorexia nervosa. These conditions also resolve with time and nutritional rehabilitation (Mitchell and Norris, 2013).

Treatment options for constipation, in addition to education and progressive weight restoration, include (1) adequate water intake; (2) fiber in very low doses (10 g per day), avoiding the high doses that can cause bloating; (3) polyethylene glycol powder, at a dose of 1–2 capfuls one to three times per day; or (4) lactulose, a nonabsorbable synthetic disaccharide (similar to polyethylene glycol products in efficacy and mode of action), 30–60 mL once or twice per day. If lactulose is used, it is useful to tell patients that although this medication tastes sweet, it is devoid of calories because it is not absorbed. Lactulose reaches the colon in an unaltered form and, because it is highly osmotic, draws water into the bowel, resulting in a bowel movement; however, it can also result in increased symptoms of bloating due to fermentation of the medication in the colon. Polyethylene glycol products work in a similar manner and have a long record of efficacy for gut cleansing in preparation for colonoscopy and surgery (Ramkumar and Rao, 2005). (See table 6.2 for a classification of laxative therapies.)

In general, it is best to avoid the use of any stimulant laxatives containing senna, cascara, or bisacodyl, given the potential for long-term

TABLE 6.2. Classification of laxative therapies

Class	Mechanism of action	Site of action	Example
Osmotic	Attracts/retains water in intestinal lumen, increasing intraluminal pressure	Small intestine and colon	Magnesium hydroxide (Milk of Magnesia) Lactulose Polyethylene glycol
Irritant or peristaltic (use with caution)	Direct action to stimulate myenteric plexus; also alters water and electrolyte secretion	Colon	Senna Bisacodyl Cascara Phenolphthalein
Bulk or hydrophilic	Holds water in stool and causes mechanical distention	Small and large intestines	Methylcellulose Psyllium Dietary bran
Stool softener	Softens stool by facilitating a mixture of fat and water (largely ineffective)	Colon and rectum	Docusate

abuse and dependence after weight restoration. Long-term use of stimulant-type laxatives may cause direct damage to colonic nerve cells and result in refractory constipation. This syndrome is termed the *cathartic colon syndrome*. Rarely, early in the refeeding process, a glycerin suppository or a stimulant-type laxative may be necessary to "jump-start" normal peristaltic motion in the bowel, but it should always be used in a limited, judicious manner. Newer medications such as lubiprostone and linaclotide, which are approved for constipation-type IBS, have not been specifically studied in anorexia nervosa but, rarely, may be of use for a brief period for early bouts of constipation. Furthermore, linaclotide reduces visceral hypersensitivity that is present in IBS and other functional bowel diseases and may have a unique role in this population (Johnston, Shiff, and Quigley, 2013).

Abdominal Pain

Abdominal pain is a frequent complaint in anorexia nervosa. It is generally diffuse, unaccompanied by tenderness, and more consistent with irritable bowel syndrome, a functional GI disease. Although phys-

iologic changes to the GI tract are inherent to malnutrition, these physiologic changes do not seem to correlate with self-reported symptoms (Benini et al., 2004; Chun et al., 1997). Indeed, functional GI disease is highly comorbid with eating disorders, and studies report a prevalence of 56%–97% in anorexia nervosa (Boyd, Abraham, and Kellow, 2010; Perez et al., 2013). Functional GI diseases are defined as a group of disorders of gut-brain interaction related to any combination of motility disturbance, visceral hypersensitivity, altered mucosal and immune functioning, altered gut microbiota, and altered central nervous system processing (Drossman, 2016). Although these disorders generally improve slowly over time with psychotherapy and weight restoration, it is not uncommon for different functional GI diseases to develop during treatment, with increased likelihood predicted by psychological distress (Boyd, Abraham, and Kellow, 2010; Kessler et al., 2020; Salvioli et al., 2013). Patients' complaints should not be immediately attributed to a functional etiology, however, discounting the need for further workup, as persistent pain may need further evaluation (Gibson et al., 2021).

Abdominal pain accompanied by diarrhea should be evaluated to rule out fecal impaction, infectious causes, celiac disease, inflammatory bowel disease, and other pathology, especially if accompanied by fever, extreme pain, or an elevated white blood cell count. The weight loss of anorexia nervosa itself, however, can cause villous atrophy in the small intestine and reduce the intestinal surface area needed for proper digestion. This, in turn, can cause diarrhea soon after eating. It is diagnosed by the finding of a low serum diamine oxidase level (Takimoto et al., 2014). Gallstones, though rare, should also be considered in the differential diagnosis of patients with anorexia nervosa who have vomiting and/or right upper quadrant pain, given the known increased incidence of gallstones in patients with weight loss. If no right upper quadrant pain accompanies the vomiting, however, gallstones are less likely. A right upper quadrant abdominal ultrasound is a simple way to exclude the presence of gallstones.

Gastric dilatation must also be considered in a patient presenting with left upper quadrant pain, significant vomiting, or early satiety in the setting of abdominal distention (figure 6.1). It most frequently develops during the early phase of refeeding and in the setting of superior

Figure 6.1. Abdominal X-ray showing gastric dilatation

mesenteric artery syndrome or following a large binge. Given the risk for gastric rupture, it should be screened for with an abdominal X-ray (Mascolo et al., 2015), with treatment including nasogastric suction of stomach contents, short-term discontinuation of oral nutrition, and potentially a surgical consult.

Celiac disease, an inflammatory condition of the small intestine resulting from gluten ingestion, frequently manifests with weight loss, diarrhea, flatulence, and malabsorption. Studies are suggestive of a potential

bidirectional relationship between anorexia nervosa and celiac disease (Wotton, James, and Goldacre, 2016). Work-up for this condition can include serum antibody testing with antitissue transglutaminase, antiendomysial antibodies, or antigliadin antibodies; diagnosis can also be made with a small bowel biopsy consistent with celiac disease. Treatment consists of a gluten-free diet, with resolution of the above symptoms; however, a gluten-free diet can also impact body image and further contribute to disordered eating (Satherley, Higgs, and Howard, 2017). Therefore, it is best to acquire objective evidence of this diagnosis, as opposed to allowing patients to otherwise follow a gluten-free diet, given the potential for ongoing exacerbation of the eating disorder with such a diet.

Inflammatory bowel disease, including both Crohn's disease and ulcerative colitis, can also often present with weight loss, abdominal pain, and diarrhea, which is often bloody in cases of ulcerative colitis. Crohn's disease and ulcerative colitis are diagnosed via colonic biopsy, with treatment consisting of pharmacologic agents to inhibit the immune response or, in some cases, the need for surgical removal of the inflamed bowel. Similar to celiac disease, there appears to perhaps be a bidirectional relationship between inflammatory bowel disease and anorexia nervosa (Wotton, James, and Goldacre, 2016). The overall incidence of inflammatory bowel disease is quite low, however, and work-up for this condition is generally not warranted when patients present with typical GI symptoms.

Laboratory studies are occasionally worthwhile for patients with eating disorders who are experiencing abdominal pain. Iron-deficiency anemia may indicate significant GI disease or inability to absorb oral iron. Elevated amylase levels are almost always due to a salivary source resulting from excessive vomiting and are not indicative of pancreatitis. This can be confirmed by a lipase measurement, which is elevated in pancreatitis but normal when amylase elevation is due to a salivary source, as seen with purging. In some rare cases, however, refeeding itself, if too aggressive, can induce refeeding pancreatitis early in the process; however, acute pancreatitis is unlikely to develop directly from malnutrition, and other secondary causes of pancreatitis, such as gallstones, should be investigated. Patients with gallstone pancreatitis typically have extremely high amylase measurements (>1,000 mg/dL) along

with elevated lipases. They require a cholecystectomy because of their risk of recurrent gallstone pancreatitis. Asymptomatic gallstones, probably due to weight loss, do not generally warrant urgent surgery.

A supine and upright simple abdominal X-ray may be useful to exclude bowel distention when symptoms of constipation persist after an adequate trial of medications aimed at alleviating constipation (figure 6.2). The absence of excessive stool on these radiographic studies provides

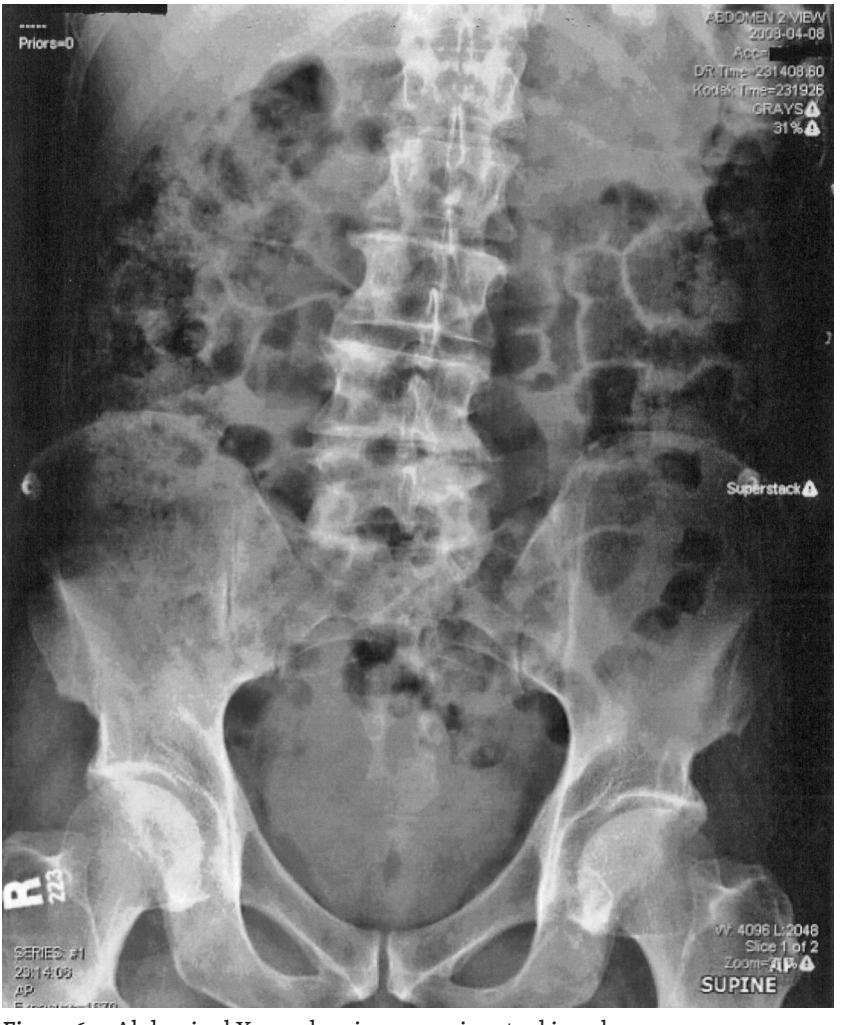

Figure 6.2. Abdominal X-ray showing excessive stool in colon

proof that bowel function is normal and no longer deserves ongoing concern. This is especially helpful because of the interplay of functional GI disorders in anorexia nervosa.

Elevated Liver Transaminase Levels

Routine blood tests for liver enzymes (AST and ALT) often show abnormalities in patients with anorexia nervosa. Bilirubin and alkaline phosphatase are typically normal in patients with eating disorders, though abnormalities may be seen in approximately 15% of AN patients (Rosen et al., 2016). Generally, ALT is elevated more than AST, a pattern typically seen in liver diseases from causes other than alcoholic liver disease, which usually demonstrates higher AST. Mild increases in transaminases are observed in approximately 75% of patients with AN, while moderate increases are less common (Kheloufi et al., 2014).

Liver test abnormalities are a helpful indicator for clinicians and patients of greater illness severity. Serum transaminases are increasingly abnormal at lower BMIs. In fact, patients at 50% IBW have a twelvefold higher risk of severely elevated liver enzymes compared to patients at 70% IBW (Rosen et al., 2016). Further, in rare cases of severe anorexia nervosa, transaminases are markedly elevated prior to refeeding, which may be a sign of serious multiorgan failure (De Caprio et al., 2006). Elevation in AST has also been found to be a risk factor for refeeding hypophosphatemia (Brown et al., 2015). Worsening liver enzyme abnormalities lead to increased risk of hypoglycemia, which has poorer prognosis in AN (Rosen et al., 2016).

Abnormalities in liver function tests may be observed both prior to and during the refeeding process. Prior to refeeding, weight loss and malnutrition lead to elevation of transaminases (AST/ALT) due to autophagy of hepatic cells. Autophagy (meaning "self-eating") is a process that occurs in cells under stressful environments, causing cellular degradation by enzymes to increase cell's energy production. In cases of energy deprivation (as in starvation and malnutrition seen in AN), autophagy mobilizes energy substrates such as glycogen and lipids to generate ATP, the main source of cellular energy. This process allows cell

function to continue despite the stressed state. If this process becomes overwhelmed, however, it can lead to cell death (Fernandez et al., 2020). Starvation is one of the prominent causes of cellular autophagy, and in severe AN patients with highly elevated serum transaminases, liver biopsies demonstrate evidence of a particular subtype of this mechanism of cell death called autolysis (Kheloufi et al., 2014; Fernandez et al., 2020). In AN, more severe liver enzyme abnormalities may indicate the progression of autophagy. Initially it has a protective energy-conserving benefit as a coping mechanism for cellular stress, but with greater weight loss, it progresses to cell death seen at lower BMIs (Kheloufi et al., 2014). Patients with liver enzyme abnormalities can be counseled that their values will likely improve with nutrition. Even in patients with severe liver disease, providing nutritional support and addressing abnormalities in electrolytes and blood glucose leads to rapid improvement of liver function (Kheloufi et al., 2014).

Mild transaminase elevations can also occur during refeeding. In one study of AN patients with normal ALT on admission, 46% developed mild to moderate elevation of ALT, which had resolved in 81% at 1 month. The degree of elevation was milder than patients with elevation at admission (Rosen et al., 2016). During the first few days to a week, this may be still related to starvation hepatitis. These elevations are usually not cause for concern and should not prompt decreased rate of refeeding. Abnormalities can persist or worsen from the refeeding process itself, however, if dextrose is excessive or fat calories are causing steatosis, a fatty liver. These elevations usually resolve if the composition of the diet is changed to fewer glucose-based calories, or if the daily caloric intake is decreased by 300–500 kcal. A higher level of caloric intake can then be reintroduced later once the liver tests have normalized.

As liver function tests generally resolve during the refeeding process, imaging or invasive procedures such as liver biopsy are not usually indicated. In rare cases, however, a simple right upper quadrant ultrasound may be obtained if the transaminases are rapidly increasing or are approaching a level of 1,000 U/L. In the case of autophagy, ultrasound may demonstrate a small shrunken liver, whereas with fatty liver, a large fat liver may be seen.

Superior Mesenteric Artery Syndrome

One other rare syndrome to be aware of in people with anorexia nervosa is superior mesenteric artery (SMA) syndrome. It results from compression of the third portion of the duodenum between the aorta and the vertebral column posteriorly and the SMA anteriorly. Anything that narrows the normal angle between the aorta and the SMA can cause entrapment of the duodenum as it passes between these vessels. The SMA is normally kept in place by a fat pad; reduction in the fat pad due to weight loss narrows the angle. Patients with anorexia nervosa who have SMA syndrome typically complain of intermittent vomiting and upper quadrant abdominal pain soon after food consumption. With weight gain of as little as 5–10 pounds and regrowth of the fat pad, tolerance of normal feeding is restored as the duodenal compression abates. The diagnosis is made by upper GI series tests or by an abdominal CT scan, with the radiologist made aware of the diagnosis being considered. Once diagnosed, treatment involves either the use of a soft liquid oral diet or the placement of a nasogastric or naso-jejunal tube until symptoms abate. Surgery has no role for SMA syndrome due to anorexia nervosa (Watters et al., 2020).

Bulimia Nervosa

Bulimia nervosa leads to a different set of GI symptoms (table 6.3). Severe GI reflux symptoms—heartburn, spontaneous vomiting, acid regurgitation, chest pain, dysphagia, and nocturnal choking when lying supine—and their complications often occur in bulimia, although this relationship is far from conclusive (Denholm and Jankowski, 2011). These symptoms, when present, are due to self-induced vomiting, which weakens the lower esophageal sphincter, a one-way valve that normally prevents stomach acid from moving up into the esophagus. These reflux symptoms may be among the few clues to ongoing illness. Spontaneous vomiting is the effortless return of stomach contents into the mouth without retching. Food-restricting patients who do not have bulimia may develop reflux presumptively due to weight loss–induced gastroparesis. Reflux in these

TABLE 6.3. Common gastrointestinal symptoms associated with purging behaviors

Vomiting related
Heartburn
Odynophagia
Dysphagia
Hoarseness
Sore throat
Tooth enamel erosion and pain
Hematemesis (vomiting bright red blood)
Laxative related
Diarrhea
Abdominal cramping
Constipation
Hematochezia (passing bright red blood per rectum)

cases generally involves only the distal esophagus and thus is milder. By contrast, in patients who induce vomiting, the acid contents travel up through the entire esophagus and mouth, and these patients are more likely to complain of more severe dyspeptic symptoms and the symptoms of reflux. Specifically, acid-BN leads to a different set of GI symptoms.

Acid-induced inflammation of the vocal cords may produce hoarseness (which persists for several days after even a brief exposure of the vocal cords to acid), laryngitis, sore throat, or cough. These patients may also have symptoms of *odynophagia* (painful swallowing) or dysphagia due to acid-induced esophageal inflammation or stricture formation within the esophagus. Cessation of self-induced vomiting without concomitant acid-suppressing medication may not alleviate these symptoms. In addition, patients often do not respond to antacids. Rather, they need either higher-dose histamine-2 blockers (famotidine, 20 mg twice a day) or proton-pump inhibitors (omeprazole, 20–40 mg once or twice a day). Prokinetic agents such as metoclopramide are also effective for controlling reflux symptoms in patients with bulimia. In general, trials have established the superiority of proton-pump inhibitors over histamine-2 receptor antagonists for gastroesophageal reflux disease.

Because of incessant exposure of esophageal mucosa to acidic vomitus, patients with BN are at risk for Barrett's esophagus (Navab et al., 1996). Therefore, the presence of refractory heartburn-type symptoms

unresponsive to 4–6 weeks of a high-dose proton-pump inhibitor suggests the need for an upper endoscopy. Barrett's esophagus is discovered in some patients who undergo upper endoscopy for symptoms of gastroesophageal reflux disease, but many patients with Barrett's esophagus do not have typical symptoms of reflux. There is no current test other than an upper endoscopy to establish the diagnosis. Therefore, endoscopy may be reasonable for patients who have bulimia with a long-standing history of self-induced vomiting, but this is less strongly endorsed than previously (Spelcher and Souza, 2014).

Individuals with bulimia nervosa who purge via laxative abuse have a distinct and different set of GI problems. The diarrhea that commonly occurs during periods of excessive laxative use results in blood chemistry abnormalities along with dehydration and dizziness (see chapter 5). The bigger problem, however, is trying to help the patient stop the abuse of laxatives. This is difficult because, if stimulant laxatives have been abused, the bowel may have become dependent on them, and cessation of their use can result in rebound constipation and fluid retention (cathartic colon syndrome). The result is a dilated, atonic colon, manifested by slowed or absent transit through some or all segments of the colon. Basically, the colon becomes an inert tube, incapable of propagating fecal material. This leads to hard, infrequently passed stools and, rarely, a state of refractory constipation. It is difficult to convince patients to stop using laxatives, given their psychological dependence on laxatives and anxiety about losing the feeling of weight control (albeit inaccurate) that laxatives provide (Kovacs and Palmer, 2004).

Clinicians need to help patients understand that the range of normal bowel function, as defined by the Rome IV criteria, includes as a minimum just three bowel movements per week (Drossman, 2016). Also, as noted earlier in the chapter, it is important to point out that laxatives are an ineffective means of achieving weight loss because most caloric absorption occurs in the small intestine, well before the site in the colon where laxatives act (Bo-Linn et al., 1983).

Restoration of normal bowel function may take several weeks. Patients should be encouraged to completely cease their use of stimulant laxatives. There is no scientific basis to support a gradual tapering off,

because ongoing exposure to stimulant laxatives might harm the neurons that oversee bowel peristalsis. Patients must be made to understand that patience is a key virtue when attempting to become independent from stimulant laxatives. If constipation lasts more than 3–4 days, a short course of a mild *nonstimulating* laxative (see table 6.2), together with ample oral fluids, is important. Scheduled polyethylene glycol 2–3 times per day should also be started as soon as the decision is made to cease stimulant laxatives. An occasional glycerin suppository may be needed early in the "detox" process. Docusate is likely of little to no benefit but is also not harmful and may have some utility for its placebo-type effect. Patients should be strongly discouraged from using self-prescribed diuretics for the temporary fluid retention that may occur with cessation of laxative abuse. Although diuretics will lessen edema formation, when they are stopped, the edema will recur to a greater degree than before due to pseudo-Bartter's syndrome (see chapter 5). In fact, moderate fluid intake is a useful adjunct for reestablishing normal bowel function, along with osmotic laxatives such as polyethylene glycol, which are safe and effective for the integrity of bowel function (DiPalma et al., 2007). If edema is marked, involvement of a physician who is informed about this issue and spironolactone should be pursued, as the edema can quickly become severe and is distressing to patients. As noted above for constipation in anorexia nervosa, lubiprostone may be efficacious for treating chronic constipation but is generally not needed. A judicious amount of exercise is also beneficial for normal bowel function (table 6.4), taking into account the propensity for overexercise in this population.

TABLE 6.4. Ancillary treatments for constipation

▪ Ample hydration and judicious exercise
▪ Distraction-free 10–15 minutes on the toilet rather than straining
▪ Elevating the legs on a footstool during defecation
▪ Substitution of less-offending laxatives
▪ Measured *small* amount of fiber rather than an undefined high-fiber diet
▪ *Tailoring medications to the complaint!*

TABLE 6.5. Guidelines for care of patients with eating disorders who have GI complaints

- It is important to reassure the patient that the intestinal symptoms generally improve with weight gain.
- Educating patients about the physiological basis for their constipation may help decrease laxative abuse.
- Lack of improvement in GI symptoms should lead to consideration of an ongoing occult illness and/or underlying primary GI disease.
- Prokinetic agents may be useful for some patients who have bloating and early satiety associated with weight loss. Metoclopramide (2.5–5 mg before meals and at bedtime) is often effective.
- Constipation is best treated with polyethylene glycol (1–3 tablespoons 1–3 ×/day).
- Symptomatic reflux may be treated with histamine-2 blockers (such as oral cimetidine, 400–800 mg, or famotidine, 10–20 mg twice a day). Proton-pump inhibitors (such as omeprazole, 20–40 mg twice a day) are probably more effective.
- Gallstones may develop as a result of weight loss but are often not the cause of the patient's pain. Surgery should not be undertaken routinely.
- Mild elevations in serum liver enzymes (ALT/AST) should resolve with weight gain and adjustments in caloric intake. If they do not, further evaluation is needed to exclude hepatitis B or C, autoimmune hepatitis, hemochromatosis, or Wilson's disease.
- Avoid barium and endoscopic GI studies for most patients unless their symptoms fail to improve with psychiatric recovery, or if iron deficiency anemia is present or to exclude SMA syndrome.

Some useful guidelines for the treatment of patients with AN or BN who have GI complications are presented in table 6.5.

Summary

The gastrointestinal system is adversely affected by both anorexia nervosa and bulimia nervosa. With anorexia nervosa, the complications are mostly manifested during food restriction but become more prominent as the patient begins the process of refeeding. Similarly, the effects of bulimia on the GI system are present during purging behaviors and during the treatment phase when purging tendencies are being controlled. Thus, it is important for clinicians to become familiar with the GI complications of anorexia nervosa and bulimia because effective treatment

and prophylaxis of these GI symptoms will increase the chances of successful treatment of the underlying eating disorder.

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7

Cardiac Abnormalities and Their Management

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COMMON QUESTIONS

Why do patients with anorexia nervosa suffer sudden cardiac death?
What are "normal" vital signs for patients with anorexia nervosa?
Is systolic and diastolic left ventricular function abnormal in anorexia nervosa?
Why do patients with anorexia nervosa have bradycardia?
What degree of bradycardia is cause for concern and needs additional monitoring?
Should a cardiac pacemaker be placed in patients with extreme bradycardia?
When do vital sign abnormalities revert to normal in anorexia nervosa?
What EKG abnormalities are seen with anorexia nervosa?
What is the significance of heart palpitations in patients who have bulimia nervosa?

Case Study

K.L. was a 21-year-old female with a 6-year history of anorexia nervosa. She denied purging behaviors. Her chief complaint was dyspnea of 3 months' duration. She had had occasional chest pain at rest and with exertion. She denied fainting or lightheadedness. There was no family history of premature coronary artery disease. Her

exercise tolerance had diminished over the preceding 2 years, but she remained active and jogged 2 miles per day. She had been in an eating disorder program for 2 weeks. A refeeding program was initiated, and K.L. gained 3 pounds. Her physical examination was notable for a blood pressure of 80/50 mm Hg, a pulse of 48 beats per minute, and a respiratory rate of 10 per minute. She was 5 feet 4 inches tall and weighed 93 pounds (78% of ideal body weight). Physical examination revealed a cardiac midsystolic click, a soft holosystolic murmur, clear lungs, no edema, and normal abdomen.

Serum electrolytes revealed normal potassium, magnesium, and bicarbonate levels. An electrocardiogram (EKG) revealed sinus bradycardia with a heart rate of 50 beats per minute, normal rate-corrected QT (QTc) interval, and no ST-T wave changes; QRS voltage was reduced in the limb leads. Chest X-ray showed an enlarged cardiac silhouette. In view of the cardiac examination and chest X-ray, an echocardiogram was ordered. It revealed mitral valve prolapse, a normal left ventricular ejection fraction, and no segmental wall motion abnormalities. A small to moderately sized circumferential pericardial effusion was noted without evidence of tamponade. K.L. was educated about mitral valve prolapse and continued to refeed successfully. She had no recurrence of chest pain or dyspnea as she gained a total of 16 pounds. On repeat echocardiographic evaluation 3 months later, the pericardial effusion was substantially reduced; the cardiac click and murmur were no longer noted on auscultation.

Background

Anorexia nervosa (AN) has one of the highest mortality rates of any psychiatric disorder (Arcelus et al., 2011). Of the litany of medical complications that contribute to the increased mortality rate for patients with AN, cardiovascular complications have historically been considered the most ominous. As research in this field has progressed, it has become well established that significant cardiac structural, conduction, repolarization, and hemodynamic changes do indeed occur. While the relationship of these changes to sudden cardiac death remains speculative, important

advances in this field include the use of long-term cardiac rhythm monitoring to objectively assess autonomic dysfunction and resulting cardiac rhythm abnormalities over time (Farasat et al., 2020). Additionally, cardiac complications are being better understood on a population basis, with new data showing that male AN patients experience more cardiac complications than their female counterparts (Kalla et al., 2017). Despite these preliminary advances in the understanding of both epidemiology and pathophysiology, a complete understanding of structural and physiologic cardiovascular changes is of growing import for clinicians caring for patients with eating disorders.

Heart Rate and Blood Pressure

Two of the most prominent and consistent findings in patients with anorexia nervosa are resting bradycardia (heart rate <60 beats per minute) and hypotension (systolic blood pressure <90 mm Hg). In addition to thinness, bradycardia may be the most common presenting sign of AN, and in the correct context, it should alert the clinician to the possibility of an eating disorder (Grover, Robin, and Gharahbaghian, 2012). The bradycardia of anorexia nervosa generally resolves, with heart rates increasing into the normal range of 60–90 beats per minute, as weight is restored to a level greater than 85%–90% of ideal body weight; it may resolve even earlier with a stable pattern of nutritional replenishment and progressive weight gain (Shamim et al., 2003). Many patients believe that their low resting heart rates are attributable to their perceived physical fitness. It is important to educate patients on an important distinction: while truly conditioned athletes have low heart rates at rest with only small increases with exertion, malnourished patients who have anorexia nervosa have low resting heart rates but, due to diffuse musculoskeletal weakness and debilitation, become tachycardic with even minimal exertion, which can result in immediate resolution of severe junctional bradycardia (Krantz, Gaudiani, et al., 2011).

Severe bradycardia with beats-per-minute rates as low as in the 20s have been observed (Yahalom et al., 2013), and this sometimes creates panic among clinicians leading to unnecessary pacemaker placement.

A 2019 case series examined this issue in detail with discussion of four cases of AN patients treated on an inpatient medical stabilization unit, all with symptomatic bradycardia (Cotter et al., 2019). It was concluded from this investigation that permanent pacing is rarely indicated in the setting of bradycardia in the acute stages of severe anorexia nervosa, and that even temporary pacing should be considered only in the instance of life-threatening hemodynamic instability. It is notable that the symptomatology commonly seen in anorexia nervosa, such as fatigue, exercise intolerance, syncope, and presyncope, overlaps with symptoms commonly associated with severe bradycardia due to structural heart disease but may in fact be noncardiogenic in origin. These symptoms are often nonspecific and may reflect severe malnutrition, dehydration, hypoglycemia, and cardiopulmonary deconditioning. Care must be taken in considering the patient's overall clinical picture, as the current class I indication for permanent pacemaker placement is rarely applicable to this population. This invasive intervention can subject patients to procedural risk, worsening of body image, and the need to explant the device (Cotter et al., 2019).

While sinus bradycardia is generally well tolerated and asymptomatic, when combined with orthostatic blood pressure changes, symptoms of presyncope or even frank syncope can occur. It is reasonable to recommend admission to an inpatient specialized medical stabilization unit for telemetry and vital sign monitoring for any patient with recent syncope, with a resting heart rate of less than 40 beats per minute, or with bradycardia other than sinus origin (Sachs et al., 2016). Resolution of orthostatic changes often lags behind normalization of the resting heart rate and may persist beyond the initial refeeding phase until significant weight restoration is achieved, that is, to >85%–90% of ideal body weight (IBW).

Although clinicians might find comfort in a "normal" heart rate, a "relative tachycardia" (pulse 70–100 at rest) in a patient with severe malnutrition due to anorexia nervosa may have more concerning implications, including occult infection (Derman and Szabo, 2006). Indeed, because resting bradycardia is such a constant finding in nutritionally depleted patients with severe anorexia, these "normal" heart rates, even

if not strictly in the elevated range (pulse >100), are likely to be either a side effect of medication or an impending medical complication and should be viewed as a warning sign in need of expert medical evaluation (Krantz and Mehler, 2004). Finally, despite resting bradycardia, hospitalized patients with anorexia nervosa, during the early phases of weight restoration, often manifest rapid cardioacceleration with simple activities of daily living, with heart rates in the 140–160 bpm range (Krantz, Gaudiani, et al., 2011), triggered by minimal activity. This phenomenon remains under active investigation, but it generally normalizes as weight restoration proceeds, especially as weight approaches 90% of IBW.

Though formal studies are lacking, there is anecdotal evidence of an increased prevalence of positional orthostatic tachycardia syndrome (POTS) among patients with anorexia nervosa. This condition is a form of dysautonomia that is typified by excessive tachycardia upon change in position from supine to standing, which may be accompanied by a wide array of other symptoms. Distinguishing POTS from the expected orthostatic changes that occur with malnutrition can be difficult. An increase in heart rate of more than 30 beats per minute with standing may alert the clinician to a possible diagnosis of POTS. If these changes do not improve as expected with weight restoration, further investigation and autonomic hygiene measures, including isometric (including squatting) exercise and hydration strategies, may be warranted to limit symptoms (Shen W-K et al., 2017). Regardless, it is prudent not to pursue or invoke the diagnosis of POTS in the early phase of treatment for those with extreme forms of AN when the symptoms are simply inherent to heightened parasympathetic tone and autonomic dysregulation seen in marked malnutrition.

Autonomic Dysfunction

The progressive bradycardia seen with increasing severity of anorexia nervosa has been an ongoing area of scientific inquiry on the influence of the parasympathetic and sympathetic nervous systems on cardiac function. Some researchers have hypothesized that increased vagal tone as part of an adaptive energy-conservation response leads to a decreased

resting heart rate (Casiero and Frishman, 2006). But the data are not as clear as would be theoretically expected. Some studies have found a marked reduction in both parasympathetic and sympathetic tone in patients with anorexia nervosa (Rechlin et al., 1998). Others have demonstrated an increase in parasympathetic activity with unchanged sympathetic tone (Galetta et al., 2003).

Autonomic changes in AN have generally been evaluated using the surrogate marker of heart rate variability. This moment-to-moment fluctuation in heart rate reflects the underlying stability of autonomic nervous system function through the integration of sympathetic and parasympathetic modulation. In limited studies of other patient populations, changes in heart rate variability were associated with increased mortality (Hon and Lee, 1963; Kleiger et al., 1987). Despite several studies on this topic in the AN literature, the findings have not been consistent, often resulting in contradictory conclusions (Mazurak et al., 2011). The complex nature of this disease, along with the likely changes in autonomic function with disease progression, presumably accounts for these inconsistencies (Nakai et al., 2015).

The most comprehensive assessment of autonomic dysfunction over time was seen in a pilot study to assess the feasibility of use of an insertable cardiac monitor in patients with severe anorexia and markedly reduced body mass index. Heart rate response to activity in more than 360 days of monitoring was assessed in 11 female study participants representing a total of 10.5 years of patient monitoring (Farasat et al., 2020). It was observed that autonomic dysfunction in these patients manifests as profound resting bradycardia as well as chronotropic incompetence, both of which improve with weight restoration (Shamim et al., 2003). Chronotropic incompetence in this study manifested as a blunted HR response to exertion, whereby weight restoration reversed this abnormality. Now that the feasibility and safety of this type of monitoring has been established, it paves the way for future studies to examine this question in more depth. Particularly valuable would be studies stratified to the duration of illness and subtype of anorexia nervosa, where it appears that chronic severe anorexia nervosa is associated with reduced heart rate variability (HRV), particularly high-frequency power

(Melanson et al., 2004), which portends a risk of sudden cardiac death. This contrasts with studies of adolescent patients with anorexia, where a marked increase in vagal activity (parasympathetic tone) on analysis of HRV was shown (Petretta et al., 1997). As such, larger, longitudinal studies are needed to better understand the complex changes in autonomic function throughout the various stages of anorexia nervosa.

Tachy and Brady Arrhythmias

Significant concern has previously centered on the potential propensity for patients with anorexia nervosa to develop QTc-interval prolongation, which can lead to torsade de pointes, a life-threatening form of polymorphic ventricular tachycardia that can degenerate into ventricular fibrillation. QTc-interval prolongation and torsade de pointes were long hypothesized to account for the risk of sudden death in anorexia nervosa (Cooke et al., 1994; Isager et al., 1985). As more data have been gathered, however, it seems much less certain that QTc-interval prolongation is inherent to AN; rather, it may be due to extrinsic factors such as electrolyte abnormalities, especially hypokalemia, or the effects of medications such as psychotropics, anxiolytics, macrolide antibiotics, and antiemetics, particularly among hospitalized patients (Krantz, Sabel, et al., 2012).

The ongoing question of whether QTc prolongation is an intrinsic feature of eating disorders has now been definitively put to rest by a prospective cohort study that constitutes the largest study of patients with eating disorders. Examination of EKG data from 1,026 adults admitted into residential eating disorder treatment showed that the population-mean QTc was similar to that seen in age- and gender-matched healthy cohorts (Krantz, Blalock, et al., 2020) and varied by eating disorder subtype. Specifically, the mean QTc interval was within one standard deviation of matched controls (figure 7.1). The few identified cases of marked QTc prolongation in this study all had extrinsic factors such as hypokalemia and use of QT-prolonging medications. A 2018 study by Frederiksen et al. examined EKGs of 430 adult women with anorexia nervosa and 123 healthy controls. Similarly, they found no difference in

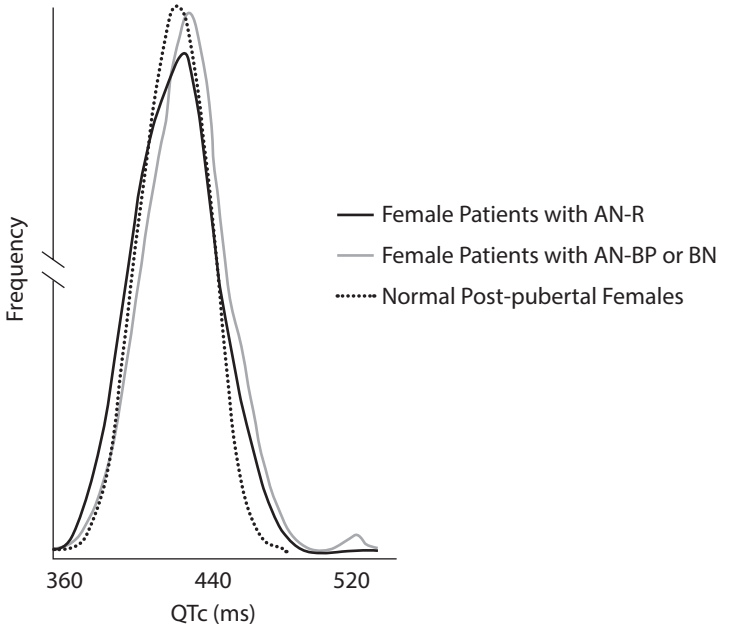


Figure 7.1. The distribution of QTc values among those with AN-R (*black line*) and those with AN-BP and BP (*gray line*) are superimposed on a previously normative QTc distribution among healthy postpubertal females. AN-BP = anorexia nervosa with binge-purge behaviors; AN-R = anorexia nervosa; BN = bulimia nervosa; QTc = corrected QT interval. From Am J Med 2020.

the proportion of subjects with prolonged QTc between the anorexia nervosa patients and healthy controls. Over a median follow-up period of 10.1 years, however, the AN cohort had an increased risk of the composite endpoint of ventricular tachycardia, cardiac arrest, and aborted cardiac arrest, as well as increased risk of all-cause mortality, compared to the healthy controls. These findings suggest that the increased risk for adverse cardiac events and death seen in patients with anorexia nervosa is unlikely to be related to intrinsic QTc prolongation with resultant arrhythmias (Frederiksen et al., 2018). Based on these conclusions, the finding of QT prolongation in a patient with anorexia nervosa should prompt a search for potential independent causes, such as electrolyte disturbances.

Regardless of the underlying contributors, however, patients presenting with a prolonged QTc interval warrant careful monitoring. Individu-

als with a QTc exceeding 500 milliseconds should be admitted to an inpatient unit for telemetry monitoring, whereas those with a QTc between 440 and 470 milliseconds should have daily EKG monitoring until it remains normal for a few days (Sachs et al., 2016). It is known that the risk of torsade de pointes increases exponentially when the QTc interval exceeds 500 milliseconds (Bednar, Harrigan, and Ruskin, 2002). Among drug-associated QTc prolongation, antidepressants, antiemetic, gastrointestinal prokinetic agents, antipsychotics, and antibiotics are the most frequent offending agents. Thus, marked QTc prolongation can occur in anorexia nervosa patients with severe hypokalemia. This has the potential to lead to torsade de pointes but is considered secondary to extrinsic factors rather than reflective of intrinsic myocardial electrical instability. Importantly, the machine-generated QTc interval cannot always be trusted by clinicians caring for eating disorder patients (figure 7.2). It has been shown that the computer-generated Bazett-corrected QT algorithms can miscalculate QTc, particularly in the setting of ectopy. Manual verification should always be considered to ensure accurate identification of potential QT prolongation (Tran et al., 2019).

Another marker of increased arrhythmic risk is a measure known as *QT dispersion*. This is defined by interlead variation in QT-segment length on a routine 12-lead EKG. Normally, the length of the QT interval is similar in each of these leads. QT dispersion refers to the difference between

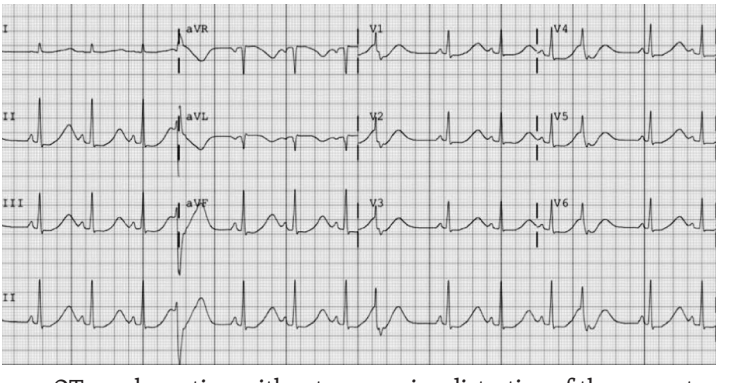


Figure 7.2. QTc prolongation with ectopy causing distortion of the computer-generated QTc calculation, including prolonged QT and premature ventricular complexes.

the maximum QT interval and the minimum QT interval occurring in any of the 12 leads. QT dispersion reflects heterogeneous ventricular depolarization and, when abnormally increased, may indicate cardiac irritability and a heightened propensity to develop serious ventricular arrhythmias, as has been shown to occur following myocardial infarction (Furukawa et al., 2006).

Over the past several years, increased QT dispersion has been found in young women with anorexia nervosa before refeeding (Mont et al., 2003). These patients may have a twofold or greater increase in QT dispersion, and this finding inversely correlates with resting metabolic rate; thus, there is a decrease in QT dispersion with refeeding and increased metabolic rate (Krantz, Donahoo, et al., 2005). A newer marker derived from continuous Holter monitoring, the QT variability index, may also be abnormal in anorexia and resolve with weight restoration (Koschke et al., 2010). If prospectively validated, this index could eventually serve as a parameter for risk stratification of patients. Regardless, QT dispersion is a widely available and inexpensive measure that may reflect both metabolic status and the potential for arrhythmia, though at present it is not recommended for routine use in clinical practice.

Though ventricular arrhythmias are the most feared consequence of repolarization changes found in some patients with AN, conduction abnormalities, including high-grade atrioventricular (AV) block, may also, albeit rarely, occur. Sinus bradycardia is both common and expected but in rare cases can progress to development of a second-degree AV block or junctional escape rhythm (Kanbur et al., 2009; Kossaify, 2010). A case of persistent junctional escape rhythm that was extinguished with exercise has also been described (Krantz, Gaudiani, et al., 2011). The finding that bradycardia is easily overcome with exercise suggests that the elevated parasympathetic tone in anorexia is an adaptive physiologic response and is not pathologic in origin. Despite this observation, however, more than 10 patient-years of monitoring with an insertable cardiac monitor showed that the predominant abnormal rhythm events were bradycardia and sinus pauses. Nonetheless, it is notable that episodes of ventricular tachyarrhythmia and complex ventricular ectopy were absent (Farasat et al., 2020). This finding suggests that the cause

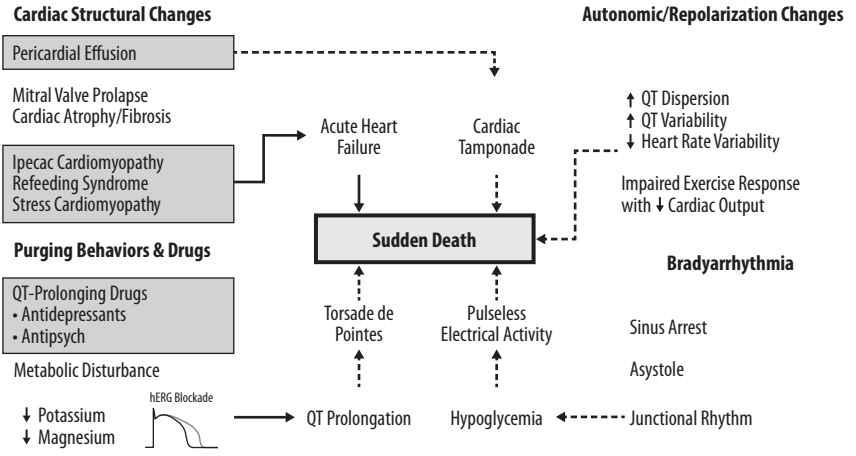


Figure 7.3. Conceptual Model: Cardiovascular Complications of Eating Disorders and Mortality Risk

of death in these patients seems less likely to be related to malignant ventricular arrhythmia as had been previously postulated by Isner et al. (1985). Regardless, it is imperative that clinicians identify marked QTc prolongation among patients with anorexia nervosa, as this portends a substantial risk for torsade de points as described in Isner's classic description of this phenomena 35 years ago (Isner et al., 1985).

A conceptual model for the risk of sudden cardiac death, as shown in figure 7.3, includes the potential for iatrogenic risk of torsade de pointes as well as the prominent possibility that bradyarrhythmic death or pulseless electrical activity may mediate sudden death in extreme starvation. The cardiac structural abnormalities and their contribution to this framework for understanding the potential substrate that heightens risk of sudden death are highlighted in the next section.

Structural Abnormalities

Substantial weight loss is accompanied by shrinkage of both skeletal and cardiac muscle. Chronic starvation results in left ventricular atrophy, reduced left ventricular mass index, and reduction in chamber dimensions

(Docx, Simons, et al., 2014). Reduction in chamber volumes can lead to annular laxity and subsequent mitral valve prolapse, despite no inherent pathology of the valves. Rarely, mitral valve prolapse can be associated with benign chest pain and palpitations and could account for the symptomatology occasionally present in these patients, as described in the case study that opens this chapter. An echocardiogram can document its presence. Overall, mitral valve prolapse is a self-limited condition that will improve with weight restoration absent underlying severe myxomatous mitral valve leaflet degeneration. Another reversible phenomena described in anorexia nervosa is Takotsubo, or apical variant stress-cardiomyopathy, also known as *apical balloon syndrome*, whereby myocardial dysfunction disproportionate to cardiac biomarkers occurs in the absence of epicardial coronary artery disease (Kim et al. 2011). The stress of severe malnutrition along with the attendant psychiatric state may underlie this phenomenon.

The most prominent and consistently documented cardiac structural change in anorexia nervosa is reduction in left ventricular myocardial mass. This change has been suggested to relate to alterations in thyroid hormone levels, although such an association remains speculative (Carlomagno et al., 2011). It makes intuitive sense that decrease in left ventricular mass would be proportional to decreases in total body mass, and the direct correlation between low body mass index and low left ventricular mass index has now been demonstrated using echocardiographic data (Kuwabara et al., 2018).

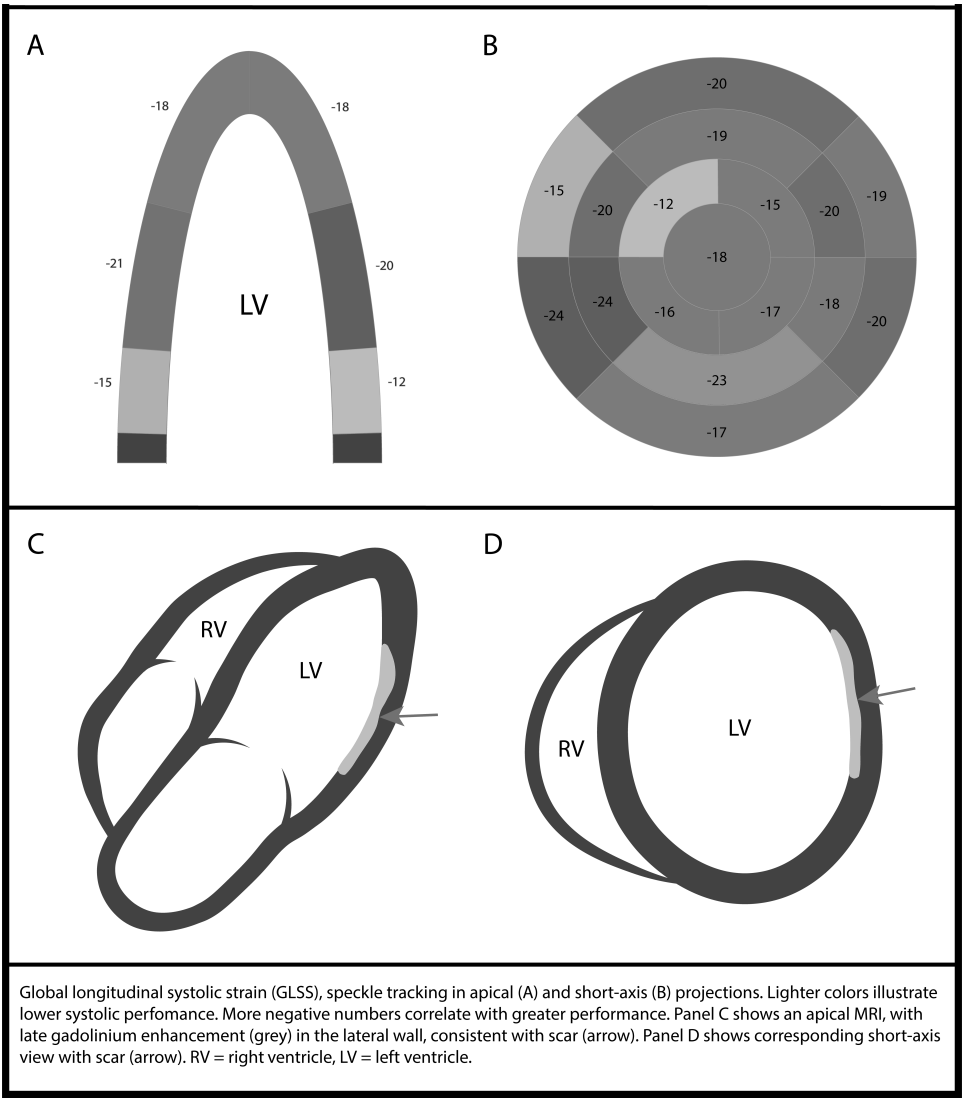
The documented left ventricular structural changes have raised questions about the possibility of resulting functional changes. It seems that while overall left ventricular systolic function is preserved, changes in the geometry of the left ventricle may adversely affect both cardiac performance and remodeling (Almeida-Jones et al., 2014). Advances in echocardiographic imaging have allowed for a more detailed assessment of myocardial performance than was previously possible. It is now widely accepted that measurement of global longitudinal systolic strain can detect subclinical left ventricular dysfunction in the presence of preserved left ventricular ejection fraction. Use of this modality has shown subclinical ventricular dysfunction disproportionately at the apex

among children with anorexia nervosa, binge-purge subtype (Morris et al., 2017). This imaging technique has great potential to shed light on the potential preclinical cardiac dysfunction that may exist in this population. Data from adult patients with anorexia nervosa, however, are currently lacking.

In addition to the gross changes in myocardial mass, there is the potential for microscopic changes of myocardial fibrosis, which may have more ominous consequences. Myocardial scarring, even on a microscopic level, could provide a substrate for malignant arrhythmias, potentially accounting for the heightened risk of sudden death in anorexia nervosa. While histological findings observed in a single autopsy report (Lamzabi et al., 2015) suggest this possibility, studies using magnetic resonance imaging (MRI) have shown inconsistent results. A 2013 observational study of 40 patients with anorexia nervosa showed myocardial fibrosis, defined as late gadolinium enhancement on cardiac MRI, associated with this cohort (Oflaz et al., 2013). A more recent study of 16 patients with anorexia nervosa, however, confirmed the presence of low cardiac mass and volumes in these patients but did not show late gadolinium enhancement (Chu et al., 2019). It therefore remains unclear whether development myocardial fibrosis is a consistent phenomenon in this patient population. Further studies, stratified to age and disease severity, would be helpful to further elucidate this point. The potential changes in myocardial strain and cardiac fibrosis that could underlie sudden death risk are depicted in figure 7.4.

Finally, pericardial effusion is now known to occur in chronic anorexia nervosa. In a study of adolescents, 22% of patients had pericardial effusions, most of which resolved following refeeding (Docx et al., 2010). A subsequent meta-analysis of 23 studies totaling 960 patients found a similarly increased incidence of pericardial effusion by 25% among these patients. Pericardial effusion, along with other identified structural abnormalities, showed a trend toward improvement with weight restoration (Smythe et al., 2020).

Despite the unexpected prevalence of this finding, nearly all patients with pericardial effusions were asymptomatic, and only one case of tamponade is described in the literature (Kircher et al., 2012). Given the



Global longitudinal systolic strain (GLSS), speckle tracking in apical (A) and short-axis (B) projections. Lighter colors illustrate lower systolic perfomance. More negative numbers correlate with greater performance. Panel C shows an apical MRI, with late gadolinium enhancement (grey) in the lateral wall, consistent with scar (arrow). Panel D shows corresponding short-axis view with scar (arrow). RV = right ventricle, LV = left ventricle.

Figure 7.4. The lefthand panels demonstrate myocardial speckle tracking maps to illustrate this emerging technology. The righthand panels show examples of MRI. *Arrows* depict areas of myocardial scar as manifested by late gadolinium enhancement on cardiac MRI.

benign nature of pericardial effusion, echocardiography should be considered primarily when there is clinical concern about a large pericardial effusion, such as an enlarged cardiac silhouette on chest X-ray or low-voltage QRS or electrical alternans on EKG, and for patients with hemodynamic instability, elevated jugular venous pressure, or complaint of prominent dyspnea on exertion.

In the past there was concern that the observed mortality in anorexia nervosa was a consequence of abnormal lipid metabolism leading to premature atherosclerosis and myocardial infarction. Autopsy studies published more than 30 years ago, however, found no evidence of obstructive coronary disease (Isner et al., 1985). Despite isolated case reports of acute coronary syndrome and myocardial infarctions in this patient population, atherosclerotic heart disease is not considered to be a specific cause of morbidity for patients with anorexia nervosa (Abuzeid and Glover, 2011). Some of the debate on this issue has stemmed from conflicting data on lipid abnormalities in this population, with some studies citing harmful slowed LDL-cholesterol transport, and others reporting elevated total cholesterol due to high levels of beneficial HDL cholesterol (Mehler, Lezotte, and Eckel, 1998; Weinbrenner et al., 2004). It appears that anorexia nervosa and its associated weight loss may actually be protective against other risk factors for atherosclerosis, as these patients have a significantly decreased risk for development of type 2 diabetes (Ji, Sundquist, and Sundquist, 2016) and generally are not hypertensive. Further studies are needed to better elucidate the subtleties of lipid metabolism, including particle size, particle number, and cholesterol efflux. This will allow enhanced prediction of global 10-year atherosclerotic risk for these patients. In the meantime, there seems to be no medical role for routine periodic lipid monitoring for patients who have anorexia nervosa (Sachs et al., 2016), beyond the more widely recommended initial screening for familial hyperlipidemia (Stone et al., 2014).

Despite this seemingly decreased risk for atherosclerotic vascular disease, it is important to remember that patients with anorexia nervosa are not immune to the development of conditions such as hypertension and hyperlipidemia, particularly with advancing age. These patients should be treated according to general clinical guidelines. Yet, while

weight-based dosing of cardiovascular medications such as antihypertensives and statins is not recommended for adults, it is nevertheless important to keep in mind both the low body weight of these patients and their decreased left ventricular mass. Thus, medications such as beta blockers, calcium channel blockers, and angiotensin converting enzyme inhibitors may be safely and effectively used, but lower starting doses should be selected, with slow and cautious titration.

Summary

Cardiac complications are likely to contribute to the morbidity and mortality seen in patients with anorexia nervosa and, to a lesser extent, those with bulimia nervosa. Most of the cardiac complications accompany the severe malnutrition associated with advanced stages of these eating disorders. A thorough cardiac history and meticulous physical examination should be performed, and basic chemistry panels, a baseline EKG, and orthostatic vital signs should be obtained for all patients after the first visit, to screen for abnormalities that need ongoing evaluation and treatment.

Anorexia nervosa is known to be associated with several serious cardiac complications. Most of these issues, however, resolve with timely weight restoration and do not lead to permanent sequelae. These cardiac abnormalities not only are present in the untreated state but can arise as a result of the refeeding process. With bulimia nervosa, most of the cardiac problems are due to electrolyte abnormalities related to purging behaviors. Despite these known and well-described changes, the true underlying cause of sudden death seen in this condition remains uncertain. Previous theories, such as ventricular arrhythmias due to inherent repolarization changes, seem less likely in the light of recent long-term internal monitoring data (Farasat et al., 2019) and remain an area of future research.

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8

Obstetric-Gynecologic Endocrinology and Osteoporosis

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COMMON QUESTIONS

What characteristic hypothalamic-gonadal hormonal alterations occur in patients with anorexia nervosa?
When does amenorrhea occur and resolve during the course of anorexia nervosa?
What is the relationship between weight loss and the onset of functional amenorrhea in anorexia nervosa?
What other endocrine hormonal responses occur in patients with anorexia nervosa?
What therapeutic interventions may help treat the amenorrhea of anorexia nervosa?
Is such treatment important?
Do patients with a history of an eating disorder have long-term problems with fertility and pregnancy?
How common is osteoporosis in patients with eating disorders?
What are the clinical consequences of low bone mass in patients with eating disorders?
What features of eating disorders contribute to bone loss?
How are the diagnoses of osteoporosis and osteopenia made?
How are osteoporosis and osteopenia treated and monitored?

What other therapeutic interventions are indicated to prevent bone loss and attenuate the risk of fracture?

Should all patients with anorexia nervosa who have osteoporosis be treated?

Case Study 1

H.M. was a 20-year-old female who presented with secondary amenorrhea. She had undergone normal puberty at age 12, initially having regular menstrual cycles. Secondary amenorrhea began at 18 years of age. Other family members had typically undergone normal growth and sexual development. H.M. was "overweight" in grade school and in the first two years of high school. She expressed a dislike for obesity, as her mother and sister were "fat." Subsequently, she began markedly restricting her caloric intake and excessively exercising to achieve weight loss. In addition to the amenorrhea, she complained of cold intolerance, brittle hair, dry skin, and fatigue. H.M. weighed 100 pounds and was 5 feet 6 inches tall, 77% IBW. Pubic hair and breast development were scant but normal. On careful questioning, she indicated that she was adhering to a strict 500-calorie-per-day diet. She exercised daily for 1–2 hours but denied self-induced vomiting. She had initiated her dieting at age 16; within the next 3 years, when her weight reached 104 pounds, her menstrual periods stopped. The diagnosis of anorexia nervosa was made, and she was referred to an internal medicine specialist for further evaluation.

Case Study 2

C.R., a 28-year-old female, was 5 feet 6 inches tall and weighed 98 pounds. She had a 12-year history of anorexia nervosa. She had initially weighed 164 pounds; after a 35% loss in body weight, she developed secondary amenorrhea. Over the course of her illness, she had lost 2 inches in height. Recently she had been seen by her primary care provider for evaluation of low back pain. X-rays showed an L2 compression fracture, which a radiologist interpreted to be of recent onset. C.R. had never had a previous bone density test and was referred for evaluation of her medical status.

Background

People with anorexia nervosa (AN) may present with an extremely complex medical disorder resulting from the sociocultural pressures for thinness. Part of the results are numerous psychoneuroendocrine-nutritional medical stresses as the patient relentlessly pursues thinness. Female patients with anorexia nervosa who experience marked ongoing weight loss may present for evaluation of the resultant amenorrhea, which is defined as having missed at least three consecutive menstrual cycles.

Gynecologic Effects of Anorexia Nervosa

The Menstrual Cycle

The neuroendocrine regulation of normal female reproductive functions depends on a rhythm of nerve impulses generated within the medial basal hypothalamus, which governs the pulsatile release of gonadotropin-releasing hormone (GnRH) from hypothalamic nerve terminals. Pulsatile GnRH release is the central controller of the pituitary gland's luteinizing hormone (LH) and follicle-stimulating hormone (FSH) secretion, which determine the onset of normal menstrual function (Doufas and Mastorakos, 2000). At puberty, an increase in both the frequency and the amplitude of GnRH secretion induces LH-FSH secretion, which is involved in the monthly menstrual cycle. Various factors contribute to the tight regulation of the process, including different sex hormones and leptin. Patients with anorexia nervosa, however, have a characteristic *hypothalamic amenorrhea syndrome*, defined by a variable reduction in pulsatile hypothalamic GnRH gonadostat secretion and signaling to the pituitary gland, resulting in a failure of ovulation. The degree of impairment varies among patients, but in general, the frequency and amplitude of the LH-FSH pulses are diminished in anorexia nervosa, with reversion to a prepubertal pattern and development of the commonly found amenorrheic state. Thus, this functional hypogonadotropic, hypogonadism-induced amenorrhea in AN reflects a temporary, reversible disturbance of hypothalamic-pituitary function

(Singhal, Misra, and Klibanski, 2014). Moreover, most amenorrhea in patients with anorexia nervosa is of the secondary type, meaning that the individual previously had normal menstrual periods prior to their caloric restriction and weight loss; this contrasts with primary amenorrhea, in which there has never been the onset of menses. When the onset of AN begins at the ages of 11 or 12, those patients may indeed have primary amenorrhea.

Of female patients with anorexia nervosa, 20%–25% may experience amenorrhea before the onset of significant weight loss, and 60%–85% will experience amenorrhea with dieting and its associated weight loss (El Ghoch, Bazzani, and Dalle Grave, 2014). Some patients experience amenorrhea only after more marked weight loss. This variability explains why amenorrhea was dropped as a criterion for the diagnosis of anorexia nervosa by *DSM-5* (APA, 2013). Menstrual irregularities are also common in bulimia nervosa (BN), albeit much less severe and less prevalent than in AN (Gendall et al., 2000). On restoring weight toward normal, many patients will have resumption of their normal menstrual cycle. More than 40 years ago, Frisch and McArthur (1974) noted a critical relationship between the occurrence of amenorrhea and body mass index (BMI), body fat, and percentage of body weight loss. This relationship was confirmed more recently in a study demonstrating that menses can be expected to resume at a weight that exceeds 90%–93% of ideal body weight (Golden, Jacobson, Schebendach, et al., 1997). Other studies have found the weight requirement for resumption of menses to be more variable and better predicted by the weight at which menstruation ceased (Swenne, 2004). A more recent cohort study of 56 adolescent females found a return of menses at a mean BMI percentile of 27 (Golden, Jacobson, Sterling, et al., 2008).

Despite these relationships, features other than weight loss may also be causing the amenorrhea. Given that the onset of amenorrhea can precede significant weight loss in about one-quarter of these women, that amenorrhea persists even after weight restoration in some women, and that menstruation resumes in some women despite a low body weight (Miller, Grinspoon, et al., 2004), other factors may also promote this process. These factors include adaptive hormonal responses to sociocul-

tural psychic stress, excessive exercise, and chronic nutritional energy deficiency (Warren et al., 2002), along with reduced thyroid hormone and leptin levels. Stress, anxiety, exercise, smoking, and abnormal eating habits may contribute. As a result of the gonadotropin failure, however, individuals with anorexia nervosa have decreased blood levels of the sex hormones (estradiol, estrone, progesterone, and testosterone) (Miller, Lawson, et al., 2007). Consequently, women with active AN may not always have withdrawal bleeding in response to a diagnostic progesterone challenge, which would indicate a more profound hypothalamic-pituitary gonadotropin deficiency state.

Thus, a critical weight may be necessary but not singularly sufficient for menstrual function, and other factors may also be of importance (Gendall et al., 2006). The variation in patterns of menstrual disturbance with anorexia nervosa is in part the basis for removal of amenorrhea as a criterion for the diagnosis of AN.

Treatment of Amenorrhea

Treating a patient with functional (hypothalamic) amenorrhea is not easy. The underlying cause of the amenorrhea, as it relates to sociocultural and environmental psychic stress, needs to be evaluated and primarily addressed. Assessment for clinical or subclinical depressive illness is warranted. Also important is a detailed history concerning parental relationships, self-image, social and environmental issues, stressors, sexuality, interpersonal relationships, and support systems. Spontaneous recovery of menstrual periods may occur with correction of both the eating disorder and the underlying psychopathology, along with weight restoration. The corollary of this, however, is that even with weight gain, menstruation may not resume if there is persistence of the abnormal eating behaviors or psychogenic factors that, in part, initiated the cessation of menses.

The progestin challenge test is sometimes used in the evaluation of amenorrhea. It may be of value to reassure the patient with an eating disorder who is working to recover that her reproductive system is in a temporary semihibernation state but is functionally intact and ready to

reactivate if she can restore her weight. The patient is prescribed 10 mg of medroxyprogesterone (Provera) daily for 5 days. Withdrawal bleeding within 10 days of the progesterone administration indicates adequate estrogen levels and is consistent simply with functional amenorrhea. Conversely, a lack of withdrawal bleeding connotes a state of profound estrogen deficiency or another independent gynecologic-endocrine problem that needs further evaluation. A serum LH level below 5 mIU/mL is indicative of hypothalamic dysfunction in patients without withdrawal bleeding.

In the clinical setting of anorexia nervosa, however, especially with excessive weight loss, the progestin challenge test is not a crucial diagnostic exercise for evaluating the commonly found amenorrhea. Rather, efforts should be directed toward overall treatment of the eating disorder because there is generally little inherent value in administering female sex hormones to patients with AN. Although withdrawal bleeding can be calming, it may also promote a false sense of well-being and minimize the urgency for engaging in therapy for the eating disorder. Some evidence suggests that when serum leptin levels revert to normal, that resumption of menses is imminent.

Pregnancy

The presence of an active eating disorder is thought to be a relative contraindication for pregnancy and for infertility treatments. When pregnancy occurs with an eating disorder, it is more often in individuals with bulimia nervosa who are near-normal body weight, although some women with anorexia nervosa do rarely ovulate and become pregnant despite their amenorrhea. Thus, it is worth warning them that some form of contraception is prudent notwithstanding their lack of periods. In fact, unplanned pregnancies are a significant problem in AN. During the pregnancy, many patients subdue their binge eating and purging behaviors but return to these behaviors with ferocity in the puerperium (Easter, Treasure, and Micali, 2011). If pregnancy does occur, both the pregnancy and lactation impose a tremendous stress on the maternal

skeleton for mineralization of the fetal and newborn skeleton. Hence, it is important to provide adequate vitamin D and calcium. Pregnancy in a patient with active AN is associated with a greater incidence of premature birth, smaller head circumference, miscarriages, and low-birthweight infants (Charbonneau and Seabrook, 2019). If women with a recent history of an eating disorder want to become pregnant, it is usually advisable to refer them to a reproductive endocrinologist for discussion of treatment options, including possible fertility treatments, and to ensure that they have sufficient psychiatric support to help control their abnormal eating behaviors during the pregnancy (Paslakis and de Zwaan, 2019).

Pregnancy for a woman with an eating disorder is a decision that should be made only after judicious deliberation. Referral to a multidisciplinary team is critical for all these patients so that the health of the mother and the fetus are preserved to promote the best possible outcome. In the long term, after a patient recovers from AN or BN, there seems to be no consistent impediment to her ability to become pregnant. In the past, fertility was believed to be permanently impaired by a history of an eating disorder, but this is not clearly the case (Tabler et al., 2018). Nonetheless, these women tend to seek consultation with infertility specialists about clomid-type medications if concerned about possible residual fertility problems. Also, sexual dysfunction, in the form of diminished libido, and postpartum depression are more common in patients with active eating disorders.

Osteoporosis in Anorexia Nervosa

Osteoporosis is a disease characterized by low bone mass and deterioration of the microarchitectural structure of bone, resulting in bone fragility and the clinical syndrome of nontraumatic fractures, as a direct result of the low bone mass. *Osteoporosis* is quantitatively defined by bone densitometry measurements, using dual-energy X-ray absorptiometry (DEXA), as a T-score of –2.5 standard deviations (SD) or less at the lumbar spine, femoral neck, and total hip. A loss of bone mass with

a T-score of –1 to –2.5 SD is classified as *osteopenia* (Lorenzton and Cummings, 2015). Although there are other methods of assessing bone density, DEXA is preferred for patients with anorexia nervosa. Peak bone mass is defined as the highest level of bone mass achieved as a result of normal growth. Most skeletal bone growth occurs during childhood and adolescence. The timing of peak bone mass may vary but generally occurs between ages 17 and 22, a time that, unfortunately, frequently coincides with the onset of anorexia nervosa. Rapid and worrisome bone loss, at an annual average rate of 2.5%, occurs in young women with AN. Osteoporosis is present in almost 40% of patients with anorexia nervosa, and osteopenia is present in 92% (Grinspoon et al., 2000). Even adolescents with AN are at markedly increased risk of low bone mineral density. Both subtypes of anorexia nervosa (AN-R and AN-BP) are highly associated with low bone mineral density. Trabecular bone, found in the lumbar spine and hips, is more adversely affected than cortical bone (Faje et al., 2013). Of note, T-scores from DEXA scans should be used for adults, as opposed to Z-scores. The latter score is used for adolescent patients, for whom, due to the continuing growth in bone size, it is necessary to adjust bone mineral density according to the average values for age and sex. Also, the term *osteopenia* is best limited to adults.

Achieving an optimal peak bone mass depends on heredity and lifestyle factors. Lifestyle influences causing less-than-optimal peak bone mass may be nutritional, such as low calcium or low phosphate intake, a diet low in animal protein, excessive intake of carbonated dark soda-type drinks, a high-sodium diet, high caffeine intake, or a strictly vegetarian diet. Other lifestyle influences that have deleterious effects on bone mass include cigarette smoking, a low level of physical activity, and alcohol consumption. Weight-bearing exercise usually promotes skeletal development and an increase in bone mineral density through beneficial effects on skeletal microarchitecture, but when exercise is excessive or overly vigorous—as in the athletic amenorrhea syndrome seen in female runners—and especially at low body weight, it may be associated with low bone mass and fractures. Weight-bearing exercise may be protective only if menstruation is preserved and only at normal body weight.

Factors Influencing Bone Remodeling

Whether an individual achieves peak adult bone mass depends on the time of onset, type, and duration of the eating disorder, as well as the degree of nutritional depletion and weight loss. Bulimia nervosa is not a risk factor for low bone density. Once amenorrhea is present in anorexia nervosa, the estrogen-progesterone deficiency plays a role in arresting bone development and promoting deleterious bone resorption. The longer the duration of amenorrhea, the higher the risk of osteoporosis (Baker, Roberts, and Towell, 2000).

Anorexia nervosa appears to be a low-turnover state characterized by both increased bone resorption and decreased bone formation, in contradistinction to postmenopausal osteoporosis, which is characterized only by increased bone resorption (Steinman and Shibli-Rahhal, 2019). This imbalance or uncoupling contributes to the rapid and aggressive bone loss in individuals with AN. In addition to the hypothalamic hypogonadal state noted above, these patients have excess cortisol secretion, low IGF-1 (insulin-like growth factor 1; somatomedin C) levels, and low androgen levels. Low levels of IGF-1 reduce the levels of osteocalcin and cause abnormalities in the osteoblasts, the bone-building cells. These low levels of IGF-1, despite increased growth hormone levels, are known to be a major correlate of low bone formation in anorexia nervosa, in contrast to the high levels of IGF-1 typically found in healthy adolescents. The elevated levels of cortisol are also inversely related to levels of osteocalcin (Misra et al., 2004). All these factors, in addition to estrogen and androgen deficiency, are likely to promote the development of osteoporosis, and they render the osteoporosis associated with AN a unique and troublesome clinical entity (Miller, Lawson, et al., 2007). Markers of bone resorption such as N-teleopeptide and deoxypyridinoline are higher in patients with anorexia nervosa, while markers of bone formation such as osteocalcin are not concomitantly elevated. This pattern is most closely analogous to steroid-induced osteoporosis. Serum levels of calcium, vitamin D, and parathyroid hormone, however, are all within the normal range. Body weight (especially fat cell mass) and muscle strength are positive predictors of favorable bone mineral

density in young women. Patients with anorexia nervosa have an altered body composition with depleted fat stores. In addition, in females, the fat cell is capable of independent estrogen synthesis from adrenal androgen precursors. A total body fat cell mass of at least 8% is generally needed for normal menstrual function. Lower BMI and a longer duration of illness both predict lower bone mineral density (Misra, 2008).

Individuals with anorexia nervosa may be intensively involved in rigorous exercise programs, contributing to their secondary amenorrhea. Secondary amenorrhea is common in many young women who continually pursue high levels of physical activities such as running, ballet dancing, cycling, and swimming. The "female athlete triad" seen in competitive female athletes is characterized by disordered eating, menstrual irregularity, and osteoporosis (Matzkin, Curry, and Whitlock, 2015). Osteoporosis and a tendency to develop stress fractures are also associated with menstrual irregularity in these women. Young, estrogen-deficient women with AN may lose bone mass at a rate as high as 3%–5% per year, which is an exceedingly alarming rate and contributes to their increased lifetime risk of bone fractures and possible kyphosis and loss of height.

Diagnosis

An assessment of bone mineral density is easily obtained with a DEXA scan, which allows a reliable and highly precise measurement of skeletal bone mineral content in the lumbar spine and hip. For every 1 SD decrement in bone mineral content of the lumbar spine, the fracture risk of an individual is increased twofold. Likewise, for the hip, for every decrement in bone mineral content of 1 SD, the attributable fracture risk is elevated 2.5 times. Currently, DEXA remains the gold standard for assessing the amount of bone mineral content, both in a screening mode and for monitoring bone density in patients with anorexia nervosa undergoing medicinal therapy for established osteoporosis. A DEXA scan should be initially obtained for all individuals with a 9- to 12-month history of AN. They should also have a follow-up DEXA scan every 2 years while the disease is active. Patients with bulimia nervosa do not require

routine DEXA scans unless a history of anorexia nervosa is elicited. Total bone strength (TBS) may eventually have value in anorexia nervosa since it measures bone quality versus bone quantity with DEXAs. Males with anorexia nervosa are also at marked increased risk for loss of bone density, and this elevated risk must be considered when treating this less common population of eating disordered patients (Mehler et al., 2008), and DEXAs should be obtained.

Onset of Bone Disease

Emerging evidence suggests that loss of bone mineral density is rapid and occurs relatively early in anorexia nervosa. Some studies suggest that an illness duration longer than 12 months predicts significant loss of bone mineral (Wong et al., 2001). In a study of 73 female patients with a mean age of 17.2 years, 20 months of amenorrhea was found to be the threshold above which the most severe osteopenia was seen (Audi et al., 2002). A longer duration of amenorrhea and later menarchal age both predict more extensive loss of bone mineral density (Maimoun et al., 2014).

Increased risk for future fracture in these young patients is a major concern, especially because fracture risk is known to double with each decrease of 1 SD in bone mineral density. Therefore, DEXA should be established as an important screening tool for all patients with disease duration greater than 9–12 months to determine the degree of reduction of bone mineral density. Moreover, there may be a psychotherapeutic benefit in providing individuals with their objective DEXA results as graphic visual evidence that they are at risk for serious medical problems due to their bone loss. Positive changes in unhealthy eating behaviors can perhaps be achieved by showing these patients that they have the same bone density as very old individuals, thus motivating them to engage more fully in treatment (Stoffman et al., 2005). Nonetheless, there is a lack of definitive scientific evidence on when exactly to order the DEXA scan for patients with AN. Finally, although the value of repeating DEXA measurements 2 years after the initial scan for women with postmenopausal osteoporosis has been recently challenged, for younger individuals with AN, a repeat DEXA scan 2 years later is recommended,

especially if the illness is ongoing or a therapeutic intervention for the bone density loss has been initiated or is being considered (Hillier et al., 2007).

Treatment

Both prevention and treatment of low bone density in anorexia nervosa are critical because, of all the potential medical complications of this eating disorder, this may be one that is never completely treatable or reversible. Moreover, young patients with osteoporosis resulting from anorexia nervosa have lifelong increased risks of fractures, in the range of 60% higher, even after their AN has been successfully treated, which can result in pain, disability, and decreased quality of life. It is worth noting, however, that there are no formal, evidence-based guidelines to inform the treatment of osteoporosis in patients with AN, but expert opinions do exist. Weight gain, resumption of menses, and adequate calcium and vitamin D intake are the mainstays of treatment for those who have either low normal bone density or osteopenia. There is no role for other pharmacological treatment options in osteopenia; more definitive treatment is reserved for osteoporosis.

Hormonal Therapy

Supplemental estrogen, in the form of hormone replacement therapy or oral contraceptives, is often prescribed in routine practice for female patients with anorexia nervosa, to minimize or ameliorate osteopenia or osteoporosis. Surveys indicate that this inappropriate practice is still common among practitioners caring for these patients. In reality, however, there is a distinct paucity of credible evidence supporting this practice (Mehler and MacKenzie, 2009). Early and small retrospective studies seemed to imply that oral contraceptives may attenuate loss of bone mineral density in AN, presumably by impeding osteoclast-mediated resorption of bone. Yet, there have been only two randomized controlled trials of estrogen therapy in anorexia nervosa. The first, a widely cited older study, demonstrated that estrogen treatment did not prevent a reduction in trabecular bone mineral density (Klibanski et al.,

1995). A more recent prospective observational trial included 50 adolescents with anorexia nervosa. After 2 years of treatment, the group that received 35 mg of ethinyl estradiol, in addition to calcium supplementation, did not show any increase in bone mineral density compared with the group that received standard treatment. In this study, osteopenia was persistent and, in some cases, was progressive despite estrogen therapy (Golden, Lanzkowsky, et al., 2002).

This lack of a beneficial effect from hormone therapy for bone mineral density in AN has been confirmed in other studies. An analytic survey including 130 women with anorexia nervosa examined four patient subgroups: (1) estrogen used in the past or the present, (2) estrogen never used, (3) estrogen currently used, and (4) estrogen used in the past but not currently. Spine and hip bone density was similar in all four subsets of women despite the differences in estrogen therapy (Grinspoon et al., 2000). An additional practical reason to refrain from using oral estrogen therapy is that it may cause resumption of menses, which may in turn give the patient a false sense of being cured and reinforce denial in those who are still at a low weight.

Despite the clear evidence that hormonal therapy is effective in maintaining bone density in postmenopausal women by impairing osteoclast-mediated bone resorption, it is increasingly appreciated that health care providers should not continue to extrapolate from the therapeutic success in this patient group to patients with anorexia nervosa, for whom estrogen's effect on bone density has been disappointing. The postmenopausal and anorectic states are very different regarding loss of bone density. The low estrogen levels that characterize the postmenopausal state, and result in bone resorption, can be effectively mitigated by the potent antiresorptive effects of estrogen replacement therapy. In contrast, the unique uncoupling of osteoblastic and osteoclastic functions in AN, which results in reduced bone formation concurrent also with increased resorption, cannot be successfully treated with estrogen. Oral estrogen replacement therapy should *not* be viewed as evidence-based treatment at this time (Robinson et al., 2017). More recent emerging evidence suggests that transdermal estrogen with cyclic progesterone may be of utility in treating the osteoporosis of anorexia nervosa

but is probably not fully restorative. This is an option to consider especially in younger patients with AN.

Most approved therapies for osteoporosis inhibit bone resorption. Another approach is anabolic therapy, in which bone formation is directly stimulated. This seems prudent in anorexia nervosa, given that this illness is also characterized by decreased bone formation. Yet, DHEA (dehydroepiandrosterone) has not proved effective in improving bone mineral density, even though it is thought to function as an anabolic factor for bone by increasing levels of IGF-1 and by stimulating osteoblast function. Similarly, transdermal testosterone has not been shown to significantly increase markers of bone formation such as osteocalcin and bone-specific alkaline phosphatase in women with AN. No rigorous scientific trials provide data on other anabolic agents such as fluoride. The recently released synthetic parathyroid hormone medication teriparatide, however, which has desirable and unique anabolic properties (increases bone mineral formation), may become a treatment option, based on the promising results of one published placebo-controlled trial that demonstrated a remarkable 6%–10% increase in bone mineral density (Fazeli et al., 2014). Caution should be exercised before using this medication for adolescents with open epiphyses or those with Paget's disease, because of the risk of osteosarcoma. Its other drawbacks are cost and the need for a daily injection (Drabkin et al., 2017).

Bisphosphonates

In view of the less-than-optimal results from most therapeutic modalities for the bone disease of anorexia nervosa and the potential permanence of low bone mineral density, there has been interest in the use of bisphosphonates for many years. Past reticence to use these agents was predicated on concerns about their safety in women of reproductive age. Bisphosphonates, whose mechanism of action is to inhibit bone resorption, carry a category C rating for safety in pregnancy because they can persist in the body for many years after discontinuation of treatment, and there is an association with teratogenesis. Thus, the long-term implications for women of childbearing age are of concern. On the other hand, the known effectiveness of bisphosphonates in decreasing bone

resorption and increasing bone mineral density in postmenopausal women has raised interest in bisphosphonates as a viable treatment for patients with AN who have osteoporosis. Oral bisphosphonates, now available in generic form, are used in weekly or monthly doses. They cannot be taken by patients with renal insufficiency or significant esophageal disease.

The first study to demonstrate the potential effectiveness of bisphosphonates was a closely monitored study of women with anorexia nervosa who received 5 mg of risedronate and had bone mineral density measurements at 6 and 9 months. Bone mineral density increased substantially in the spines of the patients who received risedronate (4.1% at 6 months), in contrast to bone loss in the controls despite weight gain (Miller, Grieco, et al., 2004). A 5% increase in bone mass over a 3-year period is clinically significant and is associated with a robust 25% reduction in fracture risk. No significant side effects were reported. In fact, bisphosphonates have been used for years in adolescent patients with osteogenesis imperfecta to increase bone density, with no reports of significant adverse events.

Bisphosphonates should not be used casually for younger female patients with AN until further research defines their long-term safety and efficacy. There are also concerns about osteonecrosis of the jaw and atypical femur fractures with the use of these medications that need to be considered (Molvik and Kahn, 2015). Therefore, given that they are effective in increasing bone mineral in adult women with AN, they should be judiciously considered for more severe cases of osteoporosis, especially when the disease is unlikely to revert in the near future. They may also have a role for male patients with AN who have normal serum testosterone levels, given biphosphonates' known efficacy in osteoporosis in men, without the concern about teratogenesis (Ebeling, 2008). There are no credible studies using calcitonin or raloxifene for patients with anorexia nervosa.

Other Medical Therapies

Denosumab is a recent addition to the medications used for the treatment of osteoporosis. It is the first biologic therapy approved to treat

osteoporosis and has a distinct mechanism of action. While it may be of future utility, there are not yet any credible studies of its use for patients with anorexia nervosa—but it is an appealing treatment given the need to receive only one injection every 6 months. Anecdotally, it has been used with success in anorexia nervosa, but bone resorption can be rapid after cessation of denosumab (table 8.1). Similarly, there are no studies of testosterone use in males with anorexia nervosa, but it may be quite reasonable to try if the serum level is low, given the hormone's well-known and favorable anabolic effects on bone. Testosterone should not be offered to adolescent males whose bones have not yet had epiphyseal closure on X-ray.

Weight Gain

The cornerstone of treatment for individuals who have anorexia nervosa is weight restoration since the negative energy state reduces bone mineral density independent of other hormonal influences (Knopf and

TABLE 8.1. Green and yellow lights and red flags for bone density challenges in AN and ARFID

Green lights
Bisphosphonates
Estrogen patches
Teriparatide
Testosterone (males)
DEXA
Yellow lights
Calcium
Vitamin D
Denosumab
Leptin
Vitamin K
TBS
Red flags
BCPs
Oral estrogen
Fluoride
Growth hormones
Early aerobic exercise

Mehler, 2019). Unfortunately, AN is often a protracted illness. Factors found to be predictive of a poor prognosis include a longer duration of illness, older age at onset, and weight loss that is persistent and a higher percentage below ideal body weight (Winston, Alwazeer, and Bankart, 2008). The direct effect of weight gain on bone density is not as clearly beneficial. Studies have yielded conflicting results on the reversibility of the demineralization. Some earlier reports about recovery suggest that restoration of normal bone mass occurs with recovery from anorexia nervosa (Wentz et al., 2003). An annualized rate of increase of 3%–4% in bone mineral density, attributed solely to weight gain, was demonstrated in older studies of patients fully recovered from AN (Zipfel et al., 2001). Many studies, however, demonstrate that bone density may never be fully recoverable. One study confirms that despite weight gain, bone mineral density did not return to baseline after one year (Misra et al., 2004). This suboptimal bone mineral accrual, in patients who have recovered from AN, may persist despite normalization of bone turnover markers and an increase in IGF-1 (Soyka et al., 2002). Of note, selective serotonin reuptake inhibitors might be associated with lower bone mineral density in anorexia nervosa, which might affect their use in these patients.

Calcium

Current recommendations suggest that adults ingest 1,200 mg of calcium a day along with 800 IU of vitamin D to achieve peak bone mass. Calcium requirements are known to increase during periods of rapid growth. Dietary calcium and vitamin D deficiency are prevalent even in healthy adolescent girls and certainly in those with anorexia nervosa (Modan-Moses et al., 2015). Thus, it is valuable to check a serum 25-OH vitamin D level in these patients and to treat a serum level <20 mg/ml. Although necessary, calcium and vitamin D are not sufficient to prevent or reverse loss of bone mineral density.

Exercise

While weight-bearing exercises are known to be beneficial for older people to maintain healthy bones, this is not so for younger people

during their anorexia nervosa illness. Aside from the predictable interference with necessary weight gain, exercise may actually be deleterious to the microarchitecture of bone while a person is amenorrheic or still at low body weight. As weight restoration progresses, and achievement of ideal body weight is near, judicious and graded recommendations to reintroduce aerobic exercise can be considered.

Summary

Significant bone loss occurs even among young patients with anorexia nervosa. Because body weight is the most important determinant of bone density, the optimal intervention is one that promotes weight restoration early in the course of the illness, before bone mineral loss has occurred. Once bone mineral is lost, the effect of weight restoration on bone density is variable. Moreover, many tested treatments for osteoporosis, including those such as estrogen therapy that are undeniably effective for postmenopausal women, have proved ineffective for individuals with anorexia nervosa. Because so few studies have been done on bisphosphonates in AN, their optimal usage is not yet entirely clear. More randomized clinical trials are needed to focus on combined anabolic/antiresorptive strategies, medications able to increase osteoblast function (including teriparatide), and bisphosphonates to prevent bone loss in young women with anorexia nervosa. As the bone quality is likely to be much better in a young patient with AN than in the postmenopausal population, judicious deliberation with patients is necessary as medications are considered. Because of the long-term increased risk of fracture in this young population of patients, there is a compelling need to define an effective and safe treatment to both prevent and reverse bone loss. Prevention, however, remains a key intervention. Therefore, identifying the patient who has anorexia, obtaining a DEXA scan, and if osteoporosis is present, starting effective treatment before the patient has suffered irreparable loss of bone density are of utmost importance, while more research is performed to define the ideal treatment approach. Similarly, gynecologic-endocrine issues in this population are still being elucidated, but thoughtful discussions regarding menstrual

and fertility concerns often arise when treating patients with eating disorders, especially anorexia nervosa.

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9

General Endocrinology

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COMMON QUESTIONS

Are there routine endocrine blood tests that should be ordered in the initial assessment of patients with eating disorders?
Why are some thyroid hormone levels often low in patients with anorexia nervosa or ARFID?
What is the significance of a low fasting blood sugar (hypoglycemia) in patients with anorexia nervosa or ARFID?
What is the significance of hypercortisolism (elevated cortisol levels) in patients with anorexia nervosa?
Is there a role for growth hormone in the treatment of anorexia nervosa?
What are the levels of appetite hormones in anorexia nervosa?
What is the relationship between diabetes insipidus and anorexia nervosa?

Case Study

G.L. was a 30-year-old woman with anorexia nervosa. At an earlier assessment, in 2010, when she reported constipation, her thyroid-stimulating hormone (TSH) level was 2.5 mIU/L; in 2011, when she reported fatigue, it was 3.5 mIU/L. Both are in the normal range (0.27–4.2 mIU/L). A repeated TSH measurement in 2012, when she

reported a family history of "thyroid problems," was slightly elevated (5.8 mIU/L). In 2013, she reported fatigue; her TSH level (5.9 mIU/L) was similar to the 2012 measurement, and her free thyroxine (T4) was normal (0.93 ng/dL). Given the stability of her TSH level, treatment was not initiated. More recently, she reported intermittent constipation and persistent fatigue.

At a recent periodic health examination, G.L.'s blood pressure was 136/79 mm Hg, and her heart rate was 57 beats per minute. Her weight had increased by 9 pounds, to 100 pounds (BMI 15.7). Findings on her thyroid examination were normal. A repeated TSH measurement was 6.5 mIU/mL, and free T4 was 1.0 ng/dL. She wondered whether she should begin thyroid replacement therapy to help her metabolism as she continued her attempts at weight restoration.

Background

People with anorexia nervosa or ARFID (avoidant/restrictive food intake disorder) have several abnormalities in neuroendocrine function. The hypothalamic amenorrhea that almost universally accompanies this disorder (see chapter 8) represents one common manifestation of hypothalamic dysfunction (Miller, 2013). Another is that, although secretion rates of cortisol (an adrenal gland steroid) are generally normal, metabolic clearance rates are decreased in AN, with the result that the half-life of cortisol may be prolonged in malnourished individuals. The normal circadian rhythm of cortisol and pituitary adrenocorticotropic hormone secretion is not disrupted in anorexia, but serum cortisol levels are not suppressed after the administration of dexamethasone as would normally be expected. Recent studies suggest that hypercortisolism develops in individuals with AN because of hypersecretion of corticotrophin-releasing hormone from the hypothalamus. The pituitary seems to respond normally to regular feedback from glucocorticoids, suggesting a defect at or above the hypothalamus that causes the elevated cortisol levels. The clinical significance of this elevated serum cortisol level remains unclear, but it may play an adverse role in the aggressive loss of bone density noted in anorexia nervosa and may facili-

tate the physiologic stress response to chronic starvation (Usdan, Khaodhiar, and Apovian, 2008). These patients with AN do not develop typical signs of Cushing's syndrome, however, given their low baseline levels of adipose tissue, and this elevated cortisol remits with resolution of anorexia nervosa.

Hormonal Abnormalities

Growth Hormone

Alterations in growth hormone (GH), insulin-like growth factor 1 (IGF-1), and growth hormone–binding protein (GHBP) are not as well understood as the abnormalities in the hypothalamic-pituitary-adrenal axis. Levels of IGF-1 are decreased in anorexia and improve with weight recovery (Støving et al., 2007). Yet, fasting GH levels in anorexia may be normal or elevated, while serum GHBP is low, indicative of GH resistance due to starvation. Serum GHBP levels correlate well with body mass index, suggesting that nutritional deprivation may down-regulate the GH receptor causing this resistance and that this effect is reversible with refeeding. Administration of IGF-1 to patients with AN has been shown to increase markers of bone turnover, but the effect of chronic administration on weight gain or bone metabolism has not been tested (Grinspoon et al., 2002). Thus, IGF-1 does not currently have a role in the treatment of anorexia nervosa, nor does GH, even for arrested linear growth and short stature in young patients with AN. There is no reason, then, for regularly measuring GH or IGF-1 levels.

Thyroid

The thyroid abnormalities in individuals with anorexia nervosa resemble those of euthyroid sick syndrome, also referred to as nonthyroidal illness syndrome, in which total thyroxine (T4) and triiodothyronine (T3) levels are low. The key, however, is that in euthyroid sick syndrome, TSH usually remains in the low-normal range (McDermott, 2020). Levels of T3 usually decrease in proportion to the degree of weight loss. Total T4 levels are low because T4 is preferentially converted to a biologically

inactive reverse T3. These changes reduce the metabolic rate and conserve limited resources in the starved state, that is, a physiologic adaption to chronic starvation. As is true for euthyroid sick syndrome, thyroid hormone replacement is not beneficial in anorexia nervous and is actually contraindicated. Only when a high TSH and a low T4 persist after several weeks of weight restoration should thyroid hormone replacement be prescribed. It is important to avoid unnecessary and potentially dangerous thyroid hormone for low-weight patients with AN when the thyroid function tests are most consistent with euthyroid sick syndrome, as these typically normalize with just nutritional rehabilitation. The risk of unnecessary thyroid hormone therapy is especially prominent because of its deleterious effect on bone mineral density in a population of patients who are already at risk for severe osteoporosis (Abrahamsen et al., 2015), and excessive thyroid hormone will impede weight gain. Thyroid hormone should be dosed based on current BMI at a dose of 1.4 mcg/kg. For a summary of general endocrine changes in anorexia nervosa, see table 9.1.

Subclinical hypothyroidism is a related condition wherein the TSH levels are slightly greater than 4.5 mIU/L with normal T4 levels. Antithyroid antibody tests may often be positive in subclinical hypothyroidism, but not in euthyroid sick syndrome. The treatment of subclinical hypothyroidism with low-dose levothyroxine is controversial (Burns et al., 2016). However, treating these patients with low-dose thyroid hormone does not appear to cause further weight loss if TSH levels are closely monitored to prevent overtreatment and when TSH is 10 mU/L or greater.

Glucose and Other Hormones

Dietary restriction accompanied by weight loss and excessive exercise leads to depletion of hepatic glycogen stores and disruption of hepatic gluconeogenesis, resulting in abnormalities of glucose metabolism. In early, milder cases of anorexia, hypoglycemia rarely causes symptoms. In contrast, individuals with advanced AN and persistent severe hypoglycemia have a poor prognosis; severe hypoglycemia has been associated with sudden death because it indicates liver failure and a depletion

TABLE 9.1. Summary of endocrine changes in anorexia nervosa

Hormonal or metabolic change	Alteration	Cause
Cortisol (hypothalamic-pituitary-adrenal axis)	Increased plasma	Increased corticotrophin-releasing hormone from hypothalamus; decreased metabolic clearance; normal pituitary response
Growth hormone	Decreased IGF-1; increased or normal fasting GH; decreased serum GHBP	Down-regulation of GH receptor by nutritional deprivation
Thyroid hormones	Euthyroid sick syndrome: 1. Low or low normal T4 and T3 2. Normal TSH 3. Increased reverse T3	T4 decreased due to conversion to inactive reverse T3
Glucose	Fasting hypoglycemia present with severe anorexia nervosa	Depleted liver glycogen stores; disrupted gluconeogenesis
Serum leptin	Decreased	Unknown; possibly low-fat mass
Cholesterol (total)	May be increased due to increased HDL	Possible changes in thyroid, estrogen, and glucocorticoids
Gonadal hormones	Decreased estrogen in females; decreased testosterone in males	Central hypothalamic hypogonadism resulting from low weight associated with inappropriately low pituitary LH and FSH levels

of substrate to maintain safe blood glucose levels (Kohn, Madden, and Clarke, 2011). In the presence of hypoglycemia, insulin levels are appropriately decreased, but insulin sensitivity may paradoxically become increased in individuals with advanced eating disorders. Documented hypoglycemia should simply imply an urgent need for weight restoration and close monitoring of blood glucose levels at an inpatient level of care, especially since patients with anorexia nervosa may not manifest typical neuroglycopenia symptoms when their sugar is in the hypoglycemic range.

Adrenal androgens in women with eating disorders are usually normal to low normal, while men with eating disorders usually have low testosterone levels, similar to the low estrogen levels consistently noted in women with AN. These low sex hormone levels probably account for

the low sexual functionality found in those with anorexia nervosa (Gonidakis, Kravvariti, and Varsou, 2015). Low testosterone levels may be associated with the osteoporosis seen in males with AN. Although there is no definite therapeutic role for testosterone replacement therapy for their bone disease (Miller, Grieco, and Klibanski, 2005), it is reasonable to offer testosterone replacement to male patients older than 20 as it may help attenuate their loss of bone mineral density. Serum prolactin is generally normal in both men and women.

Leptin is a hormone that is secreted by adipose tissue and normally plays a role in satiety and energy balance as an anorexigenic adipokine; that is, it normally inhibits appetite. Leptin levels are appropriately reduced in patients with anorexia and correlate well with weight, percentage of body fat, and IGF-1 (Mehler, Eckel, and Donahoo, 1999). The exact clinical significance of this is unknown, but as weight approaches ideal body weight, leptin levels revert to normal (Modan-Moses et al., 2007). It is slowly becoming popular for some patients with anorexia nervosa to request that their provider check their serum leptin level to demonstrate return of health and no further need for weight restoration. Ghrelin, produced by the stomach and also known as orexigenic gut peptide, normally increases appetite. It is increased in anorexia nervosa (Dostálová and Haluzík, 2009). Although some reports suggest abnormalities of vasopressin secretion, polyuria, and diabetes insipidus in AN, defined by hypernatremia, overall these are uncommon (Rosen et al., 2019).

As many as 50% of patients with anorexia have been reported to have hypercholesterolemia (Ohwada et al., 2006), often due to a high level of cardioprotective HDL without significant elevations in LDL levels (Mehler, Lezotte, and Eckel, 1998). The reason for the elevated HDL is not well understood, but presumably it reflects excessive exercise and weight loss. Abnormalities in estrogen, thyroid hormone, and glucocorticoids may explain the mildly elevated LDL-lipid levels, which should not be treated with lipid-lowering agents during the active phase of anorexia nervosa unless there is clear evidence of a significant elevated risk of vascular disease. Vitamin D levels are commonly found to be low in AN and should be orally supplemented if less than 20 IU/mL (Veronese et al., 2015). Best to confirm with a serum 25-hydroxy vitamin D level.

Symptoms and Signs of Endocrine Disorders

Abnormalities found on physical examination that might suggest endocrine dysfunction in patients with eating disorders include hypotension (low blood pressure), cold intolerance, and hypothermia. Only rarely will the hypotension represent adrenal insufficiency. The characteristic findings of primary adrenal insufficiency (decreased serum sodium levels, increased serum potassium levels, and hyperpigmentation) are generally absent. Cold intolerance, low heart rate, and hypothermia are all consistent with hypothyroidism but are more likely attributable to the malnourished state of anorexia nervosa. Other findings that might suggest endocrine dysfunction are hair loss, easy bruisability, lightheadedness, and dizziness. It is not surprising that the patient who has AN, especially occult anorexia with denial of dieting, may be referred to an endocrinologist for initial evaluation before the diagnosis of anorexia nervosa is definitively made.

Despite the high serum and urine cortisol levels that may accompany AN, the striae, hyperglycemia, hypertension, and skin atrophy seen with cortisol excess (Cushing's syndrome) are not present. The lack of symptoms related to cortisol excess suggests that tissues may be resistant to glucocorticoids. It is widely assumed that the hypercortisolism contributes to the osteopenia of anorexia, although patients with eating disorders do not have elevated urine calcium excretion, which should occur in the presence of hypercortisolemia.

Endocrine Testing

At times, a battery of endocrine tests may be necessary to define an accurate diagnosis (table 9.2). On initial evaluation, thyroid function should be tested and is best assessed by measuring free T4 and TSH levels. It is not necessary to measure T3 and reverse T3 to confirm the diagnosis of euthyroid sick syndrome, because the diagnosis can be made clinically, and neither measurement will affect therapy. If the TSH level is normal, thyroid hormone replacement is not indicated even when T4 is low. Very low or suppressed levels of TSH suggest

TABLE 9.2. Suggested hormone and hormone-related metabolic testing for patients with eating disorders

1. TSH and free T4 (anorexia nervosa)
2. Fasting glucose
3. Testosterone (males with anorexia nervosa)

hyperthyroidism, hypopituitarism, or the effects of malnutrition and need to be interpreted carefully, in conjunction with measurements of free T4 and T3, in the setting of the clinical picture. A patient with anorexia nervosa who has a low free T4 and an elevated TSH, however, has primary hypothyroidism and should receive low doses of thyroid hormone, with careful monitoring in view of the low body weight and likely need for smaller doses of thyroid replacement. A dose of 0.025–0.05 mg of levothyroxine is a good starting point. TSH level should be rechecked 2 months after therapy begins, with a goal level of 0.4–4.5 mg/dL. Weight gain should prompt sooner follow-up as the dosage requirements may change.

There is little clinical advantage in measuring GH or IGF-1 levels in individuals with anorexia nervosa, as replacement therapy is not likely to be of therapeutic utility. Typically, AN is associated with a state of growth hormone resistance. Stimulation tests of pituitary function are also rarely helpful. For an individual who has anorexia and an expected low T4 but an unexpected low TSH level and symptoms of pituitary dysfunction, a thyroid-releasing factor (TRF) stimulation test could be employed. This will be difficult to interpret, however, as malnutrition and depressive illness may affect TSH responsiveness to TRF. Referral to an endocrinologist may be prudent for some of these more complicated scenarios, but in general, extensive endocrine testing is rarely necessary. Finally, oxytocin levels, a hormone released by the posterior pituitary, are low in anorexia nervosa. Information on its exact significance is currently evolving (Giel, Zipfel, and Hallschmid, 2018).

Summary

People who have eating disorders may present to endocrinologists with symptoms caused by their eating disorder but suggestive of possible adrenal, pituitary, thyroid, pancreatic, or reproductive hormone abnormalities. Most endocrine changes are secondary to weight loss, nutritional alteration, and purging behavior. It is best to diagnose an eating disorder by history and mental status examination, not by first ruling out all possible medical causes. Most of the endocrine complications of eating disorders revert to normal with early diagnosis and successful treatment of the eating disorder, but normalization may lag behind weight restoration. Consider treating hypothyroidism in patients with anorexia nervosa only if their free or total T4 is decreased *and* TSH is also increased. Further evaluation of pituitary function should also be considered if atypical, non-starvation-related endocrine findings are present.

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10

The Dual Diagnosis of Eating Disorder and Diabetes Mellitus

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COMMON QUESTIONS

Is diabetes mellitus type 1 a risk factor for the development of eating disorders?

What is the definition of the dual diagnosis of eating disorder and type 1 diabetes mellitus?

How does insulin omission or underdosing affect weight?

How can patients with diabetes disable their insulin? Why would they do so?

To what extent are patients with eating pathologies and type 1 diabetes typically aware of the diabetes complications that can arise from insulin manipulation?

When do patients diagnosed with diabetes make the connection that insulin deficiency leads to weight loss?

How common is intentional insulin manipulation by individuals with type 1 diabetes?

Is insulin omission or underdosing an effective way of inducing weight loss?

Why is a comprehensive medical and psychiatric assessment imperative for safe management in cases of combined eating pathology and type 1 diabetes?

What do *permissive hyperglycemia* and *relative hypoglycemia* mean?

Why is an "assume then resume" stance in the care of patients with eating pathologies and type 1 diabetes a core principle of treatment?

How does eating pathology combined with type 1 diabetes compare with other eating disorders with respect to mortality risk?

Case Study 1

M.M. was a 23-year-old female with a 15-year history of type 1 diabetes mellitus (DMT1). At age 17, after using insulin injections for years, she started using a continuous glucose monitor and had an insulin pump inserted. She was treated for her eating disorder and concomitant diabetes at the inpatient and residential levels of care starting at age 15. She was also hospitalized for diabetic ketoacidosis on several occasions. During her most recent hospitalization, her hemoglobin A1c level was noted to be markedly elevated at 12%, she was 63% of ideal body weight, and liver enzymes were markedly elevated. After medical stabilization, she shared with the treatment team that she had become frustrated with her weight gain and had begun to manipulate her pump settings to reduce her insulin infusions, restricted her oral intake, and was exercising daily, with resultant hyperglycemia and associated osmotic diuresis. She required inpatient care for 6 weeks before she was stable enough to transfer to an eating disorder unit with a team who was well versed in treating both eating disorders and diabetes.

Case Study 2

A.R. was a 15-year-old male with history of DMT1 diagnosed at age 5. His glycemia was well controlled from the onset of exogenous insulin administration via SQ injections of short-acting and long-acting insulins by parents, with Hbg A1c's stable in the 6%–7% range. At age 13 he started eighth grade, and the family felt that an insulin pump and continuous glucose monitor (CGM) would best address his diabetes management. This was instituted, and for a short period his Hgb A1c increased to the 8%–9% range during the transition. Family and the endocrine team were concerned, but there were no other noticeable changes in his diabetes management. Things normalized

and Hgb A1c's improved, but on his 14th birthday he started to express a desire to be healthier, eat healthier, and be more physically active, in part to further improve his blood glucose. His parents noticed gradual weight loss over the subsequent 2–3 months but felt that the loss of weight was beneficial to his overall health. On an annual checkup as he turned 15, his weight was down by 20 pounds, and his Hgb A1c was markedly elevated at 14%. His insulin pump records showed adequate administration of insulin. When he was admitted for further weight loss and severe bradycardia, with a heart rate of 40 beats per minute, he acknowledged that he had heated the insulin in the microwave before loading his insulin pump, intending to inactivate the insulin.

Background

From a historical perspective, there is no reason to believe that eating disorders and diabetes mellitus would coincide. Both disease entities have a long and rich clinical history. Diabetes was first described by Aretaeus de Cappadocia in the first century AD as "the melting down of flesh and limbs into the urine," conveying the weight loss associated with insulin deficiency. Nearly two millennia later, in 1921, Frederick Banting discovered insulin, and in 1959, the two types of diabetes—insulin-deficient, or type 1, and insulin-resistant, or type 2—were described. In 1963, the insulin pump was invented by Arnold Kadish.

For eating disorders, the first cases that can be identified forensically date back to the 1300s; Catherine of Sienna (1347–1380) may have suffered from anorexia nervosa. In 1689, Richard Morton described the illness as "phthisis nervosa." In 1873, Charles Lasègue and William Gull nearly simultaneously coined the term *anorexia nervosa*. It was not until 1979 that Gerald Russell described bulimia nervosa. The incidence and prevalence of both types of diabetes and of eating disorders have increased worldwide over several decades, but not until recent decades did the complicated relationship between these two illnesses become better understood. Diabetes and eating disorders often coexist. Eating disorders are more common in people with type 1 diabetes than in controls

(7% vs. 2.8%; Young et al., 2013). The co-occurrence signifies more complications and worse outcomes for both conditions compared to those with either disorder alone. Diabetes in people with eating disorders is associated with higher A1c levels, as well as more frequent and longer hospitalizations. Also, autoimmune disorders may be more common in individuals with eating disorders. Individuals with type 1 diabetes can manipulate their insulin injections, either to induce weight loss or to avoid weight gain after eating excessively or binge eating, by "purging" the calories through a self-induced insulin deficiency. The lay media have dubbed this syndrome *diabulimia* (Davidson, 2014). A better and more descriptive term is "the dual diagnosis of eating disorder and diabetes mellitus type 1" (ED-DMT1). The other possible association is that of an eating pathology and type 2 diabetes (ED-DMT2), in which individuals gain weight related to excessive eating or binge eating and thus increase their risk of developing insulin resistance and, potentially, type 2 diabetes.

Thus, the relationship between diabetes and eating pathology is different for the two types of diabetes. Type 1 diabetes is a risk factor for the development of eating disorders. In contrast, any eating disorder that has associated weight gain is a risk factor for development of type 2 diabetes (Peterson, Fischer, and Young-Hyman, 2015). Still, in some individuals with type 1 or type 2 diabetes and an eating disorder, these two entities coexist but do not interact, as they do in ED-DMT1 or ED-DMT2. To clarify, a case when the two illnesses coexist but the diabetes is not used to intentionally serve the eating disorder mentality is quite different from the case in which the diabetes is intentionally manipulated to serve the eating disorder. The differentiation is critical for understanding and appropriately addressing the comorbidity. This chapter focuses on ED-DMT1.

Definition

Diabetes is diagnosed by the following means (table 10.1). The A1c test measures the average blood sugar for the previous 2 to 3 months. The fasting plasma glucose (FPG) test checks the fasting blood sugar levels

TABLE 10.1. Diabetes diagnosis

Result	A1c	Fasting plasma glucose (FPG)	Oral glucose tolerance (OGT)	Random plasma glucose
Normal	less than 5.7%	less than 100 mg/dl	less than 140 mg/dl	—
Prediabetes	5.7% to 6.4%	100 mg/dl to 125 mg/dl	140 mg/dl to 199 mg/dl	—
Diabetes	6.5% or higher	126 mg/dl or higher	200 mg/dl or higher	200mg/dl or higher

Source: American Diabetes Association, 2020.

after not having anything to eat or drink (except water) for at least 8 hours before the test. The oral glucose tolerance (OGT) test is a two-hour test that checks blood sugar levels before and two hours after consuming a beverage with a specific amount of carbohydrates. The random plasma glucose test is a blood check at any time of the day (no fasting required) when diabetes symptoms are present.

When individuals with type 1 diabetes develop an eating disorder, they may intentionally misuse insulin for the purpose of weight control. This may happen by strategically delaying the dosing, by underdosing, or by omitting insulin altogether. These patients may (1) not use insulin adequately and restrict their caloric intake, or (2) eat normally, or (3) not cover their intake with insulin for binge episodes. In the first two instances, weight loss is likely to ensue. In the third instance, there may be weight loss, weight maintenance, or weight gain. The intent of the patient, however, is to avoid weight gain or to induce weight loss (Colton, Rodin, et al., 2009).

Pathophysiology

When carbohydrates are normally ingested and digested, blood glucose rises. If insulin is available, glucose can be taken up by muscle and fat cells and utilized for energy. Insulin also reduces liver glucose production (gluconeogenesis and glycogenolysis) and ketogenesis as well as reducing fat breakdown (lipolysis). When insulin is deficient or absent,

the glucose cannot be transported across cell membranes, and thus the cells can become starved. Cellular starvation leads to weight loss. When blood glucose exceeds the renal absorptive threshold, glucose is spilled in the urine. The diuretic effect of glucosuria can lead to dehydration and weight loss. As cells starve, they resort to alternative metabolic pathways for energy, which can lead to production and accumulation of ketone bodies, ketonuria, and metabolic acidosis. This triggers a cascade of events that lead to short-term and long-term complications of insulin deficiency. The short-term complications include hyperglycemia, glucosuria, ketosis, ketonuria, dehydration, and metabolic acidosis. These may culminate in an episode of diabetic ketoacidosis, which can lead to shock, end-organ failure, cerebral edema, or death. The long-term complications are related to long-standing hyperglycemia and microvascular disease and include diabetic retinopathy, nephropathy, cardiomyopathy, and peripheral neuropathy. In addition, any individual who experiences significant weight loss can suffer from the medical complications described in other chapters. In these cases, the compounding effects of the complications of the eating disorder related to weight loss and those of the poorly managed diabetes lead to increased medical morbidity and mortality risk. In addition, these patients can suffer from psychiatric comorbidities, including the known marked increased risk for suicide for patients with an eating disorder.

Mechanisms of Insulin Manipulation

The ways in which insulin can be manipulated are growing in sophistication, and there are some commonalties and some differences between manipulations by patients using subcutaneous insulin injections and those using an insulin pump. In either case, the intended consequence in the mindset of the patient is to influence body weight. The unintended consequences, the risks of which are often well understood by the patient, are the complications discussed later in this chapter. The notion that patients understand the risk involved is important, in that simply reeducating patients about the risks of poorly managed diabetes and the subsequent persistent hyperglycemia is unlikely to alter or stop their be-

haviors once they have crossed the line into a mental illness such as an eating disorder.

The hallmark of mental illness is a loss of perspective that often relates to risk-taking behaviors. From the point of view of the patient, these behaviors are a calculated risk in which the value of avoidance of weight gain or induction of weight loss outweighs the known risks of complications. This is clearly present in other eating disorders but is more difficult for clinicians and families to grasp in the context of ED-DMT1. Thus, the mechanisms of insulin manipulation are intended to achieve the weight control that patients seek, avoid complications to the extent possible, and mask the insulin misuse so that they are not suspected of it. Like patients with anorexia nervosa, those with ED-DMT1 often rationalize their risk taking by saying to themselves and others, "If I feel well, I must be well."

Dr. Morton, in 1689, noticed and referred to this notion as "an intellectual perversion," and he considered it pathognomonic of the "phthisis nervosa" he was describing. We do not believe today that this was meant to be judgmental in any way. In the language of the times, it implied a discrepancy between what a patient was likely to experience given their degree of medical debility and what they perceived and reported, perhaps more of a disconnect between their medical state and their perceived symptoms. The associated temperamental characteristics of an individual may lead to specific patterns of insulin manipulation. In today's terms this means a discrepancy between the lack of symptoms and the well-understood medical risk. For example, a patient who tends to consistently restrict calories may more consistently underdose insulin, whereas a patient with binge and purge behaviors may more impulsively not cover binges or may go into spells of insulin omission, yet at other times attempt to manage the diabetes well. As in other eating disorder–related behaviors, for some of these patients, this pattern may be lifelong, while for others it is variable or intermittent.

Management of type 1 diabetes mellitus involves blood glucose monitoring and insulin administration, although very low-weight patients with anorexia nervosa, even patients with DMT1, require very small

amounts of insulin early in the refeeding process. Blood glucose is most frequently measured by pricking the fingers to draw blood ("finger stick") and placing a drop of blood on the test strip of a glucometer. An alternative is to use a continuous glucose monitoring device. CGMs are devices worn by the individual at all times. They contain a sensor that continuously measures subcutaneous glucose levels, and they send data wirelessly to a display device. This offers real-time blood glucose monitoring. Alerts can be set for high or low glucose levels, and trends can be followed. CGMs decrease the need for finger-stick glucose monitoring. Studies have shown improvements in glycemic control in adults and children with type 1 diabetes using CGM (Mariani, Layden, and Aleppo, 2017). Newer devices link CGM directly to certain insulin pumps to create a closed-loop system.

Injectable insulin comes in short-acting and long-acting forms. The time to onset of action, peak of action, and duration of action for the different types of insulin currently available in the United States is listed in table 10.2. Some formulations combine short- and long-acting insulin to minimize the number of injections required, but these are not likely to be used by patients with ED-DMT1 because they would be more difficult to manipulate.

TABLE 10.2. Different types of insulin

Insulin type	Onset of action (hours)	Peak of action (hours)	Duration of action (hours)
Ultra-rapid-acting analog (faster aspart)	0.1–0.2	1–3	3–5
Rapid-acting analogs (aspart, glulisine, and lispro)	0.15–0.35	1–3	3–5
Regular/soluble (short acting)	0.5–1	2–4	5–8
NPH	2–4	4–12	12–24
Basal long-acting analogs			
Glargine	2–4	8–12	22–24
Detemir	1–2	4–7	20–24
Glargine U300	2–6	Minimal peak	30–36
Degludec	0.5–1.5	Minimal peak	>42

The short-acting insulin must be injected subcutaneously before each ingestion of carbohydrates, the dose calculated as number of insulin units per gram of carbohydrate to be ingested (e.g., 1 unit of insulin for every 12 grams of carbohydrate to be ingested, or 1:12 grams). This is referred to as the *coverage ratio*. The long-acting insulin is also injected subcutaneously, once or twice daily depending on the half-life of the formulation of insulin used, as number of units per injection (e.g., "22 units before dinner," called *long-acting insulin dose*). Some patients may divide the long-acting insulin into two daily doses for better blood glucose control. Short- and long-acting insulin can be combined in a syringe to decrease the number of injections required daily. Short-acting insulin is also used to correct for elevated blood glucose levels, as number of units of insulin per milligram per deciliter of measured blood glucose above a certain level of blood glucose (e.g., 1 unit of insulin for every 50 mg/dL of measured blood glucose above 150 mg/dL, or 1:50 above 150). This is referred to as the *correction factor*.

Insulin pumps use only short-acting insulin, which is drawn from a storage chamber, passes through a plastic tube into the pump itself, and then passes through another tube into the catheter placed under the skin. Newer technologies include tubeless insulin pumps called *pods*. The pod holds 72 hours of insulin and is placed on the body directly. The amount of insulin delivered is controlled through a wireless device. The patient programs the pump by dialing in a basal rate, which can be the same or different for different times throughout the day, and by dialing in a coverage ratio for grams of carbohydrate ingested at each meal or snack. If the blood sugar is elevated, then the pump, if provided with that information, suggests an amount of insulin to be infused according to the correction factor programmed into it. As with all systems, this is not foolproof, and patients could potentially manipulate by loading the pod with disabled insulin.

The basal rate is continuously delivered as programmed into the pump. In contrast, insulin for the coverage ratio for meals and snacks and for the correction factor when glucose is elevated is delivered only after ingestion of carbohydrates or when blood sugar is elevated and the hyperglycemia needs to be corrected. Subcutaneous insulin is most effective

and is usually administered before eating, given the pharmacokinetics of currently available insulin. This leads to better blood glucose management. On the other hand, once insulin is injected subcutaneously or delivered via the pump, undereating or refusal to eat can lead to dangerous hypoglycemia, especially in very low-weight patients with anorexia nervosa or ARFID. This is one reason that diabetics learn to eat and complete meals based on the external cues of insulin administration rather than internal cues of hunger and satiety. Also, low-weight patients with restricting eating disorders may lack the typical neuroglycopenic and physiologic symptoms found in a patient with DMT1 (like irritability, confusion, sweating, hunger, tachycardia). Thus, hypoglycemia may be even more sinister in these patients, and avoiding it is a primary goal. This insulin sensitivity associated with low-weight states improves with weight gain, and thus doses required may increase as weight improves.

In the case of ED-DMT1 in patients with a history of caloric restriction, we should consider the possibility that the patient may refuse a meal or snack after the insulin has been delivered. In this case, it is safer to administer the insulin after meals or snacks to avoid the risk of hypoglycemia until the patient has demonstrated that they are able and willing to complete each meal and snack.

Another aspect of insulin delivery is to rotate injection sites or catheter insertion sites. Areas that get overused are likely to develop inflammation and scarring, which may interfere with insulin absorption.

Finally, health care providers can better understand how well a patient's blood glucose has been managed by testing for glycosylated hemoglobin, or hemoglobin A1c (HbA1c, or simply A1c), which measures the percentage of total hemoglobin that is glycosylated. The higher the percentage of A1c, the higher the estimated average blood glucose (eAG) over the preceding 8–12 weeks. The different percentages of A1c correlate with published eAG values. For example, an A1c of 6% correlates with an eAG of 126 mg/dL, and an A1c of 10% correlates with an eAG of 240 mg/dL, in the preceding 2–3 months. Optimal A1c levels are 6.8%–7.5%.

Patients can use several different mechanisms of insulin manipulation in an attempt to subvert or achieve weight loss.

1. *Sustained insulin omission* for a person with type 1 diabetes, who has no residual endogenous insulin production, is incompatible with life. People with type 1 diabetes are taught this. So total omission is short lived (1–3 days). Intermittent omission of insulin is practiced by some patients, including those who monitor for ketonuria and resume insulin use when they notice increasing ketones in the urine. For those who use injectable insulin, this implies not injecting. For those who use an insulin pump, it implies wearing the pump and turning it off (there is really no reason for an insulin pump to be turned off under normal circumstances) or not wearing it at all. Either of these methods would be reflected in the pump record and therefore difficult to justify. Therefore, pump users are likely to resort to other means, such as wearing the pump but not inserting the catheter in the skin (thus wasting the insulin) or filling the insulin chamber with water or saline solution (in which case, pump records will reflect adequate insulin delivery).
2. *Underdosing insulin* can avoid some short-term complications and still lead to hyperglycemia and weight loss. Underdosing can look different from patient to patient and for those who use subcutaneous injections versus an insulin pump.
    a. Those who use subcutaneous insulin may use the prescribed amount of long-acting insulin once or twice daily but may either decrease the units of short-acting insulin injected per gram of carbohydrate to be ingested or use the correct amount of short-acting insulin but underdose or omit the long-acting insulin. Others might underdose insulin only on certain days (usually related to subjectively or objectively overeating, experiencing worse body image, not exercising, or experiencing greater emotional distress).
    b. Those who use an insulin pump may lower or omit the basal rate, lower the number of units per gram of carbohydrate to be ingested, or undercorrect for hyperglycemia.
    c. Another important mechanism of insulin underdosing is to use insulin as prescribed when eating normally but not use

insulin to cover binges. This becomes more of a purging mechanism—hence the popularization of the term *diabulimia*.

3. *Disabling insulin* is an effective way to conceal the insulin manipulation even from a close observer. This includes replacing the insulin in a vial or storage chamber of the pump with an inert liquid such as water or saline, or diluting the insulin with water or saline, or heating the insulin vial to render it ineffective (microwaving for just a few seconds can achieve this). All these maneuvers apply to both subcutaneous insulin and pump users. Insulin pens can also be heated to disable the insulin.
4. *Delays in insulin administration* is another way in which insulin availability can be altered as carbohydrates ingested are digested and cellular glucose uptake is impaired. Patients with type 1 diabetes generally understand that optimal dosing time for insulin is shortly before a meal and that delays in administration can serve as a weight control maneuver. Yet, patients often report that when they achieve a modest sense of weight control or weight loss, their tendency is to "speed it up" and advance the severity of insulin misuse.
5. *Impairing the absorption of insulin* is usually an effort by the patient to conceal insulin misuse. This may include injecting or inserting the catheter repeatedly in an indurated, scarred, or atrophied site on the skin (referred to as "dead spots"), which leads to malabsorption or erratic absorption of insulin. Another way is to "leak the shots" by pushing the plunger of the insulin syringe too early (the insulin is lost on its way to the subcutaneous space) or too late (the insulin is lost intradermally or on the skin). The magician's expression "the hand is quicker than the eye" is apt in this context: even an experienced observer may not perceive this.
6. *Falsifying blood glucose records* is another way in which a patient may attempt to conceal insulin manipulation. It is a more common practice for patients using finger sticks for blood glucose monitoring. Diluting blood samples or using nonblood substances with a predictable glucose content are the main

mechanisms. The diluting agents can be water, saline, or alcohol. Nonblood substances include diluted fruit juices, ketchup, and others. Glucometers use a control solution for calibration, and these solutions can be used to make blood glucose records look good. It is more difficult to falsify blood glucose records with CGM devices. Patients tend to suspend the use of the device in periods when they would like to conceal their blood glucose levels.

Etiopathogenesis

Published models for the etiopathogenesis of ED-DMT1 start from the stance that the management goals of type 1 diabetes mellitus imply that patients must constantly deal with efforts to maintain near-normal glycemia, must count carbohydrates to calculate coverage doses of short-acting insulin, and must practice portion control and variable degrees of dietary restraint. This can lead to frustration with less-than-optimal blood glucose control, given patients' baseline temperament and frustrations with weight gain, or a sense of nutritional deprivation, which can lead to subjective binges (they feel they overate) or objective binges (they actually overate). These factors can result in weight gain. In addition, negative feelings about body size and weight along with symptoms of depression can lead to further efforts at dietary restraint or purging behaviors and can thus contribute to the risk of weight gain or fears of weight gain. This sets the stage for the option of insulin manipulation to avoid weight gain or induce weight loss, despite the risks.

Like most gateway behaviors for eating disorders, such as dieting and exercise, once insulin manipulation is practiced, it can be intermittent or can cross a threshold into a compulsion. This may push the behaviors into greater frequency and intensity, despite the patient's understanding of the risks, and may foster the usually false belief that "I can keep it under check" or "the complications will not happen to me." This mentality represents not only a loss of perspective but also a fairly adolescent developmental stance of not internalizing the personal risk that may come with a behavior (Treasure et al., 2015).

Risk Factors

Overall, diabetes education by the American Diabetes Association (2020) has been highly successful because of its consistency and scalability. One drawback is that it emphasizes "control of the diabetes," and with that comes a focus on a patient's consistency, numbers, "good control," dietary restraint, adequate portioning, carbohydrate counting, insulin calculation, timely administration of insulin, eating in response to external cues such as having administered insulin, and exercise as an aid in blood glucose control. At the onset of type 1 diabetes mellitus, before it is adequately treated, patients usually experience increased hunger and thirst, excessive urination, and weight loss. With the implementation of insulin therapy, these symptoms subside, and the lost weight is regained. In a weight-vigilant cultural environment, even young patients tend not to miss this association: weight loss with insulin deficiency; weight gain with appropriate insulin administration. In addition, over time, patients with type 1 diabetes tend to have higher BMIs than their peers without diabetes (Meltzer et al., 2001), as well as experiencing more symptoms of depression (Colton, Olmsted, Daneman, and Rodin, 2013). Some studies have demonstrated twice the incidence of clinical depression among individuals with type 1 diabetes compared with the general population. Being educated about and living with type 1 diabetes, in a westernized cultural environment that values thinness, is a risk factor for the development of eating-related pathology, including an eating disorder. Insulin restriction and/or disordered eating have been documented for one in three females and one in six males with early onset type 1 diabetes after an average of 10 years of living with the diabetes (Wisting et al., 2013).

Some studies have found predictive factors for those with type 1 diabetes who are at higher risk of developing ED-DMT1. Some of these factors are related to clinical characteristics or behaviors that individuals may already show or may develop. These include disordered eating behaviors such as caloric restriction, binge eating, purging behaviors, or excessive exercise; a prior history of insulin restriction or manipulation; poor management of diabetes, especially if associated

with elevated A1c levels, repeated episodes of ketoacidosis, or presence of microvascular complications; and recurrent episodes of hypoglycemia. Other researchers have cited psychological or demographic features, including lower socioeconomic status; less cohesive family structure; perfectionism, with a focus on numbers; and a higher sense of emotional distress (Neumark-Sztainer et al., 2002). Nonetheless, *every* individual with type 1 diabetes is at risk for engaging in insulin manipulation.

Epidemiology

Several authors have looked at the incidence and prevalence of insulin manipulation in patients with type 1 diabetes. Some of their findings are as follows:

1. Partial and full syndrome eating disorders are twice as common in females with type 1 diabetes than in their peers without diabetes: full syndrome eating disorder in 10% of those with diabetes versus 4.5% in controls; partial syndrome eating disorder in 14% versus 8% in controls (Jones et al., 2000).
2. Increased risk of disturbed eating behaviors was found in preteen girls (ages 9–13) with type 1 diabetes: full or partial syndrome eating disorder in 8% of those with diabetes versus 1% in controls (Colton, Olmsted, Daneman, Rydall, et al., 2004).
3. Deliberate insulin omission is the most common form of purging for girls with type 1 diabetes; it starts early and increases through the teenage years: about 2% for preteens, 11%–15% for midteens, and 30%–39% for late teens and in the early adult years.
4. A study of disordered eating habits in 87 females with type 1 diabetes between the ages of 11 and 25, using a semistructured interview, found that about 25% had disordered eating or weight control behaviors, and 36% reported intentional insulin dose reduction or omission; there was a significant relationship among disordered eating habits, insulin misuse, and microvascular complications of diabetes (Peveler et al., 2005).

Epidemiologic conclusions can be summarized as follows: in females with type 1 diabetes, the risk of an eating disorder (by intentional insulin misuse) is greater than in peers without diabetes, and this can start at young ages, increasing with age until young adulthood. Therefore, individuals with type 1 diabetes should be considered at risk for the development of disordered eating behaviors and eating disorders. Although all this research has been done in female patients, the findings are likely to apply to patients of all genders. This implies that for health care professionals who oversee diabetic care in young patients, educating them with the goal of increasing their index of suspicion and promoting greater surveillance is imperative for early recognition and timely intervention in ED-DMT1.

Clinical Presentation

Children diagnosed with diabetes at a young age are likely to begin insulin therapy and blood glucose monitoring under parental or caretaker supervision. When diagnosed later in childhood, this is less likely to be the case. As young people mature while living with diabetes, there is a transfer of responsibility from caretaker to patient. For different families this happens at different ages and stages of developmental readiness and happens more gradually or more suddenly. Once young people have significant autonomy in the management of their type 1 diabetes, they have the option to intentionally misuse their insulin. When insulin misuse happens in such a manner as to avoid noticeable weight loss or measurable derangement in blood glucose control, the misuse may be difficult to detect. Adolescence is a time when patients with type 1 diabetes may experience less tight control of their glycemia, for reasons related to rebelliousness, competing demands, sense of invincibility, or resentment at having to live with diabetes as, with their growing autonomy, they revisit grief and loss issues. Adolescence and puberty in themselves may challenge the tight management of diabetes; these shifts in blood glucose management are expected and identifiable in the developmental context. Both psychological and biological factors may make diabetes management more difficult during adolescence. Nevertheless,

deteriorations in blood glucose management, especially if accompanied by weight changes, should raise the index of suspicion for the possibility of insulin manipulation. Significant weight loss in a patient with type 1 diabetes should be a concern unless actively sought with a health care provider's endorsement, and it should be considered abnormal.

Patients with type 1 diabetes who engage in insulin manipulation can present with weight loss, poor blood glucose control, elevated levels of A1c, repeated episodes of ketoacidosis, and early onset or rapid advancement of microvascular complications of diabetes (Criego et al., 2009). This is especially true when these changes are unexpected, given how long the diabetes has been diagnosed (Rydall et al., 1997). In addition, as with any other clinical presentations of an eating disorder, if these patients are verbalizing body dissatisfaction or concerns about body weight, size, or shape; making negative comments about their appearance; changing their eating or exercise habits; or are suspected of purging behaviors, the index of suspicion should be even higher.

There are some data to support the notion that once individuals with ED-DMT1 understand the impact of insulin manipulation on weight loss, they may stop using other purging modalities. In a 4-year follow-up study of girls between the ages of 12 and 18 years living with type 1 diabetes for an average duration of 7 years, at baseline, 38% were dieting, 45% were binge eating, 14% were engaging in insulin omission, and 8% were self-inducing vomiting. Four years later, 50% were dieting, 50% were binge eating, and 33% were omitting insulin. Of note, at the follow-up, none of the girls were self-inducing vomiting. This is probably due to the effectiveness of insulin manipulation in inducing weight loss in a group of patients whose focus on quick results may trump their concern for the risk of complications of their diabetes.

Other important clinical considerations include the increased diabetic morbidity and overall increased mortality faced by patients with ED-DMT1. Anorexia nervosa has the highest mortality rate of any psychiatric illness with the recent exception of opioid addiction. Its medical morbidity correlates with the degree of underweight and malnutrition. Some studies have looked at the high morbidity related to early onset or rapid advancement of diabetic complications in ED-DMT1, but none have

looked at the increased morbidity risk of the complications of malnutrition in this population. It is logical to assume that a patient who develops severe malnutrition related to an eating-related pathology and whose survival physiology also must contend with preexisting metabolic challenges such as diabetes is less resilient to the effects of malnutrition (Goebel-Fabbri, Fikkan, et al., 2008). Such data related to morbidity may be lacking, but not to mortality. The association of an eating disorder that leads to an underweight state (as in anorexia nervosa) and type 1 diabetes carries a heightened mortality risk that far surpasses that of diabetes alone or anorexia alone.

In a 1997 study that prospectively followed 91 patients with type 1 diabetes for 4 years, those who at baseline were identified as engaging in disturbed eating behaviors, including insulin manipulation, had three times the rate of retinopathy 4 years later (Rydall et al., 1997). These findings suggest that for patients with type 1 diabetes, the association with eating-disordered behaviors was more predictive of retinopathy than was the duration of diabetes. As to mortality, a 2002 publication reported increased risk for individuals with ED-DMT1 compared with those with diabetes alone or anorexia alone (Nielsen, 2002). This was a 10-year retrospective study based on a Scandinavian mortality registry per 1,000 person-years. There were 2.2 deaths per 1,000 person-years for those with type 1 diabetes alone, 7.3 deaths for those with anorexia nervosa alone, and 34.6 deaths for those with ED-DMT1. Thus, the mortality risk of ED-DMT1 surpasses that of anorexia nervosa alone. In an 11-year follow-up study of women with type 1 diabetes with a history of insulin restriction, this behavior conveyed a threefold increased risk of mortality after controlling for baseline age, BMI, and A1c. Higher rates of nephropathy and foot problems were seen as well (Goebel-Fabbri, Fikkan, et al., 2008). Another way of stating this is that ED-DMT1 is the eating disorder with the highest mortality rate. This makes a strong case for clinicians to maintain a high index of suspicion to facilitate early recognition. Screening in primary care settings and endocrinology clinics can be made more efficient by using a screening tool. The SCOFF questionnaire is a five-question tool validated to screen for eating disorders. It has been modified as the mSCOFF to screen for

ED-DMT1 (Zuijdwijk et al., 2014). Once the diagnosis is made, rapid and effective interventions are essential to improve mortality and morbidity risk. Finally, maintaining surveillance once patients have achieved recovery may prevent relapses, which carry a high cost for these patients.

Management

Because of the high associated morbidity and mortality risk, when a patient with type 1 diabetes mellitus is diagnosed with an eating disorder that includes intentional manipulation of insulin, initial hospitalization—medically, psychiatrically, or in an eating disorder treatment facility with expertise in the management of ED-DMT1—should be considered (Bermudez et al., 2009). This is especially true when the patient is in denial of the risks, has already experienced severe acute complications such as ketoacidosis, or has low motivation for change. A protracted assessment period or outpatient management efforts that prove unsuccessful may give the patient time to continue or increase these behaviors, and serious deterioration can follow.

Management should begin with a comprehensive assessment. This includes evaluation for medical risk related to diabetes (both short- and long-term complications discussed above) and the eating disorder (may include weight loss, malnutrition, and the consequences of purging behaviors by various modalities, binge eating, or excessive exercise). Assessment also includes evaluation for psychiatric risk involving self-injurious behaviors, suicidality, impulsivity and risk taking, psychosis, unstable mood, depression, anxiety, panic, obsessive compulsive features, and use/abuse of alcohol or drugs. It is also important to understand the patient's motivation for change since this may influence treatment decisions such as outpatient versus inpatient and required level of care. If a referral is made to a facility that the patient would need to travel to, then consideration should be given to the risk of travel: either stabilize the patient locally first (as long as experienced care is available) or plan for adequate, secure travel services, medically or psychiatrically. Finally, once the dual diagnosis of ED-DMT1 is made, the patient and

family should be informed and educated about the diagnosis, current state of understanding, risks, and recommendations for management. It is unfortunate when a lack of understanding leads patients and families to not fully grasp what this diagnosis entails and to continue to focus solely on managing the diabetes. Like other eating disorders, ED-DMT1 is a serious mental illness, and patients are best served when it is conceptualized as such and managed accordingly.

The principles that guide treatment and the goals of interventions are similar for different levels of severity at the time of presentation and at any level of care. The goals are to support and supervise the patient to the degree required to achieve the full interruption of insulin manipulation and all eating-disordered behaviors; to reestablish consistent, appropriate insulin use; to establish gradual and consistent weight recovery, when indicated, while avoiding refeeding complications; to establish weight maintenance when the patient is back to a normal weight; to normalize glycemia and A1c; and to ensure the patient demonstrates the ability and willingness to sustain those changes. If the patient has engaged in other purging behaviors, full interruption of those is imperative. When applicable, the clinician should diagnose and treat to remission any psychiatric comorbidities. Thus, the level of care required depends on the possibility for these changes to start right away in the least restrictive care environment, but clearly prioritizing effecting immediate behavior change.

The patient must have ongoing supervision and accountability. When the care is implemented in an outpatient setting, very close and consistent follow-up with accountability to a support system (usually family or a school health clinic) (Hasken et al., 2010) should be in place and closely monitored (Goebel-Fabbri, Uplinger, et al., 2009). This is true for an intensive outpatient program or partial hospitalization program, in which the program offers some structure, but the patient is responsible for self-monitoring outside the program. For patients with more severe findings on presentation, 24-hour care is indicated to start with, and the severity of blood sugar abnormalities, other medical findings, and psychiatric concerns will probably determine whether inpatient or residential care is needed. When 24-hour care is required, it is critical

that the program have expertise, or at least experience, both medically and psychiatrically, in managing ED-DMT1. This includes having protocols in place for consistent blood glucose monitoring and insulin administration and the interruption of other eating-disordered behaviors, as well as the ability to manage decompensation of diabetes, including hypoglycemia, hyperglycemia, fluid and acid-base abnormalities, and psychiatric decompensation. Residential treatment may be better than PHP or IOP level of care. In a 100-patient study from Toronto, less than 20% of outpatients had a good treatment outcome, and almost 40% had a poor outcome (Colton, Olmsted, Wong, et al., 2015). But in a residential treatment program, more favorable results were recorded (Dickens et al., 2015).

At times these patients may require tube feedings during refeeding because of oral intake restriction or a large meal plan needed during weight recovery. In such cases, glycemic control while on tube feedings can be challenging. In our opinion, if this is required, it should be offered by expert providers and in a specialized unit. When physiological and metabolic challenges of diabetes and recovery from a severely underweight state overlap, the complexity of how such an intervention may or may not be tolerated should be considered. Achievement of this requires a well-trained staff across all disciplines to be in place: professionals who have developed a significant level of comfort working with this specific pathology and have established clear pathways of communication and role definition.

At Eating Recovery Center, based in Denver, Colorado, a large residential eating disorders treatment center, we have developed a highly specialized treatment protocol for ED-DMT1. This protocol is guided by several principles. First, start from an "assume then resume" stance toward management of the diabetes. This means that the staff initially assumes responsibility for all aspects of diabetes care, then works with the patient so that they gradually resume the responsibility by demonstrating the ability and willingness to manage the diabetes effectively and consistently. This is usually achieved in three phases.

In phase 1, the care is assumed fully by the staff. In phase 2, the care is shared by staff and patient with strict staff supervision. In phase 3, the

patient resumes responsibility for care, with gradual weaning from staff supervision until they can successfully practice full self-care. Phases 1 and 2 often include removal of the insulin pump and continuous glucose monitor if one has been in use. Patients and families may become concerned about losing the pump and the CGM because they have learned that, when used adequately, a pump and CGM have advantages over finger sticks and subcutaneous insulin injections for glycemic control. The patient's ability to manipulate the pump even under close staff supervision, however, makes this too risky a practice. The CGM is often removed so that the patient does not have access to their glucose levels during early phases of care. Once the clinician carefully explains this and reassures them that interruption of pump and CGM use can be a temporary measure, patients and families are more likely to understand and accept this.

The second principle is consistency on the part of all staff interacting with the patient. In the treatment of eating-related pathology, often when patients feel able to get away with behaviors, they constantly test boundaries and attempt to practice such behaviors—and this is so for a patient with ED-DMT1. Having protocols in place that are consistently followed 24 hours per day, and by all staff, is essential. In addition, one must remain vigilant to detect new behaviors, since the patient may struggle with challenges often seen when individuals with eating disorders are in a group setting. These include contagion (they may learn things from each other), comparison (they notice how others manifest their eating pathology, or who is thinner, or who has lost weight more rapidly), and competition (they may think they have to "enhance their efforts" if they feel they are not "the sickest person there"). For patients with ED-DMT1, another aspect of their experience in treatment may be their sense of alienation from others with more traditional presentations of eating pathology. Comments such as "I do not restrict what I eat or make myself vomit so I am not like these other people" imply that a patient may be questioning whether they truly have an eating disorder. Educating patients about the similarities among eating-related pathologies, regardless of size or shape, is important. Helping patients connect through the similarities in their plights may help them integrate and make better use of the treatment experience.

The third principle is to approach improvement in glycemic control gradually. It is medically unwise to try to go rapidly from poor glycemic control to normalization of blood glucose. Such attempts may increase the risk of dangerous hypoglycemia, both subjectively and objectively, and may exacerbate the patient's focus on glucose management rather than addressing all aspects of the eating disorder. It is best to aim to improve glycemic control gradually, even if it means transiently accepting blood glucose levels higher than normal, which has been called "permissive hyperglycemia" (Brown and Mehler, 2014). To achieve this, it is important to discuss the principle with the patient and the family at admittance and again as needed thereafter, especially because many of these patients, even though aware of how they have elected to induce hyperglycemia, become obsessive about "excellent diabetes management" as they enter treatment. This is particularly true as patients experience weight recovery. Normoglycemia is difficult to achieve as a patient is gaining weight, amid the metabolic shift of refeeding and concomitant changes in insulin requirements. So, preparing patients to accept episodes of hyperglycemia and hypoglycemia is important. Also important is to monitor and record blood sugars in a format in which the trends are clear. In our experience, a graph format gives a clear visual clue of trends, which are helpful for all involved to track.

The fourth principle involves other ways to prepare the patient and family and set realistic expectations for the treatment experience. Preparation of the patient and family should also address the notion of *relative or subjective hypoglycemia*. Some of these patients have become accustomed to experiencing near constant hyperglycemia. As blood sugars trend downward, they may experience symptoms of hypoglycemia even at abnormally high blood glucose levels. These events should not be treated as true hypoglycemia because that would exacerbate the persistence of hyperglycemia. In addition, the patient needs to expect that with normalization of insulin management, some weight gain is likely. When the patient has become underweight and weight restoration is indicated, weight gain is usually difficult but expected. There are other circumstances in which a patient with ED-DMT1 may present at normal weight or overweight. In this case, accepting weight gain tends to become

more emotionally distressing. Patients who were significantly overweight when their eating disorder started and then lost weight may particularly fear losing control as they gain weight and return to their former overweight state. This scenario can cause significant distress and should be proactively addressed in treatment.

Our program does not view any foods as unacceptable for those with diabetes. So in preparing the patient and family, rather than focusing on abstinence from "bad foods," we focus on moderation and appropriate insulin management—including for foods with higher carbohydrate or caloric content.

One more issue to address is the role of exercise. For many individuals with type 1 diabetes, physical activity can support normalization of blood glucose. Some with *exercise sensitivity* may need to incorporate regular physical activity to manage their glycemia adequately. Physical activity may need to be curtailed or interrupted, however, for those who are underweight, including while they are refeeding. Patients with ED-DMT1 may feel that this could cause difficult-to-control blood sugars during the refeeding period. Although this may be partially true, the risk-benefit ratio of including physical activity during the refeeding and weight recovery phase of treatment must be taken into account. At least some patients will need to forgo physical activity until their cardiovascular and metabolic status improves.

Last, some degree of retraining in diabetes management is imperative, or at least verification that the patient can fully understand the principles of and implement appropriate diabetes self-care. Some patients are resistant to this; they may feel that they are the ones who have lived with their diabetes and better understand it. They sometimes become so accustomed to intentionally mismanaging their diabetes, however, that getting them reacquainted with what they were trained to do when they were first diagnosed with diabetes or started to use an insulin pump is by far in their best interest.

These management principles make it evident that a multidisciplinary team approach in which each discipline contributes its individual expertise is necessary. Understanding role definition and integration is critical to a well-functioning clinical team. The dietician on the team has a par-

ticularly critical and active role in preparing the meal plan, carbohydrate counting, tracking completion of the meal plan and the timely and appropriate use of insulin, and making sense of blood glucose trends in light of carbohydrate intake, level of activity, weight recovery trend or weight stability, and the patient's overall progress.

Eating Recovery Center's Management Approach

At Eating Recovery Center, we begin by carefully vetting potential admissions to the program. The intake team completes a comprehensive telephone assessment with the patient, family, or referring professional (as the situation requires or allows). The team also gathers pertinent medical records, including a detailed history of diabetes management and, specifically, recent endocrinology records, including A1c results and insulin orders. This information is reviewed by the admissions team, which may request further information, refer the patient to a more appropriate setting, or accept the patient for admission. When necessary, one or more members of the clinical team may communicate with the patient and/or family to clarify expectations and begin to shape the treatment plan. This rigorous process is necessary to ensure that the patient meets our admission criteria, that there are no exclusion criteria, and that our approach is a good fit for the needs of the patient.

On the day of admission, members of the clinical team work to understand the role of their particular discipline. After a nursing assessment that includes measuring vital signs, height, and weight and doing initial blood work, the clinical team meets with the patient and the family (if available) to complete the interdisciplinary admission assessment. Each discipline has a specific role in preparation for the admission and on the day of admission.

1. Role of the physician (pediatrician, family practitioner, internist)
    a. Identify medical complications of the diabetes or the eating disorder that require urgent attention, including vital sign abnormalities and severity of malnutrition. Some patients

may need to be diverted temporarily to a different facility for intensive care services, medical stabilization, or psychiatric stabilization.
   b. Review blood glucose trends and adjust insulin orders as necessary.
   c. Establish a plan for the management of hypoglycemia and hyperglycemia.
2. Role of the nurses
   a. Implement orders related to blood glucose testing, administration of insulin, and all other medications.
   b. Document blood glucose values and insulin administered.
   c. Manage hypoglycemia or hyperglycemia as per orders.
   d. Communicate to the rest of the team the patient's extent of compliance or resistance.
3. Role of the dietician
   a. Plan meals and snacks for the day of admission and complete carbohydrate counts for each meal and snack, using the *macronutrient spreadsheet*.
   b. Review available data to determine insulin-to-carbohydrate (I/C) ratios for meals and snacks.
   c. Discuss I/C ratios and plan for insulin administration with MD and RN (e.g., Is insulin to be administered before each meal and each snack or administered before each meal only but dosed to cover the subsequent snack also? Is insulin going to be administered before eating or after eating?).
   d. Initiate and update the *diabetes management sheet* and the glucose graph.
   e. Report on these values in team meetings.
4. Role of the psychiatrist
   a. Assess for psychiatric safety.
   b. Explore psychiatric comorbidity diagnoses and establish a plan for addressing them.
   c. Monitor the patient for safety as treatment progresses.
   d. Prescribe and adjust psychotropic medications as required.

5. Role of the psychotherapist
    a. Assess motivation for change.
    b. Establish a therapeutic alliance.
    c. Maintain communication with the family.
    d. Implement psychotherapeutic efforts.

All patients then progress through the steps of the Blood Glucose Management Level System, which has six levels. The first is the evaluation phase; all patients admitted start at this level. They can then progress through levels A through E.

1. *Evaluation phase*: Expectations and level system are clarified and communicated to the patient and family. All care is assumed by the staff.
2. *Level A*: At least 24–48 hours of collaboration from the patient is achieved; staff is doing all blood glucose checks, insulin dose calculations, and insulin administration. Insulin is administered after meals.
3. *Level B*: At least 48–72 hours of collaboration from the patient is achieved; staff is doing all blood glucose checks, insulin dose calculations, and insulin administration. Insulin is administered before meals because the patient has demonstrated that they can complete meals or use oral liquid supplementation if unable to fully complete the meal or snack.
4. *Level C*: Collaboration from the patient is ongoing, including improving trend in blood glucose levels and establishing weight recovery, if applicable. Blood glucose checks, carbohydrate count calculation, and insulin administration are done by the patient, with strict staff supervision. Insulin dose is still calculated by staff. There is no minimum or maximum time at this level. CGM may be considered for resumption at this level as the patient will now be learning their glucose levels and calculating insulin dosing.
5. *Level D*: The patient demonstrates the ability to adhere to the treatment plan, achieves blood glucose normalization, has

continuing or completed weight recovery, and demonstrates competency in counting carbohydrates, calculating insulin dose required, and administering insulin with limited (and gradually less strict) staff supervision. At this level, the patient may request to resume the use of an insulin pump.

6. *Level E*: The patient is managing all aspects of diabetes care independently and reports to the team, as they would to the endocrinology service as an outpatient.

Levels A to C are usually used in 24-hour care settings. Patients often step down to a partial hospitalization program level of care on level D, or tend to start at this level if admitted directly to the partial hospitalization program.

Some patients who have interrupted the use of an insulin pump may not wish to return to that right away, and we usually support this decision until they feel ready. Others may wish to resume using the pump sooner, and for those we offer a process of "applying" to resume pump use. The reason for the cautious approach is that the time when patients reach level D often coincides with their reaching a weight close to their target weight, and this tends to raise the distress that they experience—including worsening of negative body image. Thus, it is important not to resume pump use casually at a time when the lure of weight loss through insulin manipulation can pose a significant challenge to successful completion of treatment. When patients request to resume the pump, this is vetted by their clinical team and often by their peers, and finally the clinical team signs off on the application form approving the request. Heightened vigilance is suggested during this transition period. Core components of the discharge and aftercare plan are clear expectations about continued appropriate diabetes management, weight maintenance according to the team's recommendations, consistent completion of the prescribed meal plan, adherence to any prescribed medications including psychotropic medications, and close follow-up by a multidisciplinary team with expertise or experience in managing both eating disorders and diabetes.

Other Important Considerations

Varied clinical presentations of eating disorders are on the rise. Terms such as *orthorexia*, *pregorexia*, *brideorexia*, and *drunkorexia*, popularized by the lay press, describe new or atypical presentations of eating pathology that most clinicians see in their practices, and all can carry serious consequences both physically and psychologically. In addition, we are seeing "new" medical comorbidities and eating-related pathologies beyond ED-DMT1, and a high index of suspicion for these should be maintained. Some of these are also related to diabetes. These include a syndrome of self-imposed hypoglycemia in patients with type 1 diabetes, which involves intentional overuse of insulin sometimes coupled with excessive exercise to maintain a low body weight (Moosavi, Kreisman, and Hall, 2015). It turns out that chronic hypoglycemia also leads to weight loss or maintenance of an abnormally low body weight. We have also seen patients with type 1 diabetes who have used diabetic supplies or paraphernalia for self-injury (such as cutting themselves with lancets or stabbing themselves repeatedly with insulin needles).

Other "new" medical comorbidities involve not diabetes but other medical illness in which intentional manipulation of the treatment or the illness itself can lead to weight loss or avoidance of weight gain. Examples are:

1. Patients with hypothyroidism who take excessive doses of the prescribed thyroid hormone to induce a hyperthyroid state and thus lose weight or maintain a low body weight
2. Patient with hyperthyroidism who strategically underdose medications prescribed to down-regulate thyroid hormone production and thus "allow" the hyperthyroid state to persist for the purpose of weight reduction
3. Patients with inflammatory bowel disease who strategically underdose or omit their anti-inflammatory medications to allow enough bowel inflammation to impair absorption of nutrients and thus favor weight control
4. Patients with cystic fibrosis who underdose or omit their digestive enzymes to impair absorption of nutrients

5. Patients who do not have diabetes but abuse metformin for its known weight loss side effect and run the risk of dangerous lactic acidosis (Geer et al., 2019)

Given the readily available sharing of information on the internet, we will likely see more of these types of manipulation of medical diagnosis as part of the repertoire of manifestations of eating pathology.

Summary

Type 1 diabetes mellitus is a risk factor for the development of eating disorders. Insulin manipulation is prevalent in this patient population and warrants a high level of vigilance from any health care professional interacting with these patients. Intentional insulin manipulation should be considered in any individual with type 1 diabetes whose management of their diabetes deteriorates. Early recognition and timely intervention for patients with ED-DMT1 can prevent the high morbidity and mortality associated with this presentation of eating-related pathology. Care should be offered by clinicians with expertise or experience in the care of patients with ED-DMT1 and in settings where a multidisciplinary approach brings to bear the necessary medical, endocrine, psychological, dietetic, and psychiatric know-how to meet the needs and clinical complexity of these patients. There remains a need to reexamine how diabetes care education is imparted to see whether more creative approaches can be less triggering to vulnerable individuals with type 1 diabetes and thus at risk for insulin manipulation.

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11

Oral, Dental, Ear, and Eye Complications

Jeffrey Hollis, MD, and Philip S. Mehler, MD, FAED, CEDS

COMMON QUESTIONS

What dental findings are consistent with purging through self-induced vomiting?
What effect does bulimia nervosa have on the salivary glands? When does it usually occur?
What are the treatment options for swelling of the salivary glands (parotid hypertrophy)?
What should you recommend as oral care for a patient who has bulimia nervosa and engages in self-induced vomiting?
What is the significance of the amylase level in a patient who has bulimia and purges by means of self-induced vomiting?
What adverse effects do eating disorders have on the eyes and ears?

Case Study

S.B., a 25-year-old female, had a 5-year history of bulimia and admitted to intermittently vomiting five to seven times per day. She presented to her internist complaining of painless swelling on both sides of her face around the mandible. Although she was working with a therapist, she admitted that she had lapsed into a binge and purge episode of several days' duration. She stopped the behavior 4 days prior to the visit to her internist.

On physical examination, S.B. had obvious enlargement of her parotid glands bilaterally, which were soft and not very tender to palpation. Oral examination revealed erosion of the enamel on the lingual surfaces of her maxillary teeth and a reddened posterior pharynx. Laboratory blood work results demonstrated a total serum amylase of 220 U/L (normal 16–90). Her serum lipase, bicarbonate, and potassium levels were normal.

The internist recommended that S.B. suck on tart candies to induce excessive salivation, apply warm compresses to the swollen area multiple times per day, and increase her visits with her therapist. On a follow-up visit 2 weeks later, she reported no further purging episodes, and the parotid swelling had completely resolved.

Six months later, S.B. returned with more severe bilateral parotid swelling, as well as submandibular gland enlargement. This time, her parotid glands were tender and painful. She had not seen her therapist in more than 2 months and reported numerous self-induced vomiting episodes, but none in the last 2 weeks. Although she had tried warm compresses and tart candies, there was no decrease in the size of her glands. She was frustrated that the swelling seemed to develop a few days after she had decided to stop purging but was not present during her bouts of excessive vomiting.

Over the next 2 years, S.B. had four episodes of parotid swelling, which once required the use of pilocarpine (while she was hospitalized for electrolyte abnormalities and under close observation with cardiac telemetry). Generally, her parotid swelling resolved with conservative care. She had a job as a receptionist in a large firm and was concerned about the cosmetic appearance of the swollen glands. The option of having a surgical procedure to remove her parotid glands was raised, but given the potential morbidity and facial scarring, S.B. opted to continue to use the standard treatments of warm compresses, anti-inflammatory medication, and tart candies.

Background

Oral complications are common among individuals who have bulimia nervosa and can adversely affect quality of life (Chiba et al., 2019). These

complications are primarily related to the chronic regurgitation of acidic gastric contents with self-induced vomiting. Oral complications may be the first and only clue to an underlying eating disorder, and up to 25% of patients are first diagnosed with bulimia during a dental appointment (Douglas, 2015). The main oral-facial complications of self-induced vomiting include angular cheilosis (sores in the angles of the lips), loss of enamel and dentin on the lingual surface of the teeth (perimylolysis), pharyngeal soreness, gingivitis, and hypertrophy of the salivary glands (table 11.1). Focusing the history and physical examination on these possible complications can alert a health care provider to an underlying eating disorder. Dentists and dental hygienists can therefore play a crucial role in the secondary prevention of bulimia nervosa, through timely identification of the oral and physical manifestations of this disorder and referral of the individual to an eating disorder professional. Early identification is especially useful for individuals with bulimia, who often maintain normal body weight and are better able to hide the severity of their illness than individuals who have anorexia. Subconjunctival hemorrhages and patulous eustachian tube dysfunction are eye- and ear-specific complications that may be noted in patients with anorexia nervosa.

TABLE 11.1. Oral complications associated with bulimia nervosa and self-induced vomiting

Oral finding	History and physical findings	Proportion of patients
Cheilosis	Erythematous, dry, painful fissures at angles of lips	Uncommon (<10%)
Erosion of the enamel and dentin of teeth (perimylolysis)	Erosion primarily of the lingual and occlusal surfaces of the anterior maxillary teeth; sensitivity to hot and cold foods	Up to 40%
Gingivitis	Painful, erythematous gums	Uncommon (<10%)
Salivary gland enlargement (parotid hypertrophy)	Bilateral enlargement of the parotid (less often submandibular) glands; generally painless	10%–66%
Sialadenosis	Painful parotid gland swelling	Uncommon (<10%)
Hyperamylasemia	None	10%–66% of patients with sialadenosis

Oral and Dental Complications

Oral Soft Tissue Findings

Oral complications among patients with eating disorders may be related to dry mouth, or xerostomia; these complications include gingivitis, tooth decay, cheilosis, and cracked lips. Dehydration and medications such as antidepressants may contribute to the xerostomia. Patients with eating disorders report a significantly increased frequency of dry or cracked lips compared with controls (Johansson et al., 2012). Angular cheilosis is a form of stomatitis (inflammation of the mucous membranes of the mouth) seen in patients with eating disorders, characterized by pallor and maceration of the mucosal lining at the corners of the mouth. The lesions are due to the direct caustic effects of the acidic content of the vomitus. In severe cases, linear fissures can leave scars after healing. The lesions are typically painful and should be distinguished from herpes simplex vesicles, which are more often unilateral and in the middle of the lips, away from the corners of the mouth. Herpetic lesions are also different in that they have a prodrome of mild pain and itching a few days before the lesions appear. Cheilosis may represent an underlying deficiency in B vitamins, zinc, or iron. It may be prudent to check serum levels of these nutrients and to prescribe supplements if levels prove to be low (Park, Brodell, and Helms, 2011). Topical petroleum jelly following gentle washing with warm water can assist in healing these sores. Keeping the area clean and dry is the main way to hasten resolution (table 11.2). When examining the oropharynx of patients with bulimia, the clinician may note traumatic lesions and, in severe cases, ulcerations on the palate and oropharynx resulting from the repeated use of a finger or other object to induce vomiting (Douglas, 2015; Solomon et al., 2007).

Tooth Erosion (Perimylolysis)

Perimylolysis, or erosion of the enamel and dentin of the teeth, is the most specific manifestation of bulimia nervosa. Perimylolysis has been reported to occur in up to two-thirds of people who have bulimia and purge by means of self-induced vomiting (Conviser, Fisher, and Mitchel,

TABLE 11.2. Treatment of oral complications associated with bulimia nervosa

Oral finding	Treatment
Cheilosis	B complex multivitamins; topical petroleum jelly
Enamel erosion (perimylolysis)	Dental consultation; rinsing of mouth with baking soda solution (1 teaspoon in 1 quart of water); consider brushing gently after vomiting
Gingivitis	Mouth rinses; flossing
Salivary gland enlargement (sialadenosis)	Hot compresses, sialagogues (tart candies), and pilocarpine in recalcitrant cases

Note: The primary goal of treatment is the cessation of self-induced vomiting.

2014; Pallier et al., 2019). Chronic contact with gastric contents leads to loss of superficial tooth enamel and the dentin that lies underneath the enamel. The average pH of vomitus is 3.8, which is quite acidic, and this may contribute to tooth erosion. Proteolytic enzymes present in saliva after purging, such as pepsin and trypsin, may also play a role in perimylolysis (Schlueter et al., 2012). Finally, consumption of acidic or sweetened foods has also been linked to more severe perimylolysis (Otsu et al., 2014). The dental areas affected first are primarily the lingual surfaces of the upper anterior teeth, followed by the palatal and posterior occlusal surfaces of the maxillary teeth. By contrast, other causes of enamel erosion, such as eating a highly acidic diet (e.g., lemons), first affect the facial teeth surfaces and spare the lingual surfaces. Early perimylolysis is characterized by a smooth, glossy appearance (Aranha, Eduardo, and Cordas, 2008). As erosion continues, the teeth appear shortened in length and have dull enamel surfaces with irregular incisal edges. In severe cases, there may be loss of enamel on incisal edges of the anterior teeth and, finally, involvement of the posterior teeth. If a tooth with a previous restoration is involved, a characteristic prominence is created when loss of enamel leads to projection of the amalgam restoration above the tooth's surface. Additionally, erosion of some dental aesthetic ceramics occurs when exposed to gastric acid (Alnasser et. al., 2019).

If destruction of the enamel is visible, the care provider can assume that the patient has experienced at least 6 months to 2 years of regular and excessive vomiting. Other factors that influence the severity and

rate of enamel loss include the types of food consumed, the quality of tooth structure, and overall oral hygiene, including oral care after purging episodes. Patients with severe cases will complain of excessive tooth sensitivity to hot and cold food due to exposed dentin, along with chipping of the edges of the teeth.

Cavities (Caries)

Bulimia does not put individuals at increased risk of dental caries (Johansson et al., 2012). It may be that people who binge on sweet, high-carbohydrate foods are more likely to develop caries. On the other hand, some people with eating disorders are fastidious in their dental care and have healthy teeth. Other factors are important in the development of caries, such as the use of fluoridated water, oral hygiene, type of diet, and genetic predisposition.

Gingivitis

Gum disease (gingivitis) is a result of chronic irritation from the low-pH (acidic) gastric contents and is associated with pain and erythematous gums. Gingivitis has also been attributed to xerostomia. Xerostomia can occur as a result of dehydration due to chronic purging, reduced salivary flow rate (observed in both restricting and binge-purge subtypes of anorexia), or as a side effect of certain medications (Steinberg, 2014; Paszynska et al., 2017). Although patients with eating disorders appear to have an increased incidence of gingivitis, it is not clear whether they are predisposed to progressive periodontitis. Sometimes gingivitis is associated with throat erythema and soreness, which can also be a manifestation of gastroesophageal reflux disease (discussed in chapter 6).

Dental Hygiene

Prevention of perimylolysis, dental caries, and gingivitis among patients with bulimia requires meticulous oral hygiene and dental care. Thus, it is extremely important that patients with oral complications of eating

disorders establish care with a knowledgeable and understanding dentist. The most beneficial means of preventing and treating dental complications is to stop the self-induced vomiting. Because success in this effort takes time, care providers need to counsel patients on what to do if they are still engaging in self-induced vomiting. Typically, it is recommended that patients undergo fluoride treatments, both in the dentist's office and at home. Artificial saliva can be used to mediate the effects of xerostomia. Rinsing one's mouth with water or a slightly basic solution containing baking soda or sodium fluoride after vomiting may help to neutralize acid and protect tooth surfaces (Steinberg, 2014). A mouth guard for use during vomiting episodes may also be considered to protect the teeth from vomitus. The optimal tooth-brushing practice for patients with bulimia remains controversial, as there is concern that the abrasive mechanical action of brushing after vomiting may accelerate enamel erosion (Schlueter et al., 2012). Most clinicians recommend gentle horizontal brushing, given the lack of evidence that tooth brushing itself contributes to erosion. Crowns or restorative dental work are typically delayed until bulimic behaviors are controlled and recovery is imminent. Palliative treatments for pain or temporary cosmetic procedures may be considered for patients who are not yet in recovery from their eating disorder (Steinberg, 2014).

Enlargement of Salivary Glands

Parotid hypertrophy is common in patients with bulimia nervosa, occurring in up to 66% of patients whose purging behavior is self-induced vomiting (Mandel and Surattanont, 2002). The diagnosis of bulimia should be considered for any young person with persistent bilateral enlargement of the parotid glands without another apparent cause. Other causes of salivary gland enlargement are obstruction of the ducts, local infection, Sjögren's syndrome, diabetes mellitus, alcoholism, cirrhosis, hypothyroidism, and hypovitaminosis A. In patients who have bulimia nervosa, the swelling is usually painless, bilateral, and readily apparent on physical examination, with the parotid salivary glands being enlarged to two to five times their normal size. The severity of parotid hypertrophy

is related to the frequency of vomiting. Further, patients with parotid swelling seem to be more likely to also have enamel erosion. The submandibular glands, too, are occasionally involved. In some cases, acute, severe swelling begins 2–3 days *after* a purging episode, which can be disconcerting for the patient with bulimia nervosa who has finally decided to cease purging. An acute increase in parotid gland size, which is often painful, is termed *sialadenosis* (Gaudiani and Mehler, 2016). It should be noted that sialadenosis is similarly seen in patients with the binge-purge subtype of anorexia nervosa who engage in self-induced vomiting.

The exact pathophysiology of parotid hypertrophy is undetermined; however, one theory implicates dysregulation of the autonomic nervous system, which controls salivary gland function as neurovegetative/hormonal dysregulation. It has been hypothesized that intracellular secretory zymogen granules begin to accumulate as a result of dysregulation of the acinar cells' sympathetic nerve supply, thus leading to the enlargement of individual parenchymal cells (Mandel, 2014). Biopsies of the parotid glands often show only normal cells but may demonstrate increased acinar size and increased numbers of secretion granules without inflammatory cells (Aframian, 2005).

Autonomic dysregulation may occur as a result of chronic regurgitation of stomach contents, leading to increased cholinergic nerve stimulus. Alternatively, the stimulation of lingual taste receptors by pancreatic proteolytic enzymes, which come in contact with the oral mucosa during vomiting, may lead to autonomic dysregulation. Another proposed hypothesis is that of *work hypertrophy*, in which the glands enlarge because of their extra work in providing saliva during normal eating as well as during the regurgitation of gastric contents.

Treatment of sialadenosis depends on the severity of the swelling and the patient's concern over their cosmetic appearance. Abstinence from vomiting alone will lead to the resolution of swelling in most cases. In addition, hot compresses, nonsteroidal anti-inflammatory medication (such as ibuprofen), and sialagogues (tart candies) can aid in the resolution of swelling. More severe cases have been successfully treated with pilocarpine tablets (5.0 mg three times per day). Pilocarpine is a para-

sympathomimetic agent that works by causing increased salivation and resultant decompression of the glands (Mehler and Wallace, 1993). The main side effects of pilocarpine are transient blurring of the vision, lacrimation, sweating, a lowering of heart rate, and dizziness due to decreased blood pressure. Patients with significant cardiovascular disease may be unable to compensate for the transient changes in blood pressure and pulse induced by pilocarpine; therefore this medication should initially be administered to patients only under close observation and perhaps with cardiac telemetry.

Recalcitrant cases of parotid hypertrophy have been treated with the surgical procedure of parotidectomy. Unfortunately, parotidectomy can lead to facial scarring and disfigurement, which can be particularly troublesome to these patients, who are already extremely fixated on their self-image. Additional morbidity from the surgery, such as dry mouth and salivary fistulae, can also be problematic. Finally, patients who revert to self-induced vomiting after parotidectomy risk recurrence of salivary gland enlargement (Wilson and Price, 2003). Another potential treatment for refractory rare cases is the use of sialendoscopy as a technique to visualize and treat salivary ductal system pathologies.

Hyperamylasemia

Of patients with parotid hypertrophy, 10% to 66% also have an elevated serum amylase level. Amylase is an enzyme found in saliva, but it is also present in pancreatic fluid and aids in digestion of carbohydrates. Elevated blood amylase level is a known indicator of bulimia. Some research suggests that amylase is more commonly increased in patients who binge and purge than in those who induce vomiting without binge eating (Wolfe et al., 2011). Since elevated amylase levels may be of pancreatic or salivary origin, ordering laboratory tests for both salivary isoamylase and pancreatic isoamylase will aid in confirming the source: only the salivary fraction is elevated in bulimia. Another way to differentiate salivary from pancreatic hyperamylasemia is to test for concurrent lipase level. Normal lipase in the presence of the elevated amylase indicates a salivary gland source rather than a pancreatic source. As is the case with

salivary gland enlargement, elevated amylase levels are more common in patients who have more frequent binge eating and vomiting. Salivary amylase level should be considered helpful but not definitive in the diagnosis of bulimia. Some patients who engage in self-induced vomiting have a normal amylase level, and salivary amylase may be increased secondary to other conditions, such as hyperemesis gravidarum of pregnancy or suppurative parotitis. In addition, the amylase level does not necessarily correlate with severity of symptoms (Wolfe et al., 2011). If cessation of binge eating and purging is successful, the amylase levels return to normal within a few days to weeks.

Conditions Affecting the Ears and Eyes

Problems with sensory organs only add to the many discomforts associated with eating disorders (table 11.3). Patients may report bothersome changes in hearing, including, "I can hear my own voice very loudly in my head," or "I can hear myself breathing very loudly," symptoms termed *autophony*, or *autophonia*. Patulous eustachian tubes are likely the cause of these hearing complaints (Godbole and Key, 2010). Under normal physiologic conditions, the eustachian tubes, which track between the inner ear and posterior oropharynx, are closed. Yawning, chewing, or swallowing temporarily opens the tubes and allows pressure in the inner ear to equalize with pressure of the outside environment, such as when descending in an airplane. It is hypothesized that loss of the tissue, or "fat pad," that aids in keeping the eustachian tubes

TABLE 11.3. Eye and ear complications associated with anorexia nervosa

Sensory organ finding	History and physical findings	Proportion of patients	Treatment
Patulous eustachian tubes	Autophony: hearing one's own voice and breathing very loudly	Uncommon (<10%)	Weight restoration; referral to an otolaryngologist
Lagophthalmos	Dry, irritated eyes	Uncommon (<10%)	Artificial tears; ophthalmic ointment at night; rehydration; weight restoration

closed leads to perpetually open (patulous) eustachian tubes and the associated autophony (Yoshida, Takahashi, Morikawa, 2018). Rapid weight loss in anorexia of either subtype may therefore cause loss of this tissue and result in autophony. On otoscopic examination, movement of the tympanic membrane in concert with breathing is a reliable indicator of a patulous eustachian tube (figure 11.1). Weight gain is the most effective method of resolving symptomatic patulous eustachian tubes among patients with anorexia nervosa (Gaudiani and Mehler, 2016). If the patient is unsuccessful with weight restoration goals, and if the autophony symptoms do not subside, referral to an otolaryngologist is necessary.

Dry eyes are another common complaint among patients with eating disorders. Specifically, patients with severe anorexia nervosa who are dehydrated and extremely underweight may develop enophthalmos, or sunken eyes, thereby disrupting the normal physiology of eyelid movement so that they may not close completely. Lagophthalmos, the inability to completely close the eyelids, is diagnosed when part of the eyeball is still visible after the patient closes their eyes. Without treatment, the patient risks developing corneal abrasions due to lack of normal lubrication and excessive exposure to air. Treatment of lagophthalmos

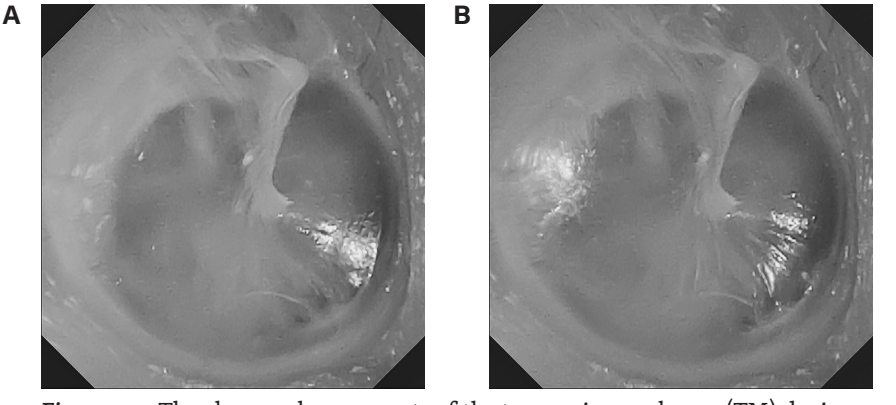

Figure 11.1. The abnormal movements of the tympanic membrane (TM) during the respiratory cycle caused by the transmission of pressures through an abnormally open (patulous) eustachian tube. *Left*, the TM retracting inward (toward the middle ear) during inhalation. *Right*, the TM "bowing out" (away from the middle ear) during exhalation.

consists of frequent use of artificial tears and use of ophthalmic ointment at night. One may consider taping the eyes closed at night as well. Finally, volume repletion, either with intravenous fluids or with nutrition alone, and weight restoration ultimately work to alleviate the enophthalmos and associated dry eyes (Gaudiani et al., 2012). Usually symptoms resolve within 2 weeks in the treatment center setting.

Rarely, subconjunctival hemorrhage, or bleeding under the clear outer layer of the eyeball, may occur in patients with self-induced vomiting. Retching causes an acute increase in pressure in the eye that may cause the fragile blood vessels under the conjunctiva to rupture. Subconjunctival hemorrhage causes the white part of the eye to turn bright red. The condition is fortunately benign and typically resolves without intervention within 2 weeks but can cause much anxiety for the patient.

Summary

Oral, dental, eye, and ear complications are easily overlooked but important indicators of the presence of anorexia nervosa and bulimia nervosa. Individuals with suspected eating disorders should be asked about mouth, teeth, eye, and ear symptoms and have a thorough examination of the oral cavity and eyes. Health care providers should ask about a history of posterior throat pain, thermal sensitivity of the teeth, problems with the gums, sores at the angles of the mouth, dry eyes, and changes in hearing. Likewise, the physical examination should focus on detecting cheilosis, perimylolysis, posterior pharyngitis, swelling of the parotid glands, and lagophthalmos. If the history or physical findings are consistent with an eating disorder, then a more complete general history and physical are warranted to look for other manifestations and complications associated with eating disorders. Ultimately, alleviation of oral, eye, and ear complications hinges on concerted and coordinated efforts directed at resolving or improving the underlying eating disorder.

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12

Athletes and Eating Disorders

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COMMON QUESTIONS

Does participation in athletics predispose to or protect from eating disorders?
Which sports tend to increase eating disorders in females?
What does the term *relative energy deficiency in sport* (RED-S) mean?
Which sports tend to increase eating disorders in males?
What are some vulnerabilities that predispose particular athletes to developing eating disorders?
When does a sport become unhealthy in relation to development of eating disorders?
How can physicians, mental health professionals, coaches, and dieticians work together?
How do you approach an athlete who raises your concern about disordered eating?
Can athletes who have eating disorders return to their sport after treatment?
Do preventive efforts work in sports?
How does muscle/body dysmorphia relate to eating disorders?
How risky are performance-enhancing drugs in relation to the symptomatology of eating disorders?
What role do primary and secondary schools play?

Does losing weight increase performance?
What do overactive, underfed rats teach us?

Case Study 1

A.B. was a 19-year-old female long-distance runner whose eating disorder began at age 12. Shortly after she entered puberty, she lost weight by dieting because she disliked the changes in her body shape, and menses stopped. She responded to outpatient treatment with limited insight but a 10-pound increase in weight. At age 14, she began to challenge herself with endurance running, a sport in which she excelled, winning many championships. She maintained her weight at an improved but still low-for-age level; by age 16, she weighed 115 pounds, at 5 feet 7 inches tall (BMI 18). Menses did not return, because of a combination of low weight and strenuous exercise. In addition to chronic restricted eating, at age 15 she developed a pattern of small binge episodes followed by purging, which led to chronic gastroesophageal reflux; at times, unwanted food spontaneously came up into her mouth. At 16, she began taking metoclopramide 1 hour before meals, but this was of limited help. In addition to diagnosing and treating her gastroesophageal reflux disease, her primary care physician referred her to orthopedics for evaluation of chronic knee and hip pain. The more she ran, the more painful her symptoms became, but she did not disclose her discomfort until her physician found knee pain on palpation.

The orthopedist noted hamstring tendonitis with pes anserine bursitis. Upon examination, A.B. experienced tenderness along the semitendinosus tendon of the pes anserine bursa. With resisted flexion of the knee, she had significant pain. The bursitis was treated with injection into the bursa of 1% lidocaine (Xylocaine) and 2 mL of injectable betamethasone. She obtained complete relief but would agree to rest for only 1 day before running 5,000 meters the second day after treatment. The DEXA scan of bone mineral density showed a deficiency of T = –2.5 standard deviations in the left hip and T = –2.0 in the lumbar spine.

Because of significant anxiety, A.B. was started on escitalopram, titrating up to 20 mg per day, with partial relief. She continued to be preoccupied with staying thin and continued to be very competitive.

Secondary amenorrhea continued. She started taking Ortho-Novum 1/35 (norethindrone and ethinyl estradiol) to restore menses artificially. At age 17, she complained of feeling tired, out of energy, and drained. She began falling asleep in classes, a new problem, and started to take naps—something she previously disdained as a sign of weakness. Laboratory work revealed total serum iron of 35 μg/dL (normal 72–130). Iron saturation was 7% (normal 27%–44%). Hemoglobin was 11.0 g/dL (normal 11.9–15). Weight was stable. The night before track competitions, she experienced high anxiety as well as an increase in regurgitation, and she induced vomiting multiple times. At the examination, for the first time, she had a weepy affect and scratched herself to obtain relief. Escitalopram was increased to 30 mg per day.

At the end of her eighteenth year, she began college. Menses were regular while on birth control pills but only 3 days in duration. She took a break from competitive running, working as a library assistant her freshman year. Her anxiety decreased; she felt "stronger and healthier." Hemoglobin increased to 12.5 g/dL. Weight increased to 120 pounds. She was considered to have improved but to still meet the diagnostic criteria for the female athletic triad because of her continued amenorrhea without oral contraceptive, continued low weight resulting from disordered eating with fear of fatness, and deficient bone mineral density.

At age 19, in the fall of her second year in college, A.B. resumed running, working up to 60–80 miles per week, with several runs of more than 10 miles. Her gastroesophageal reflux was improved. Anxiety had lessened over her year of abstention from competitive running. She still had joint pain but continued to override it with a "runner's high." Only the future will tell whether she will remain healthy enough to run competitively without serious medical complications. She said she planned on a career as an orthopedic surgeon.

Case Study 2

C.D. was a 16-year-old male high school sophomore who was referred by his parents for evaluation of abnormal eating and weight fluctuation. In the eighth grade, at 5 feet 8 inches, he had weighed 185 pounds

(BMI 28.2). He would become winded while running across the football field. When a teammate called him "fat ass," he stopped drinking soda pop and cut out snack food. He reduced his weight to 140 pounds over 18 months but by then had grown to 6 feet 3 inches tall (BMI 17.5). He felt cold compared with others, felt weak, and was mentally preoccupied with his body much of the day.

C.D. decided to develop his body through bodybuilding, eating six meals a day that consisted only of fruits, vegetables, whole grains, chicken, skim milk, and yogurt. He consumed five 2-pound containers of soy protein in short periods, causing distress to his parents. He insisted on eating every 90 minutes and consumed a gallon of soy milk and a gallon of water daily. When, on New Year's Day, the fitness club was closed, he went up to the door and pounded on it to no avail; then he went home and used his weights in the basement. He underwent cycles of weight increase and bulking, followed by cutting weight with running. His goal was to win a bodybuilding contest at age 17. He stated, "I don't care what I weigh, as long as I keep getting bigger, get well-defined, and have 'cut' muscles with no body fat."

His BMI at the time he was seen in the clinic was 22.3 (178.2 pounds, 6 feet 3 inches tall). C.D. looked in every mirror he passed, as well as at his reflection in shop windows. He said his self-esteem depended on his body size and shape. He met the criteria for muscle dysmorphic disorder with a history of anorexia nervosa. His "emergency weights" went with him whenever he left home. He would not answer questions about steroid abuse. He agreed to therapy with a psychologist experienced with athletes who was located close to his home.

Case Study 3

E.F. was a 21-year-old female junior who transferred from an East Coast university and self-referred for continuation of treatment for an eating disorder. She had been diagnosed with an eating disorder in her sophomore year of high school. Despite having had surgery on her knee, she decided to lose weight because her coach told her to get thinner. She went from 140 pounds at 5 feet 8 inches tall (BMI 21.2) to 105 pounds (BMI 16.8), eating 300 calories a day. E.F. realized she was

too thin, decided to increase her lean muscle weight by weightlifting, and restored to 130 pounds (BMI 19.8), with body fat of 13%. Her menses stopped. She switched to basketball, again restricted, and began to binge eat and induce vomiting before each game. A stress fracture was diagnosed when she reported foot pain. At 135 pounds (BMI 20.6) she reported to volleyball summer camp. The coach said she needed to lose "quite a bit of weight" if she wanted a college athletic scholarship. She reduced to 105 pounds again, the weight at which she had started college.

In college, she could not stop throwing up spontaneously and lost weight to 95 pounds (BMI 14.5). She required treatment in an emergency department for dehydration. A shoulder tear required surgery, and her weight increased during convalescence. At the soonest possible time after surgery, she returned to working out, exercising 8–10 hours per day, and reduced her weight to 93 pounds (BMI 14.2). Her binge-purge cycles, throwing up 20 times a night, required repeated emergency room treatments with IV fluids. She often had EKG abnormalities. The school restricted her athletic training and forbade volleyball practice. When she tried to restore weight using weightlifting, a trainer threw her out of the training room because she did not officially belong to a team anymore.

When E.F. began to work out informally with the tennis team, the coach said he wanted to "break her," and the trainer kept asking her if she had "had enough yet." She continued to binge and purge two or three times a day, with binges now triggered by any "crummy feeling" rather than hunger. She continued to work out for 4–5 hours per day, despite stress fractures, engaging in running and weightlifting. She met the criteria for anorexia nervosa, binge-purge subtype, and obsessive-compulsive disorder.

A year later, E.F. presented herself to the eating disorder diagnostic clinic, weighing 169 pounds (BMI 25.7). By that time her illness had migrated to a diagnosis of bulimia nervosa, purging subtype. She complained of a sore throat and hoarseness and had significant parotid swelling. Despite being at a higher weight, she did not regain menses. She used six laxatives a day, felt cold, and was constantly

preoccupied with thoughts of food and weight. Her mood was low, and the perfectionist aspects of her temperament were apparent. She agreed to treatment, but having concealed her disorder from college officials, she required that they not be told. Her future was unclear. This case illustrates, again, the complex course of an eating disorder in an athlete driven to put sports ahead of health.

Background

Participation in sports may either predispose to the development of eating disorders or be a strongly protective factor. The literature differs widely in its findings. One study, for example, found that the prevalence of eating disorders was higher in nonathletes than in athletes (Wollenberg, Shriver, and Gates, 2015), as also reported by Fortes et al. (2014). Other studies have found the opposite (Martinsen and Sundgot-Borgen, 2013). Much depends on the nature of the sport, the gender of the athlete, the motivation and temperament of the athlete, and the actions of the coaching staff, especially for elite athletes. It may also depend on the stage of life for the athlete. It is believed that collegiate athletes specifically are at a higher risk for eating disorders than nonathletes (Poll, 2020). While this could be caused by increased pressure and training from their sport, it is likely influenced by other factors, such as a change in environment, distance from a support system, academic stress, or the food insecurity that many college students face.

For most of the population, sports serve as recreation, stress release, a contribution to identity, and a bonding experience from childhood throughout life, in addition to providing well-documented health benefits, both physical and psychological.

For elite athletes—those on school varsity teams, in competition for the Olympics, in training for competitive sports such as the Boston Marathon—and for professionals, the pressures to succeed in sports are greater. Powers and Johnson (1996) distinguished between thinness for performance goals and thinness for appearance goals in sports as separate risk factors. The combination of the two, as in girls' gymnastics, appears to pose a greater risk than either one alone. Since delineation of

these variables, additional studies have noted the predisposition to eating disorders, primarily in males, when the sport requires substantial muscle development, as is increasingly common in professional sports (football, baseball) and in competitive nonprofessional sports, such as weightlifting and varsity football.

A comparison of photos of Joe DiMaggio, a great baseball player of the past, and of Barry Bonds or José Canseco in games of several years ago shows the difference in body bulk that has become almost normative. A review of *Sports Illustrated* covers over 60 years found that the bodies portrayed became significantly more muscular over the years and that females were much more likely to be sexually objectified in their portrayals (Daffern, Campagna, and Rodgers, 2019). A report on a university football team defensive line documented an increase in average body weight of approximately a pound a year for the past 100 years. Compulsive running and gym workouts by nonathletes who are in the process of developing eating disorders represent secondary overexercise syndromes, rather than predisposing factors. Sports emphasizing thinness or muscle bulk alone and those emphasizing thinness or muscle bulk plus the added pressure of weight-related appearance norms for adjudicated performances are summarized, by gender, in table 12.1.

TABLE 12.1. Body norms or requirements, by gender, of some of the most common sports

	Sport	
Body norm/requirement	Females	Males
Slimness for performance	Cross-country and marathon running	Low-weight crew (rowing), cross-country and marathon running, horse racing (jockey), low-weight wrestling, rock climbing, cycling
Slimness and appearance for adjudicated sports	Ballet, gymnastics, figure skating, diving	Diving, figure skating
Increased muscularity for performance	Sprinting, basketball, softball, weightlifting	Football, baseball, basketball, hockey, sprinting, weightlifting
Increased muscularity and appearance for adjudicated sports	Bodybuilding	Gymnastics, bodybuilding

Prevalence of Eating Disorders in Athletes

The prevalence of eating disorders and eating disordered behaviors differs greatly from one sport to another. In a large study of 1,445 student varsity athletes in all sports, surveyed from Division 1 schools (Johnson, Powers, and Dick, 1999), almost 3% of females had a clinically significant problem with anorexia nervosa, compared with 0% of males; 11% of females reported binge eating at least weekly, compared with 13% of males. Thirty-one percent of women versus 5% of men had BMIs of 20 or lower. Women desired a body with 13% fat, while men desired 8.6%. Norms for body fat content for people of college age are 19%–23% for women and 10%–15% for men. The study authors estimated that 34.75% of women were at risk for anorexia nervosa, compared with 9.5% of men. For bulimic disorders, 38% of both females and males were at risk. These were conservative estimates, given the nature of the survey criteria. When looking at both subclinical and clinical eating disorders, higher prevalence among athletes (13.5%) than nonathletes (4.6%) was found (Sundgot-Borgen and Torstveit, 2004).

More recently, Giel et al. (2016) compared adolescent athletes participating in technical sports, endurance sports, aesthetic sports, weight-dependent sports, ball games, and power sports. They confirmed that the frequency of positive findings on SCOFF screening was twice as high for female as for male athletes. Constant dieting was highest in females in aesthetic sports (e.g., synchronized swimming); the large majority of both males (76.5%) and females (80.4%) in weight-dependent sports used compensatory behaviors (dehydration, vomiting, laxatives, diuretics).

Diagnosis and Evaluation

In sports that encourage thinness for excellence, several issues are involved. Thinness is probably overrated despite the almost unquestioned iconic status given to a skeletal body in some sports. Powers (1999) noted that "athletes think being thinner, no matter what, improves performance. There is good evidence that this is not the case." How much thinness is too much? No easy answer can be given because the weight-

performance relationship varies from sport to sport, but a few guidelines may be helpful. One obvious measure of too much is a decrease in performance. A more subtle measure is when, despite improved performance, thinness becomes an all-consuming goal in cognition and behaviors. In such cases, the athlete pays excessive attention to limiting calories, weighs frequently, and exhibits the psychological features of starvation, such as decreased mental concentration, lowered mood outside of competitive situations, and obsessive fear of becoming fat. Synonyms for anorexia-like syndromes in athletes are listed in table 12.2.

A diagnosis of anorexia nervosa is made—when a clinical interview is possible—with the same criteria noted in chapter 1. These criteria are primarily an overvaluation of the benefits of thinness, self-induced starvation to a degree producing medical or psychological symptomatology, a morbid fear of becoming fat, and, often, overestimation of body size despite obvious thinness. For bulimia nervosa, symptoms are frequent episodes of binge eating that lead to guilt, regret, or medical discomfort, followed by compensation by purging (80%) or nonpurging methods, such as stricter dieting or increased exercise (20%). Screening questionnaires are useful for identifying eating disorders, and recent work on athlete-specific questionnaires, such as the Eating Disorders Screen for Athletes (EDSA), show promising outcomes (Hazzard et al., 2020). While the tools are available, unfortunately there is a tendency for athletes to "fake normal" to avoid limitations in sports activities. Additionally, some less ethical coaches may limit health professionals' access to athletes. Or, more benign in intent but no less damaging in consequence, coaches may simply ignore or be unaware of eating disorder symptomatology in the face

TABLE 12.2. Other terms for eating disorders and weight disorders in athletes

▪ Obligatory running	▪ Weight cycling
▪ Anorexia athletica	▪ Reverse anorexia
▪ Pathogenic weight control	▪ Exercise dependence
▪ Activity-induced anorexia	▪ Cutting weight
▪ Female athletic triad	▪ Manorexia

of a "win at all costs" attitude. Bulimia in athletes is not as obvious as the publicly visible body of an athlete with anorexia, but athletes with bulimia have similar preoccupations with weight at less thin levels. Binge eating disorder is often present in sports that mandate thinness. Eating disorders often go undiagnosed because of a lack of screening or the athlete's fear of shame or embarrassment.

Predisposing Factors

An array of factors may predispose athletes to developing eating disorders (table 12.3). The nature of the sport is a major factor, but an unresolved question is whether predisposed individuals are more attracted to certain sports, such as endurance running. Some sports, such as girls' gymnastics with its requirement of thinness for both performance and appearance standards, seem to try to maintain girls at a prepubertal weight with minimal breast development and continued open epiphyses for longer bones. This presents ethical problems that are sometimes overlooked by trainers and coaches. There are exceptions, thankfully, as seen in the performance of some normal-weight world-class female athletes competing in gymnastics, such as Simone Biles and Suni Lee, both with a BMI of 22.

TABLE 12.3. Psychological and behavioral characteristics that predispose athletes to eating disorders

Perfectionism	Emotional immaturity relative to age
Low self-esteem	All-or-none reasoning: winning or failure seen as only outcome
Sports injury limiting participation	Pressure from coaches
Rejection sensitivity	Excess demands by parents or coaches
History of childhood/preadolescent obesity	External locus of control
History of abuse	Participation in sports to please others
Body/muscle dysmorphia	Body weight dissatisfaction
Social isolation	Chronic dieting, frequent weighing
Family history of mood or eating disorders	Anxious or depressive illness or traits

Ballet is in many ways an athletic activity, requiring increasing degrees of athleticism in modern dance. In preprofessional schools of ballet, a high percentage of girls (50% in one study) experience amenorrhea, and there is a sevenfold increase in cases of anorexia nervosa compared with the general population. Anorexia nervosa is the correct diagnosis when there is a failure to increase in weight proportional to increases in height and age just as when there is a loss of weight through dieting and excess exercise.

Psychological factors play a major role in predisposition to eating disorders in athletes. An external locus of control—psychological terminology for basing self-esteem primarily on approval from others—mandates continued winning in athletics at all costs, a fragile basis for self-esteem. An external locus of control predisposes an athlete to be excessively sensitive to comments from coaches and peers. Perfectionism is associated with all-or-none thinking: not winning equals losing and being a failure. An added burden is rejection sensitivity, with the perception that anything less than first place is a rejection. Performance anxiety may be so severe that an eating disorder comes to be an alternative strategy for relief from anxious participation in sports.

A sports injury may be an annoyance or a major tragedy in the mind of an athlete. For those with little identity outside athletics, an injury that requires a time of healing away from the sport—or, more traumatic, discontinuation of the sport in which the athlete has shown excellence—can be devastating. During time away from the sport, an injured athlete may overcompensate for reduced energy output by excessive restriction of food intake, predisposing them to anorexia nervosa. In addition, for those with an almost exclusive focus on sport for identity, depression, demoralization, and a perception that life has passed them by may ensue. Overuse injuries represent an imbalance between activity and recuperative forces, fostered by drivenness in athletics. The number of women participating in sports has increased dramatically as a result of both cultural factors and governmental requirements for gender parity in college sports (Hilibrand et al., 2015), leading to an increase in the number of female athletes with eating disorders and sports-related injuries.

Risk by Gender and Sport

Gender-Neutral Sports

Numerous studies have evaluated symptoms of eating disorders in athletes who participate in endurance running and triathlons, which are gender-neutral sports. Thompson (2007) found that 19.4% of female cross-country runners had previous or current eating disorders, 23% had irregular or absent menses, and 29.1% had inadequate calcium intake. Low bone mineral density of the lumbar spine was found in both male and female endurance runners (Hind, Truscott, and Evans, 2006). Most female triathletes with a healthy BMI wished to be smaller, in contrast to a minority (19.3%) of males (DiGioacchino DeBate, Wethington, and Sargent, 2002). High dietary restraint, a prelude to an eating disorder, has been documented in adolescent female endurance runners (Barrack et al., 2008). A Scandinavian study found that a high proportion (46.7%) of athletes in leanness sports had clinical eating disorders, compared with 19.8% in non-leanness sports (Torstveit, Rosenvinge, and Sundgot-Borgen, 2008). Disordered eating and eating disorders in aquatic sports ranged from 18% to 45% in female athletes, compared with 0% to 28% in male athletes.

Female-Related Sports

The term *relative energy deficiency in sport* (RED-S) has been proposed as a broader label to include the commonly used *female athletic triad*, a construct referring to the combination of an eating disorder, amenorrhea, and osteoporosis that can develop in young female athletes who have achieved a low body weight (Sundgot-Borgen, 1994). The "Authors' 2015 Additions to the IOC Consensus Statement" (Mountjoy et al., 2015) describes RED-S as a gender-neutral condition in sports that includes impaired physiologic functioning caused by relative energy deficiency, combining such features as cardiovascular health, protein synthesis, bone health, and metabolic rate. In one study, most distance-running young women with amenorrhea had a family member with a mood disorder and/or an eating disorder, whereas no relatives were affected with

an eating disorder or a mood disorder in a comparison group of eumenorrheic runners (Gadpaille, Sanborn, and Wagner, 1987). A family history of mood or eating disorders predisposes female athletes to seek low body weight.

Women's gymnastics contrasts markedly with men's gymnastics. The ideal female performer is usually young, often prepubertal in endocrine development. Pubertal changes may diminish flexibility and competitive performance in complex gymnastic maneuvers. Female gymnasts accepted into college on sports scholarships may find that development of an adult body endangers their performance and their scholarship status. In contrast, male gymnasts often do not reach their peak until the end of their college years or shortly thereafter, with developmental body changes along with strenuous training improving their performance. In one study, more than 60% of elite female athletes in sports demanding leanness reported pressure from coaches regarding body shape (Kong and Harris, 2015).

Only a few female sports require increased muscularity and weight. These sports, such as female bodybuilding, are somewhat anomalous for most young women. Lean muscularity is more normative in female sprinters. In both categories, women may be at risk for anabolic steroid abuse, more common in male athletes. Professional runner Mary Cain recently brought light to and started a movement around the toxic and sometimes abusive environment surrounding female athletes, one that has both physical and emotional consequences. A movement involving awareness, creating safe training protocols, and eliminating toxic training environments is necessary to improve the long-term health of female athletes (Ackerman et al., 2020).

Male-Related Sports

Eating disorders in males are often thought to be underreported, with recent reports suggesting that cisgender males may account for up to 25% of people with eating disorders (Eichstadt et al., 2020). While it is more common, and perhaps easier, to recognize eating disorders in female athletes, we cannot allow male athletes to fly under the radar. In

male athletes, dieting and disordered eating occur primarily in the male-related sports in which leanness improves performance or aesthetic judgments are involved (Goltz, Stenzel, and Schneider, 2013). Thinness for performance as a risk factor for disordered eating behavior in men has been documented in lightweight rowers compared with heavyweight rowers (Pietrowsky and Straub, 2008), with lightweight rowers scoring very high on restrained eating and body dissatisfaction. Thinness for performance as a risk factor has also been documented in low-weight wrestlers (Thiel, Gottfried, and Hesse, 1993), as well as in male racing jockeys (King and Mezey, 1987), young rock climbers (Morrison and Schöffl, 2007), and male cyclists (Riebl et al., 2007).

Many primarily male sports require increased muscle bulk, usually accompanied by striving for extremely low body fat. The more professional the level of participation in the major sports (football, baseball, basketball, hockey), the more muscularity is valued—with a few exceptions, such as the quarterback in football, who may maintain slim muscularity in place of bulky muscularity. For male sports requiring both greatly defined muscularity and low body fat, such as bodybuilding, the dark side of competitiveness enters with the abuse of anabolic steroids, often at large doses (Piacentino et al., 2015; Pope and Kanayama, 2004). These athletes may have *reverse anorexia*, which is characterized by perceptual distortion of the opposite kind seen in female anorexia nervosa: never being large enough or muscular enough, being insufficiently "cut" and too fat, even at very low percentages of body fat. Binge eating is common.

Some of the health hazards of anabolic steroid abuse, in both genders, include excessive weight increase, abnormal liver function, increased hemoglobin (predisposing to strokes, blood clots), and abnormal functioning of reproductive hormones. The quest for performance and winning gold, unfortunately, overrides rational consideration of the health hazards of anabolic steroids (table 12.4). In males, especially, the areas of the brain primarily involved in judgment and decision making are located mostly in the prefrontal cortex, which develops later than in females—but, one hopes, by the midtwenties; the robust, less well-regulated, earlier developing impulsivity centers in the amygdala are highly active during

TABLE 12.4. Medical and psychological problems associated with abuse of anabolic steroids

Males	Females	Both genders
Atrophy of testes	Facial hair	Acne
Reduced sperm count, impotence	Irregular or absent menses	Abnormal liver function, jaundice
Baldness	Clitoral enlargement	Reduced HDL ("good") cholesterol
Gynecomastia (enlarged breasts)	Deepened voice	Increased chance of injury to tendons, ligaments
Premature prostate enlargement	Reduced breast size	Edema, fluid retention, increased body weight, increased hemoglobin (blood clots, strokes)
Increased rage and lack of anger control in predisposed males	Laryngeal enlargement	

the teenage years. The catchphrase "Get big or die" is, unfortunately, too often promoted. "Get big and live—by healthy means" is a rational choice but less appealing before full psychosocial maturity.

Treatment Recommendations

How can a concerned sports-related person approach an athlete with a possible eating disorder? This is the first question asked by worried peers, teachers, coaches, and parents. A nonjudgmental expression of concern is the beginning. When several concerned individuals, such as peers and a coach, together approach the athlete and recommend to them a specific expert in eating disorders who is familiar with athletes, the athlete will often accept a confidential referral. A period of absence from a sport until improvement occurs is often necessary, so the long-term goal of coming back as an even better athlete needs to be stressed. Occasionally, an athlete is simply not suited to a particular sport. For example, a larger-boned woman who wants to compete with low-BMI endurance runners may do better in another sport, but that is an individual choice that needs to be respected. Similarly, a larger male who

wants to row in a lightweight crew is at increased risk for an eating disorder because of the weight loss required. Here again, personal choice is respected.

The core of eating disorders, of course, has little to do with weight, shape, or food. Eating disorders are strategies for dealing with deeper issues in life, such as peer approval, self-esteem, perfectionism, lack of balance, developmental arrests, mood regulation, and relationship conflicts. Activities therapy experts in an experienced eating disorder team can guide the athlete back into a slowly graded increase in athleticism as the eating disorder improves. A significantly lowered bone density, probably less than –2 standard deviations from norms, suggests that high-impact sports should be avoided. Specific advice about participation in a sport should be geared toward individual differences. Early intervention in the female athletic triad benefits the long-term health of girls and women (Lebrun, 2007).

The injured athlete presents a challenge, especially when that person's identity is largely centered on excellence in sports. Early intervention for injuries, rather than after repeated trauma to joints, ligaments, and tendons ("Let me complete the season"), is essential.

Inappropriate dietary restraint is unhealthy, whereas abstention from junk foods high in transfats or concentrated sweets is healthy. Excess dietary restriction limits calcium, an essential component of improved bone mineral density. Up to 95% of bone density is completed by the late teen years. Lowered calcium intake during those years has long-term consequences for building strong bones that will last a lifetime. Garner, Rosen, and Barry (1998) summarized recommendations for intervention and treatment for disordered eating behaviors in athletes.

While the eating disorder itself may not always be different between athletes and nonathletes, the treatment approach should be unique. Sports performance concerns and life after treatment must be taken into consideration when treating athletes, as their performance is often a theme of their treatment and recovery (Arthur-Cameselle et al., 2018). Typically, a team approach to treatment of the athlete with an eating disorder works best, with the coach and the trainer being involved in nonconfidential aspects of the treatment. It is recommended that all coaching

and athletic personnel be informed about and sensitive to eating disorders (Conviser, Schlitzer Tierney, and Nickols, 2018). No specific psychopharmacologic agents have proved to be of benefit in the treatment of anorexia nervosa, but specific antidepressants may be individually prescribed, especially for comorbid depression and anxiety. For bulimia nervosa, cognitive behavioral therapy is still the gold standard, but here, too, antidepressants may be of use as minority partners in treatment.

The Role of Coaches in Promoting or Preventing Eating Disorders

Coaches often find themselves in a difficult position when facing the potential absence of one of their athletes, especially those who are star players in their sport. Accepting a potential decline in team outcomes, keeping confidentiality of the athlete in question, and protecting scholarships in collegiate sports are a few of the common concerns that arise in these situations. Coaches either can promote eating disorders or can be essential partners in their treatment by supporting the clinician and encouraging the athlete to seek full remission of the eating disorder rather than a small amount of improvement. Coaching style clearly affects an athlete's vulnerability to an eating disorder. There are important ethical issues in coaching. Placing the long-term welfare of the athlete first is crucial. The "winning at all costs" philosophy is unethical, certainly from grade school through college. Professional athletes may choose to play when injured, being of reasonable age to make such a choice even though it may still be an unwise decision that leads to early career drop-out due to injury. Young athletes who are still developing are not always sufficiently mature to make choices in their best interest, despite what they think, and may be overly influenced by pressure from coaches to place performance above all else. Good coaches are among the most influential and admired persons in an athlete's life. One study found that 67% of athletes with eating disorders reported that they were dieting on advice from their coach (Sundgot-Borgen, 1994). Sherman et al. (2005) summarized the role of coaches in 23 sports in identifying and managing athletes who had disordered eating, noting that "athletic

trainers, teammates, and coaches are frequently involved in identification." A modified version of a team approach to the athlete with an eating disorder is shown in figure 12.1 (Joy, Kussman, and Nattiv, 2016). Martinsen, Sherman, et al. (2015) document a sevenfold improvement in coaches' knowledge after an interventional teaching program about nutrition, weight regulation, and eating disorders.

The Role of Schools in Healthy Athleticism

Schools, by their action or inaction, promote or hinder healthy athleticism. School policies and practices can promote healthy sports (table 12.5). A 2013 report by Sundgot-Borgen (a long-term leader in the field of sports and eating disorders) and coauthors summarizes how to minimize the health risks to athletes who compete in weight-sensitive sports. The ruthless decimation of sports programs for average students during the school years leads to more obesity and, in later years, more heart disease—major public health issues. It is said that World War II was won on the playing fields of Eton, in terms of building character and fitness. Likewise, many of the lifelong health hazards of adulthood in modern society might be prevented or lessened on the nation's playing fields—now often unavail-

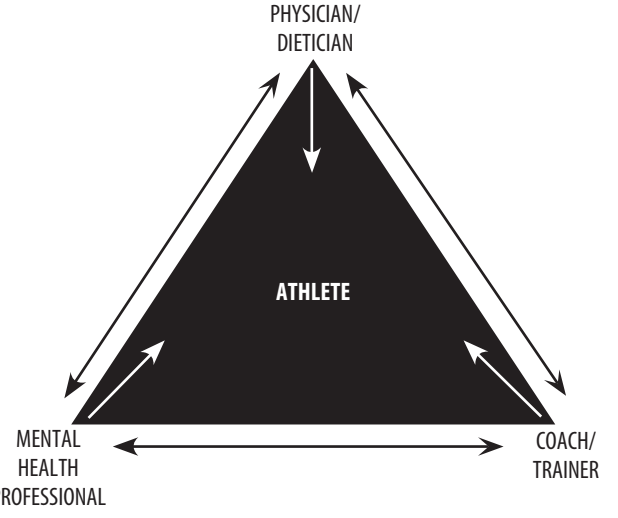

Figure 12.1. Multidisciplinary care of athletes with eating disorders

TABLE 12.5. School policies and practices to promote fitness and prevent eating disorders

- Involve all students in athletics: cardiopulmonary, strength, flexibility, team sports.
- Stop limiting athletics to elite athletes, as is currently done beginning in junior high/middle school.
- Regulate the body fat and recent weight loss allowed in wrestlers, cross-country and endurance runners, and gymnasts.
- Identify eating disorders early and refer students for treatment.
- Involve coaches in designing a program balance between promoting competitiveness and preventing disordered eating and weight behaviors.
- Monitor in particular all sports requiring leanness or aesthetic appearance (girls' gymnastics, cross-country, ballet, etc.).
- Involve parents with coaches in program balance.
- Include judges of adjudicated sports in the loop with health professionals, coaches, and parents in discouraging excess/rapid weight loss for participation in sports.
- Emphasize fitness in place of thinness.
- Make nutritional education and school lunches both fun and healthy (as in France and Scandinavia).
- Remove soda machines.
- Provide individualized sports/exercise programs for students for the school year.
- Provide psychological counseling for injured athletes unable to participate.
- Promote a functional view of the body for girls and young women rather than an objectification of body size and shape that leads to critical judging of worth by self and others.
- Emphasize lifetime carryover sports and fitness patterns for during and after the school years.
- Emphasize participation by students with disabilities.

able—by the participation of all students in sports of some kind, and certainly in daily physical activity of 45 minutes, five times a week. The contribution of regular participation in an aerobic sport, as well as in strength training, to *mens sana in corpore sano* (a healthy mind in a healthy body) is supported by many evidence-based studies. Regular exercise can be as effective as antidepressants in nonpsychotic depression and in anxiety states.

In the United States, students at about the middle school level are too frequently divided into elite athletes, for whom facilities are provided for intense participation in sports, and most students, for whom athletics is neglected but who would benefit from encouragement to participate in sports that could carry over for a lifetime of better health. The rise of electronic games and social media have contributed to obesity and physical passivity for many students. Electronic sports games provide an illusion of participation. School board decisions as well as busy parents govern these practices of inattention to sports for the average student and the neglect of physical activity. While political advocacy is not the usual focus of an academic physician, I strongly support the use of tax monies for construction of facilities for mandatory daily physical education in schools and the participation of all students in some level of sports—with financial support for the teachers and coaches of these activities as assured as the funds for teaching English and mathematics. Unfortunately, most families cannot afford the expensive preparatory schools that usually implement this kind of balanced education.

When it comes to intervention, it is important that coaches and administration are working together to create processes that protect athletes whose well-being may be at risk, policies that protect confidentiality, create accountability, involve support systems, protect scholarships, and approach these situations in a safe and sensitive manner.

Possibilities for Prevention

Whether measures for the prevention of eating disorders and RED-S in athletes are possible is a subject debated among eating disorders experts and approached in different ways by different nations. Some would argue for primary prevention (Martinsen, Bahr, et al., 2014), while others recommend secondary prevention (early intervention). Norway, for example, encourages primary prevention. It designates a nurse clinician in each district who is trained to intervene early for athletes who might have an eating disorder. The United Kingdom and the United States tend to wait until eating disorders are more fully entrenched, or to ignore their existence in the service of winning performances. Powers and

Johnson (1996) addressed "small victories" occurring in the prevention of eating disorders in athletes. Oppliger et al. (1995) reported on states such as Wisconsin that have instituted mandatory standards to limit unhealthy dieting among student wrestlers. Limiting the amount of weight that may be lost to participate in endurance running or ballet can sometimes turn around a potential eating disorder before it reaches the point of having no voluntary component.

More indirect, long-term preventive efforts include changing the culture in which women's bodies are objectified. Young women need to learn to view their bodies from a stance of functional instrumentality (i.e., having a healthy body to *do* things), not as something to be objectified and judged by peers or oneself in terms of thinness and appearance norms. Skepticism of the media is healthy, as is replacing magazines and other media that promote thinness with healthy role models. *Normative cultural distress*—giving lip service to the need to diet a little but having that dessert anyway—is healthy; a total unconcern about weight is unrealistic. Building "body armor" by encouraging a balance between sports participation and academics for girls will allow them to increase their self-esteem by internalizing skills in athletics during developmentally critical years.

Dieting is the crucial behavior that promotes a transition from an athlete with a culturally healthy concern about weight to one with an eating disorder. There is increased, and appropriate, focus on identifying disordered eating (DE), which represents unhealthy eating habits that do not quite meet criteria for a clinical eating disorder. DE is common among athletes and can be a warning sign for clinical eating disorders and therefore a trigger for prevention. The United States is both an underfit and an overfat nation. If every parent required each school-aged child to present a sweat-stained T-shirt before allowing them to use a computer or play video games, there would be more fitness and perhaps less adolescent obesity, with a resulting decreased need or desire to diet.

Basic Scientific Research: What Can Rats Teach Us?

In laboratory studies on rats, scientists have reproduced conditions similar to those faced by athletes: unrestricted access to an activity wheel

and restricted food intake. This combination of hyperactivity plus "dieting," very common in athletes, leads to a vicious circle of losing more weight and eating less food, despite the availability of adequate calories. Activity-based anorexia is facilitated by neuropeptide Y (Nergårdh et al., 2007) and suppressed by leptin (Exner et al., 2000), suggesting that in this model of anorexia nervosa, hyperactivity and self-starvation are regulated in the hypothalamus and other areas of the limbic system, areas in which neuropeptide Y and leptin interact.

The basic scientific model of anorexia, of course, cannot duplicate the overvaluation of thinness, but it does strongly suggest that biological factors may lock in anorexia nervosa in the presence of conditions similar to those under which athletes compete. Obligatory runners and other compulsive exercisers often feel dysphoric when not allowed to run. Eating disorders may be initiated by choice but then sustained past a certain point by brain peptides.

Summary

Eating disorders are a major concern in athletics. The emphasis on thinness for performance and for appearance increases the probability of developing an eating disorder. The participation of predisposed young athletes, those with several risk factors, in thinness-mandating or aesthetic sports leads to the highest prevalence of eating disorders. Clinicians need to be knowledgeable about the healthy and unhealthy aspects of sports and to work closely with coaches, trainers, and sports educators. Success in detecting eating disorders by requiring athletes to respond to questionnaires is limited by several factors, including an athlete's wish to appear healthy and fit and not be excluded from sports participation. A well-educated, ethical coach, as well as a programmatic school-based approach to healthy eating and activity, can identity and treat or, even better, prevent eating disorders. The preponderance of evidence supports the encouragement of lifetime athletic participation for all persons, including those with disabilities. Whether elite athletes are more likely to develop eating disorders than the average nonelite athlete or nonparticipatory student remains controversial. In the meantime, it

is important to identify and treat the subset of athletes who do develop eating disorders, recognizing certain sports as posing a higher risk.

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13

Males with Eating Disorders

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COMMON QUESTIONS

How do males and females with eating disorders differ?
Are the differences important?
What kind of sociocultural milieu do males face?
What is the most common ideal body physique for men?
What is *physique anxiety*?
Why are there fewer males than females with eating disorders?
What economic forces are involved in male body dissatisfaction?
Is the diagnosis in males different from that in females?
Has *DSM-5* changed the diagnosis of eating disorders for male patients?
What is "reverse" anorexia nervosa?
Is muscularity-driven eating disorder the same as reverse anorexia?
What do the terms *macromusculophilia* and *microsomatophobia* mean?
Which males are more likely to develop eating disorders?
Are eating disorders and muscle dysmorphic disorders addictions?
What are the medical needs of male patients with eating disorders?
How can male patients with anorexia nervosa achieve a desired body shape and simultaneously decrease recurrence?
Do males with eating disorders develop osteoporosis?
What are the most common comorbid disorders in male patients?
How do you determine a target weight for males with anorexia nervosa?

How do you treat binge eating disorder in a male patient who is obese?
Can males with disordered eating behaviors return to intensive sports?
How common is a gay orientation in males with eating disorders?
Why are male military personnel and prisoners at risk?
Can eating disorders in males be prevented?
What possible role does feminism play in males with eating disorders?
How do males with eating disorders view their treatment programs?
Is a change needed in the conceptualization of males with eating disorders that better fits with their history and presentation? Is it timely to add the category of muscularity-driven eating disorder (MDED) to the subtypes of males with eating disorders?
What is the good news in the field of males with eating disorders?

Case Study: Binge Eating Disorder

J.B. was a 30-year-old man evaluated in a weight management clinic for complaints of depression, insomnia, and obesity. He was accompanied by his father. The history documented that the patient was overweight as a child but compensated by taking part in sports. During high school he excelled in basketball and baseball despite increasing in weight, which reached 310 pounds at graduation. Over the next two years, he fluctuated between 320 and 280 pounds while attempting to lose weight on various programs, including two liquid meals a day, followed by a keto diet, and then by the addition of Vyvanse. By age 25, his weight was 400 pounds. At the time of the evaluation in the Weight Management Program, his weight was 550 pounds. His father said that he had been intermittently depressed for more than 2 years, with episodes of dysphoria lasting more than 2 weeks, associated with worsening tiredness, loneliness, and increased eating. He had voiced suicidal ideation with a plan twice in the past. During middle school, he was diagnosed with ADHD and oppositional defiant disorder. He required a CPAP for his sleep apnea. He was given diagnoses of major depressive disorder, autism spectrum disorder, and morbid obesity (BMI 62). Between the evaluation at the weight management clinic and the referral to the eating and weight disorders clinic, he had been told by the bariatric surgery

clinic that he needed to lose 50 pounds before being considered for surgery, which he did not accomplish.

J.B. was referred to the eating disorders clinic for a greater focus on his abnormal eating pattern and a consideration of treatment options. The eating disorders clinic documented many features typical of binge eating disorder (BED) in J.B. He reported, "I am not successful. In anything. I don't have anything going for me. Eating makes me feel good. When I eat, it's like a drug." He would skip breakfast but then go to several fast food restaurants for lunch, feeling "stuffed but warm." He overate even if he was not hungry, primarily on dense, high-calorie foods. He ate supper with his parents, but then felt unable to interrupt urges to binge eat after his parents went to sleep. His PHQ-9 score was moderately elevated at 15. His GAD-7 was 5, mild anxiety. His maladaptive eating appeared to be his primary coping strategy for dealing with the stresses of his life, including loneliness, disappointment in himself, physical pain, and fatigue.

A diagnosis of binge eating disorder (BED) was made, in addition to confirming the diagnosis of major depressive disorder. A diagnosis of a learning disorder was added after a full neuropsychological evaluation. He is being followed by a multidisciplinary team to meet the weight loss goal set by the bariatric surgery clinic. He is prescribed armodafinil, 250 mg BID; escitalopram, 20 mg/day; phentermine, 37.5 mg q a.m.; tramadol, 50 mg prn for joint pain; lorazepam, 2 mg at bedtime; melatonin, 3 mg at bedtime; and Vyvanse, 60 mg capsule daily. He meets weekly with a psychotherapist. His internist is given regular reports of his progress. He is thinking of taking one course in the community college. A physical therapist helps him to decrease his leg and joint pain. He has begun to take part in a hospital-based mindfulness course. This case illustrates the complex longitudinal course of a typical patient with severe obesity and BED.

Case Study: Anorexia Nervosa with Inadvertent Onset

L.S. was a 31-year-old medical student referred by his father, a social worker, who was concerned that his son might be suffering from anorexia nervosa or depression. The patient had been about

135 pounds in college, at 5 feet 6 inches tall (slim normal). In the context of competing in triathlons and the Iron Man competition, he compulsively exercised to increase speed in the three areas of competition: swimming, biking, and running. His weight decreased over several years of competition until he fluctuated between 100 and 105 pounds. Despite feeling cold and lacking in appetite, when confronted by his physician for being too thin, he said, "I don't see it." L.S.'s primary goal was increased athletic skill. He did not initially desire to decrease his weight. Only after his weight fell into the clearly anorexic weight category did he develop a fear of eating normally and becoming fat. He began to experience binges at night without any purging but engaged in compensatory starvation the following day and increased his exercise program despite extreme fatigue (diagnosis: anorexia nervosa, binge-purge subtype with other compensation). "I started feeling fat. I'm sure my weight is controllable. I feel some shame about the overeating. I don't think I'm too thin." He had impossibly high standards of excellence in work as well as in athletics. On mental status examination, he was not depressed and did not meet criteria for OCD. He did have a morbid fear of fatness and a relentless drive to remain at his anorexic weight.

After motivational interviewing, L.S. was given psychoeducation about the nature of anorexia nervosa, the risks of continued underweight, and a summary of the steps in treatment. He was informed about the genetic predisposition to eating disorders and noted that his mother was an extremely anxious person with whom he could not discuss anything. He was referred to a local psychiatrist experienced in the treatment of eating disorders but declined to meet. His was a case of inadvertent onset of anorexia nervosa, not initially wanting to lose weight or perceiving himself as being too fat. Only after he had lost a considerable amount of weight did he develop a distorted perception of his body size as being too big along with a fear of eating too much and becoming fat.

L.S. consulted with several additional mental health professionals to determine whether there was consensus that he needed treatment. After several months of his weight continuing in the anorexic range,

and despite his honest and nondelusional belief that he was not too thin, he cautiously began outpatient therapy with an experienced psychiatrist. After failing to restore weight as an outpatient, he reluctantly accepted a referral to the day hospital (partial psychiatric hospital). In day hospital his treatment was effective in restoring a healthy weight, decreasing his fear of fatness, and achieving a healthy body image. He was able to eat meals with buddies in the community and engaged in moderate sports activities.

Overview of the Field of Males with Eating Disorders

The field of eating disorders in men and boys is an exercise in deciding if the glass is half empty or half full. It is not uncommon for a newspaper to write about males with eating disorders that "It's not healthy and it's decidedly not masculine." MacLean, Sweeting, et al. (2015) addressed the issue of stigma toward males with eating disorders. They concluded that media present eating disorders in men as being atypical, associated with demasculinizing/feminizing characteristics, marked by shame and concealment, leading to a delay in help seeking, and reducing chances of success in treatment. Richardson and Paslakis (2020) documented that men were reluctant to recognize their disorders, faced minimization of their symptoms by health professionals, and had difficulty finding treatments that were tailored to their specific needs and preferences as men or boys. Strother et al. (2012) titled their publication "Eating Disorders in Men: Underdiagnosed, Undertreated, and Misunderstood."

Encouraging research and clinical experience support the opposing view—that the glass is half full, not half empty, without minimizing the challenges that men and boys face. The example below illustrates how initial, comprehensive, multidisciplinary treatment including male-specific components, followed by a long arc of stepwise full recovery, have led to enduring wellness and a flourishing life.

Note that BMI in males with anorexia nervosa grossly understates the severity of their illness (Philip Mehler, personal communication, 2020). Where possible, the percentage of ideal body weight (IBW) is presented in addition to BMI.

Case Study: Anorexia Nervosa, Binge-Purge Subtype (Other Compensation) with a Healthy Long-Term Outcome

T.J. was a 24-year-old young man when referred to the eating disorders inpatient treatment service. Between age 17 and 18, he experienced turmoil in his family relationships. His mother had suffered from a long depressive disorder, followed by terminal breast cancer. Custody battles in court preoccupied his parents. Finances were meager. His father was supportive but busy during the separation. T.J. weighed 175 pounds at 72 inches tall (100% IBW; BMI 23.8). For reasons that are not completely clear, T.J. reported that his weight loss began indirectly: "I tried to see how long I could go without eating. In my mind, I thought I needed to sacrifice my body. I felt depressed. Hunger was my way not to feel the depression. It felt safe when I was hungry. I wasn't in control when I was eating. I lost control of my eating and my depression. I was first hospitalized for depression, then they referred me to the University of Iowa, Department of Psychiatry, because they didn't know how to deal with the weight loss." He had decreased weight to 96 pounds at age 24, despite increasing one inch in height (55% IBW; BMI 12.7).

During his inpatient treatment, T.J. added information about his course of illness: "I did lots of walking. I was preoccupied with food. I was walking around grocery stores. I was looking at the food, but I didn't let myself buy or eat any food." He believed that restoring weight meant losing control. He had also begun to binge eat: "The first time I binged, it was followed by four or five hours of exercise. I thought I was too fat. I thought I was eating too much."

T.J. was treated with a multidisciplinary program focused on a common modality of cognitive behavioral therapy for all participants, including the attending psychiatrist, the resident, social worker, advanced nurse practitioner for group therapy, PhD psychologist, registered dietitian, occupational therapist, educator, and staff nurses. Rounds each morning consisted of a review of each patient with an eating disorder by all members of the team, with a resulting revision of the therapeutic plan. Then each patient was interviewed individually by the attending psychiatrist, resident, and social worker. Assignments

were given to each patient on most days to focus their therapeutic work. The resident conducted two individual psychotherapy sessions weekly.

T.J. experienced night sweats for several weeks during his weight restoration. His low testosterone at admission gradually increased to normal levels. He suffered no serious medical symptoms. Even before he moved to the psychiatric partial hospital (at 85% of target weight), he began to participate in a men's group on Friday afternoons with the male attending and the male social worker from the partial hospital. All men and boys with eating disorders were invited to come to the men's group, including those discharged from inpatient care and returning for follow-up, as well as those in the community who were considering treatment. Fathers were welcomed. T.J. has reflected positively on the afternoon group for men and boys, the strength training, and the male members of the staff: "It was fantastic—the strength training, the male nurses—I felt a lot closer to them than the females. The strength training was a huge help. I was actively doing something to improve my body."

Fast-forward 20 years: T.J. maintains a stable weight of 185 pounds. For a while, when he was doing more intensive weight training, his weight was 200 pounds. He is now comfortable with maintaining 185 pounds. He estimates that his complete recovery from his eating disorder took about 15 years. He reflects on his eating disorder: "Treatment was good. It was the depression that led to the eating disorder. To alleviate the depression, I used hunger. I thought that if I felt hungry, I would be less depressed. If my weight went lower, I knew I was in control." He now enjoys spending time with his father and brother. He works part time in two restaurants. He rows on a local river when the weather is good. "I love the work. I have a great girlfriend." He enjoys the simple pleasures of life: "Walks, coffee. I like being around people in a coffee shop, but I don't have to be talking to them. I sit and I relax." He concludes his retrospective reflections: "I'm very fortunate. I'm happy where I'm at."

T.J.'s history and response to treatment illustrate several features that merit consideration. His first treatment facility was not able to treat the

combination of an eating disorder and depressive illness. He has had a slow but uneventful and robust response, beginning with his initial multidisciplinary treatment. His reflections on the reason for his weight loss do not fulfill all the textbook components for a typical young man with an eating disorder: He had an intermix of depressive disorder combined with a desire to lose weight to gain a sense of control of his life and to become less depressed. He at times recognized that he was too thin but feared that weight restoration would lead to being out of control of life in general, a life that had multiple challenges. He believed that he benefited especially from the components of the treatment program that were directed to men and boys: his confiding relationship with male nurses, his participation in strength training, his attendance at the men's group. He also participated in all the gender-neutral treatment parts of the program, which were roughly 90% of the total treatment time. He has kept in touch with me by occasional cards and notes, sharing his reflections, which he gave permission to use in this book. It should be noted that in the inpatient and partial hospital program described above, separate groups are provided for female participants, as well as males, according to their needs and experiences. For example, groups for female victims of sexual abuse and trauma are always present. The other components of most eating disorder programs originated in a female-oriented milieu but can without difficulty be carried out in a gender-neutral way.

The Evolving Sociocultural Milieu for Males

The sociocultural environment for men and boys has adaptive, healthy, gender-specific components as well as maladaptive features that predispose to the stereotypical picture of some males: aggressive; hypermasculine; suppressing emotions, allowing only feelings of anger and sexuality to be expressed; fearing emotional intimacy; using alcohol excessively; exhibiting bravado; and objectifying women. Even separate from this almost-comic Neanderthal male depiction, the sociocultural milieu aimed specifically to males promotes other, less obviously unhealthy stereotypes: males should be lean but muscular, nutritionally wise yet always picking the steak option, sensitive to a fault but brave

when required, fearless and commanding in war, acute listeners in long-term partnerships always; in control but never controlling; wellsprings of comfort; carrying out sexual relationship by mutually agreed steps, yet skillfully taking the initiative in sexual relationships; agreeable to psychotherapy when needed but completely self-sufficient; longing for abiding male friendships while able to withstand aloneness; competitive in sports but collaborative in love and business; and above all, without visible fat, bulges, or jiggles; not having too little or too much body hair; having broad shoulders, an abdominal six-pack, slim hips, and strong legs; being tall with symmetrical, strong but kind facial features, with warm smiles.

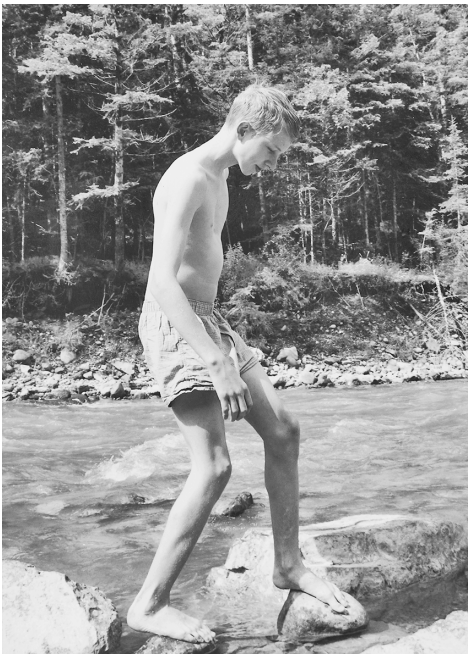
A 40-year-old male with an established career, a warmly interactive partner relationship, enjoying abiding community friendships, growing in his spiritual depth, and endowed with a good personality would find the above stereotypical requirements oppressive and more than faintly ridiculous; this stereotype, however, is not at all ridiculous for preteen boys, adolescent males, and young adult men below about 25 years of age. For them, a central imperative in their lives is to do whatever it takes to conform to the desired physique: diet, lose weight, bulk up, strength train, hit the gym twice a day, stretch, use strength-enhancing substances, forget about sensitivity, become a weekend warrior, always take the lead in sports and sex, squash the opposition in work or play, don't show any weaknesses. While this adverse path in development toward eating disorders has been modeled mostly in Caucasian males, it is increasingly sought after in minority groups, and to some extent in sexual minorities.

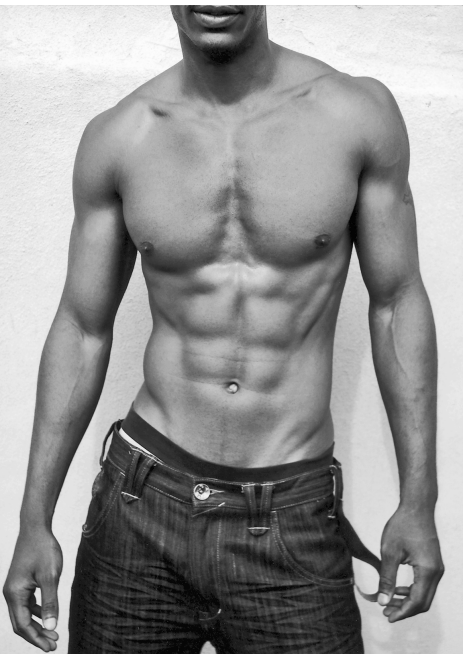
Regarding dieting, Galen (authoritative ancient Greek physician) said that men did not withstand fasting as well as women. Western societies have been less preoccupied with promoting thinness in men compared with the relentless sociocultural pressures promoting weight loss directed at women (Zhang, 2014). But throughout most of western history, men have been vitally concerned about body shape, with records from Sparta in ancient Greece documenting the demand for an athletic physique. Today, half of all males wish to increase weight, while almost half wish to decrease weight. Overriding such weight-change goals,

however, for males from the preteen years onward, are increasing demands to attain the "holy grail" of lean muscularity as the only acceptable body physique. David Beckham, Tom Brady, and Brad Pitt are examples of lean muscularity. Higher weight is acceptable if associated with large, defined muscle mass and low body fat. A small minority may desire the boney thinness of Mick Jagger. Soto Ruiz et al. (2015) summarize the current social milieu regarding gender-specific body goals as follows: "Current models of beauty represent an extreme thinness in the women and a muscular body in the men . . . In general women saw themselves as being fatter than [they] really were while men saw themselves as being thinner than they really were."

Strong economic forces present boys and men with relentless images of impossibly fit males showing "washboard abs" and lean muscularity as ideals, resulting in negative self-comparisons in consumers of the media (Hobza et al., 2007; Pritchard and Cramblitt, 2014). The economic goal is to create a negative self-image that can be remedied only by purchasing health, appearance, weight, muscularity, and shape-improving consumer products. Compared with girls and women, who most often desire only thinness, boys and men seek a plurality of physique goals, although lean muscularity is the most vigorously promoted image. The three body image ideals for males with eating disorders are shown in figure 13.1: the very thin, the lean muscular (most common), and the extremely muscled bodybuilder ideal. *Physique anxiety* in otherwise healthy boys and men is common if not pervasive. The ideal of lean muscularity becomes a source of envy (figure 13.2).

In the social learning process, boys learn to perceive themselves as fat, on average, at weights slightly above population norms, whereas women perceive themselves as fat at more than 15%–18% below population norms. This early social learning process takes place largely between the third and fifth grades. After that time, there is a continuing and increasing perception of body fatness in 40%–75% of school-aged girls (Schreiber et al., 1996). The prevalence of actual obesity in grade-school children is lower than that of perceived overweight, despite recent increases in average weights. Adolescent males who go on to develop an eating disorder are most likely to begin their dieting at one or two standard deviations





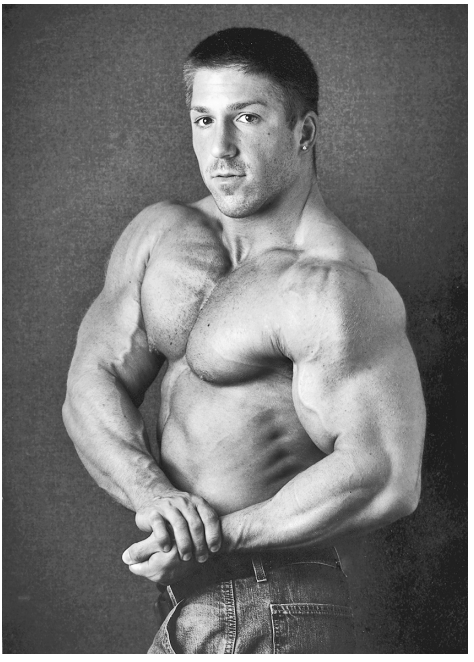


Figure 13.1. *Top left,* Thin ideal. *Top right,* Lean muscular ideal. *Bottom left,* Extreme muscular ideal. istockphoto.com.

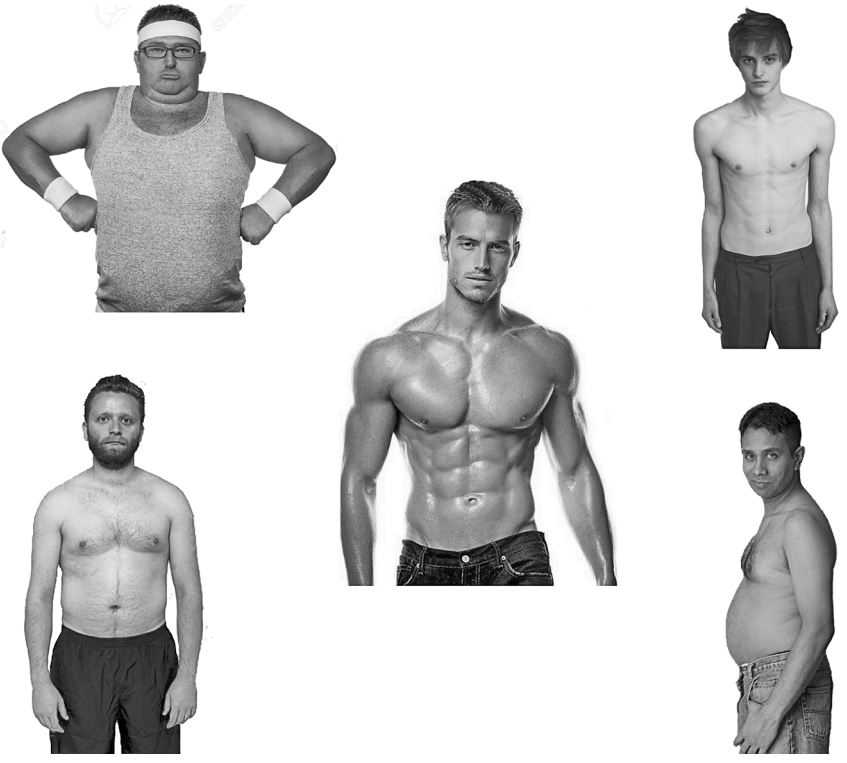


Figure 13.2. The reality versus the ideal physique

above population weight norms (Welch, Ghaderi, and Swenne, 2015), especially if teased or criticized for their weight or if weight and shape change are demanded for sports.

Because there is less general cultural endorsement for boys to diet, they tend to do so only when they are objectively heavier than average and usually in response to one of four specific provocations: (1) to avoid being teased again as they were for childhood obesity; (2) to increase sports performance; (3) to avoid developing weight-related medical illnesses that they have seen in their fathers; and (4) to improve a gay relationship. More than half of male patients with eating disorders at the University of Iowa began dieting for one of these reasons, in contrast to approximately only one-tenth of female patients.

Males with eating disorders fall into three age categories: those with childhood onset before 12 years of age; adolescents, from preteens

through the early twenties; and young men and more mature adults. There is increasing recognition of eating disorders in men at midlife and beyond (Reas and Stedal, 2015). The issues prompting dieting in these three groups differ significantly: very young boys often express family conflict or changes in location or family composition; adolescent males struggle with attaining an individual identity, wanting to develop confidence in sports, dating, and peer recognition; older males may initiate weight or shape change as means of achieving workplace advancement, becoming attractive to new sexual partners, meeting armed services annual review weight criteria, or alleviating weight-related health concerns. Eating disorders have been diagnosed in males from 10 to 60 years of age, even though onset in most cases is in the teens and early twenties.

About 16%–20% of males with eating disorders have a gay orientation, approximately four or five times the average for the population as a whole but still a minority (Fichter and Dasher, 1987). The reason for this higher percentage appears to be the increased valuation of thinness, especially thin muscularity, in the gay culture rather than any intrinsic consequence of sexual orientation. Males with gay orientation who have bulimia are at increased risk for HIV infection due to impulsive, unprotected sexual behavior. Many young males with anorexia are simply asexual.

Prevalence of Eating Disorders in Males

The widely quoted ratio of 10 females to 1 male for individuals with eating disorders is derived from presentations at clinics and hospitals, representing a serious underestimation of eating disorders among males (Sweeting et al., 2015). Population-based surveys are more accurate and typically find a ratio of 3.6:1 (Akgul et al., 2015), and an almost equal prevalence when the female-based diagnostic criteria and assumptions are revised so that attitudes and behaviors specific to males are taken into account. Most recent community-based studies support a ratio of 1:2–3 males to females with some form of eating disorder (Hudson et al., 2007; Woodside et al., 2001). *DSM-5* has eliminated references to amenorrhea in eating disorder diagnoses and now requires a gender-neutral finding of significant restriction of energy intake. Binge eating disorder

afflicts approximately equal numbers of males and females when studies are averaged. A rough estimate for ARFID patients is that males make up about 30%, but many ARFID studies have small numbers of participants.

Diagnosis

There are multiple contributions to the underdiagnosis of eating disorders in men and boys. Men, and society in general, stigmatize males with eating disorders more than females (Griffiths, Mond, Li, et al., 2015; Griffiths, Mond, Murray, et al., 2014; MacLean et al., 2015). In one study, health care providers perceived eating disorders and anabolic-androgenic steroid use less favorably than cocaine use (Yu, Hildebrandt, and Lanzieri, 2015). Diagnostic criteria and standardized testing are normed for females, with some recent improvement.

Clinicians may hold unvoiced assumptions that eating disorders are female disorders. Thus, the first concern in assessing male patients is simply to think of eating disorders as a diagnostic possibility rather than to overlook abnormal eating and preoccupation with body image as normative "guy behavior." For some time, psychiatrists dismissed the possibility of eating disorders in males because boys and men did not fit within archaic psychoanalytic formulations or because they could not manifest amenorrhea. Yet the first report of anorexia in the English language, in the late 1500s, as well as the first modern studies in England by Sir William Gull in 1873 included males.

The diagnosis of an eating disorder in males is made by the same *DSM-5* criteria used for females. The diagnosis is *not made by ruling out all conceivable medical possibilities*. Males with bulimia nervosa who are in the normal or overweight range experience binge eating followed in 85%–90% of cases by purging and in approximately 10%–15% by other forms of compensation, such as overexercise or additional, more severe food restriction. Increasingly recognized by clinicians is the syndrome of binge eating disorder in male patients—binge eating with no purging or any other compensation (Pope et al., 2006). Binge eating disorder occurs primarily, but not exclusively, in older adult males, usually

in their thirties to fifties, and is more common than anorexia nervosa or bulimia nervosa. Most studies find an approximately equal prevalence of binge eating disorder in males and females. It is often intermixed with various degrees of obesity. Male patients with bulimia are more likely than females to have the companion disorders of substance abuse, impulsivity, and antisocial personality features. In contrast, most males who have anorexia with restricting subtype have self-critical, anxious, persevering, perfectionist, or obsessive-compulsive personality features. ARFID patients experience a high probability of comorbid anxiety disorders, above 50%.

Why there are fewer males with eating disorders than females is not a settled issue: whether there are fewer males because males are less exposed than females to social pressures promoting thinness, because they have intrinsic biological protection against eating disorders, because they are simply underdiagnosed, or for other reasons. The media are relentlessly promoting muscularity as a norm for males.

Screening tests are useful to identify individuals with an increased probability of suffering from an eating disorder, to be followed by a full individual clinical evaluation when appropriate. Unfortunately, most of the standardized screening tests for the most common eating disorders are normed on females. We have found that two commonly used tests, the Eating Attitudes Test (EAT) and the Eating Disorders Inventory (EDI), scored males considerably less symptomatic than females despite a matched BMI. Several tests that appreciate the characteristics of males with a higher probability of suffering from an eating disorder are available, but none have achieved universal acceptance. These tests are intermixed with features of body/muscle dysmorphic disorder.

Murray et al. (2017) have advocated for changes to current conceptualizations of eating disorder pathology in males to improve diagnosis and treatment in boys and men. They note that the belief that males with eating disorders are rare and atypical has resulted in a systematic underrepresentation of males in eating disorders research. Additionally, males are likely to be stigmatized and marginalized. Cases of males with eating disorders may be more severe at presentation to clinicians because they come out of the closet later as a result of stigma.

Risk Factors

Males who participate in sports, hobbies, or vocations requiring thinness for appearance or performance, or both, develop disordered eating behaviors more commonly than do other males (Thiel, Gottfried, and Hesse, 1993). About 18% of male high school students who wrestle show some form of disordered eating behavior and thinking during the wrestling season (Oppliger et al., 1993), although most of them improve spontaneously when the season is over. A high percentage of males in wrestling, gymnastics, swimming, track, horse racing, and football are vulnerable to eating disorders, with the desired direction of weight change determined by the nature of the sport. Defensive football players have been increasing in weight year by year. Before stricter regulations were recently put in place in some states, many wrestlers attempted to wrestle in artificially low-weight categories by dieting and dehydration through fluid restriction or diuretics. The deaths of three wrestlers, reported in the media, have highlighted the dangers of rapid and excessive weight loss. Other sports in which primarily males participate, such as bodybuilding and football, may require artificial gains in weight, resulting in the use of anabolic-androgenic steroids. The jury is out on whether the common use of creatinine supplements to increase muscle mass is unhealthy. Male weightlifters are especially subject to muscle dysmorphia (Olivardia, Pope, and Hudson, 2000) and eating disorders (Ruffolo et al., 2006). Adolescent males with gynecomastia are sensitive about chest shape and may initiate dieting to get rid of their breasts (Fisher and Fornari, 1990). Surgical reduction can be successful. Compared to females who develop AN, males are more likely to be objectively overweight prior to their search for thinness, especially those who go on to exhibit binge-purge features.

Eichstadt et al. (2020) have found that athletes with eating disorders may be more difficult to identify and diagnose for various reasons related to differential presentation of symptoms, secretiveness or shame around behavior, and sex-related stigma. Eating disorders in male athletes may lead to serious negative outcomes.

Older men and women are increasingly being recognized as suffering from eating disorders. Mangweth-Matzek and Hoek (2017) have

documented that between 1% and 2% of men older than 40 suffer from eating disorders, but they are generally not in treatment and may suffer even more stigma than younger males.

Reverse Anorexia Nervosa and Muscularity-Driven Eating Disorder

One form of eating disorder occurs almost exclusively in men and boys. Males who think of themselves as never big enough, never muscular enough, even though large and "cut," may suffer from "reverse" anorexia nervosa, considered by some to be a form of body dysmorphic disorder—more specifically, muscle dysmorphia. The literature has blossomed with studies on reverse anorexia nervosa compared with and contrasted with muscle dysmorphia and body dysmorphic disorder (Dingemans et al., 2012). *DSM-5* includes body dysmorphic disorder under the category of obsessive-compulsive and related disorders.

Body dysmorphic disorder is a general diagnosis requiring preoccupation with a perceived defect or flaw in physical appearance that is not observable to others or is considered insignificant by others. Males with large defined muscularity and very low body fat are not adequately included in the body dysmorphic disorder category. *DSM-5* includes muscle dysmorphia as a specifier within body dysmorphic disorder, describing it as a syndrome characterized by a person's preoccupation (predominantly among males) with the idea that "his or her physique" is too small or not sufficiently muscular.

Reverse anorexia nervosa overlaps with but differs from muscle dysmorphia. Muscle dysmorphic disorder, within the category of body dysmorphic disorder, focuses on the inaccurate perception of smallness rather than on the abnormal eating and excessive athletic behaviors of reverse anorexia nervosa, which includes abnormal eating patterns. Muscle dysmorphia does not necessarily include any abnormal eating for a diagnosis. A new diagnostic category within eating disorders that is more mellifluous than reverse anorexia nervosa, and that unites the growing trend toward muscularity as a male ideal along with disordered eating, is the term muscularity-driven eating disorder (MDED).

Males dissatisfied with their body image generally face the divergent paths of becoming thinner or more muscular, or both, with higher body weight often not objectionable if accompanied with low body fat as well as more defined musculature. Ideals for low body fat are often in the range of 5% or lower. Common terms in the popular literature for the drive for muscularity include "getting shredded" and "go big or go home." A combination of bulking and "cutting" methods are used.

Since the early 2000s, numerous publications have focused on the drive for muscularity in males dissatisfied with their body image. In a follow-up survey of 14,891 young adults, 22% of males and 5% of females reported muscularity-oriented disordered eating behavior (Nagata et al., 2019). Scales for muscularity-driven eating disorders are evolving. Clearly, the muscularity ideal is in competition with the thin ideal for males dissatisfied with their body image (Ralph-Nearman and Filik, 2018), with some evidence that the muscularity ideal is winning. Dissatisfaction with body image appears early in life in males. McLean, Wertheim, and Paxton (2018) have found that 32.6% of 6-year-old boys wished to be more muscular, while 20.8% desired being thinner rather than larger and fatter. Klimek et al. (2018) have documented that men who internalized muscularity and thinness were more likely to exhibit both muscle dysmorphia symptoms and disordered eating. In summary, there is growing recognition that eating disorder symptoms associated with muscularity-oriented drives are more common in men than previously understood.

The terms *macromusculophilia* (desire for huge muscles) and *microsomatophobia* (fear of being too small) are more specific for the psychopathology of the condition of MDED—the first term recognizing the strong desire for large muscles in association with a morbid fear of having too small a body. The term *reverse anorexia nervosa* is awkward linguistically but has the advantage of implying that the condition is a mirror image of the common perceptual distortion found in anorexia nervosa of being too large despite being medically starved.

It may be timely to add a category to the formal subtypes of eating disorders experienced by males, in addition to anorexia nervosa, bulimia nervosa, binge eating disorders, and ARFID—muscularity-driven eating disorder. This addition would correspond to a type of disordered eating

found in many males and would eliminate the use of the awkward term reverse anorexia nervosa. MDED would have the requisite components of disordered eating and overvalued beliefs in the benefits of extreme muscularity, in addition to other abnormal behaviors and functional impairment.

Case Study: Male with MDED and Binge Eating

The following brief summary is based on treatment notes for an incarcerated male with MDED who gave permission to share his story anonymously (nonspecific features are modified). The patient was a 30-year-old man serving out a prison sentence for burglary. He was in treatment for anxiety and depression, treated with sertraline, when a request was made to evaluate him for a second opinion. It soon became clear that he was also suffering from reverse anorexia nervosa in the context of defending himself in the prison environment. The patient viewed his body as too small and his muscles as not big enough. He had been teased as a child for being small. In prison he embarked on a program of muscle development and increased weight. He worked out 3–4 hours every other day. He did not engage in cardiovascular activities such as running because of fear of losing weight. His stated goal was "to look huge." "I'm eating crazy." "I just want to be muscular. I don't want to be fat. I want to be huge." Despite his enormous biceps, he believed that "my biceps don't look large enough from my viewpoint." His ideal was "all muscle, no fat, built like a bull or ox or horse." He was preoccupied for much of the day thinking about how to build up his body and about getting enough food, leading to binge eating. He discounted comments that he was muscular and huge. "I've been asking people how big I look—thinking about my body almost all day. Once someone starts being a smart ass and saying I can't get any bigger, then I start thinking I need to do more workout." He lifted up to 700 pounds of weights. "I'm doing 700 pounds halfway, 435 all the way down." "I want to be so big that I'll look intimidating." He continued sertraline and a few cognitive behavioral therapy (CBT) sessions until he was moved to probation status.

Comparison of Diagnosis and Body Dissatisfaction in Males and Females

Starved male and female patients are similar medically with the exception that the male starts with a lower reserve percentage of body fat and a higher percentage of lean muscle mass, allowing him less weight loss before the onset of ketosis from protein breakdown. Males appear to be less attentive to taking vitamins while dieting and may suffer more vitamin deficiency syndromes. In contrast to the occurrence of amenorrhea in females, males have no comparable signal that alerts a family and others to the medical consequences of weight loss. In addition, boys and men who suspect they may have an eating disorder often perceive, accurately, stigma from society, females with eating disorders, health care providers, and peers, and therefore they may be hesitant to discuss this possibility with clinicians. Unfortunately, many professionals also fail to recognize eating disorders in male patients, through either theoretical bias or lack of diagnostic training. Not uncommonly, in our program we frequently treat male patients whose eating disorders were recognized only belatedly because of their denial of illness, who avoided treatment because of their feelings of stigma, or who were refused treatment in other programs simply because they are male.

Men in general are more dissatisfied with their bodies from the waist up, while women are predominantly dissatisfied with their bodies from the waist down. Most men and boys with anorexia find that relentless self-starving does not produce the shape change they desire, despite drastic weight change; they become morbidly afraid of being fat, despite evident thinness, if some body part, especially the abdomen, has not been visibly reduced. There is a strong possibility that the cultural pattern of valuing thinness in women has not been followed by men because, in the early to mid-1980s, the thin, cachectic male became socially identified with possible AIDS.

Males, compared with females, develop within a different social learning experience regarding ideal body weight and shape, have a different hormonal milieu, value different goals in sports, diet for different and more personal reasons, may have differences in comorbid disorders,

TABLE 13.1. Clinical features of males with eating disorders

Males often perceive stigma from self, society, providers, media, and female peers with eating disorders.
Recognition by professionals of an eating disorder in males is less likely than in females.
Risk factors include sports requiring weight loss for performance or appearance, childhood obesity, fear of weight-related illness, gay orientation, being a member of minority groups.
Lean muscularity is the overriding desired body image, but higher weight muscularity with low body fat is an alternative.
Preoccupation with appearance is focused above the waist: abdomen, chest, shoulders, arms.
Screening tests underscore males.
Males have an increased probability of comorbid substance abuse as well as depression, anxiety, and OCD.
Anorexia nervosa presents with low testosterone, LH, and FSH.
Binge eating disorders are as common as in females.
Prevalence is greater than 1:10, closer to 1:2–3 for males for AN and BN.
The ARFID diagnosis in males is associated with multiple origins.
Prognosis is good to equally good for males who are adequately treated as for females.

and experience a different gender role in society. For all these reasons, in diagnosis and treatment, it is of great importance for the clinician to recognize and treat males with eating disorders while respecting the male-specific components. Clinical features of males with eating disorders are summarized in table 13.1. Of course, sociocultural pressures for weight or shape change play on underlying psychological vulnerabilities.

Is an Eating Disorder a Form of Addiction?

The meaning of the term *addiction* has been broadened considerably in recent years. Previously, it referred to a condition of physical dependence with medically recognizable withdrawal features, as well as causing functional impairment, with negative consequences to society. Eating disorders, especially reverse anorexia nervosa and muscle dys-

morphia, do not result in physical dependence in the way that opiate or alcohol use disorders can cause physical dependence. Males who are interrupted in their quest for huge muscularity and low body fat, can, and do, experience psychological withdrawal, sometimes accentuated by the physiologic withdrawal from anabolic-androgenic steroids. The question of what the term *addiction* covers is a semantic one, but the clinical condition is a reality. Interruption of the established behavioral patterns in any of the eating disorder subgroups, especially those that are to some degree ego-syntonic, may well lead to psychological distress and a form of mourning the eating disorder before healthier thinking and new behaviors are well established.

Medical Evaluation and Comorbidity

In the history-taking part of the evaluation, unless the patient has been referred with a diagnosis of an eating disorder, the physician, physician's assistant, or advanced registered nurse practitioner can make the diagnosis in a straightforward manner by appropriate questions as described in chapter 1. It is helpful to have a short set of diagnostic screening questions for each common behavioral disorder and age group, comparable to the CAGE questionnaire for alcohol abuse. A comparable set of screening questions regarding eating disorders is the SCOFF questionnaire or the Screen for Disordered Eating (SDE).

A young male patient's participation in a higher-risk sport that emphasizes thinness for appearance or performance goals should alert the physician to pose diagnostic questions even in a routine physical, as should unexplained low potassium, weight loss of more than 15 pounds, parotid gland swelling (from purging), or nonspecific gastrointestinal discomfort. The primary care physician should ask about changes in eating and exercise history and any changes in the results of the physical examination compared with previous visits. Once the diagnosis of an eating disorder is made, the circumstances around the onset of the weight loss, the binge-purge process, or the binge eating alone should be explored, especially the reasons for the desired weight or shape change, and the emotional needs and drives associated with the eating disorder.

Eating disorders almost always come as "package deals," with one to several comorbid psychiatric diagnoses separate from the eating disorder. Depression is seen in 50%–70% of males with eating disorders, comparable to the number for females. Other diagnoses are more likely in males than in females. At times, the comorbid condition—such as substance abuse, OCD, or impulsive or antisocial personality traits—will determine the long-term prognosis more than the eating disorder itself. If street drugs have been used, their relationship to the eating disorder should be explored; for example, it is not uncommon for patients to snort a line of cocaine to inhibit an urge to binge or to promote further weight loss. The presence and medical consequences of compulsive exercising, including *march hemoglobinuria* (blood in the urine) and stress fractures, should be noted.

The history of the male patient with an eating disorder includes changes in sexual desire and functioning, usually a decrease in sexual drive, sexual fantasies, and masturbation frequency, in proportion to weight loss or frequent binge-purge activity. An awareness of sexual orientation and gender identity will help guide psychological treatments. Physical examination notes the general degree of emaciation and decline in lean muscle mass, the presence of obesity, stigmata of purging, as well as general medical findings including vital signs. Bulimia nervosa is less obvious on physical examination; patients may be secretive about binge-purge behaviors. With obese males, screening questions should be asked to determine whether binge eating disorder is present.

Laboratory studies for male patients, in addition to the general evaluation noted above, include serum testosterone level (both total testosterone and free testosterone) if weight loss is present. Normal absolute testosterone varies typically from 340 to 800 ng/dL, while free testosterone is usually in the range of 1.8–6.8 ng/dL (for ages 20–49). Testosterone declines in proportion to weight loss (Andersen, Wirth, and Strahlman, 1982). LH and FSH correspondingly diminish in anorexia nervosa because the changes in gonadotropins are due to central hypothalamic hypogonadism secondary to starvation, rather than increasing in level as would be expected with a failing gonad. Testicular examination often reveals testes that are decreased in size in anorexia nervosa, comparable to the testicular volume of prepubertal males. If unprotected sex or intra-

venous drug use is disclosed in the history, then a request for HIV testing is appropriate (Ramsay, Catalan, and Gazzard, 1992).

For males with binge eating disorder, the medical complications of the commonly associated obesity should be evaluated through lipid panel testing, with attention to the presence of type 2 diabetes mellitus and hypertension. Any male with anorexic levels of weight loss for more than 6 months should generally be referred for a DEXA scan to assess loss of bone mineral density. Osteoporosis and osteopenia are common in chronic eating disorders (anorexia nervosa and bulimia nervosa). Males, somewhat surprisingly, have even worse deficiencies in bone mineral density than females with eating disorders (Mehler et al., 2008).

Treatment

The treatment of low-weight males with anorexia nervosa is similar to that of underweight females in most regards, while addressing additional, male-specific concerns (Andersen, 1990). The general goals of all eating disorder treatments are as follows:

1. To normalize weight, with a healthy goal weight range of about 4 pounds (e.g., 143–147 pounds rather than a single number). A subset of patients with bulimia nervosa are mildly underweight, and this contributes to binge urges if not corrected. Obese patients with BED often do best with weight loss less than that needed to attain a putative normal BMI. For ARFID patients, attainment of normal age-specific weights is closely tied to the specific medical disorders underlying the weight loss or failure of normal weight gain.
2. To normalize eating behavior by teaching patients how to choose balanced, adequate meals anywhere, anytime, whether by oneself, in a school cafeteria, at a family meal, or at a celebration.
3. To challenge and change the patient's overvaluation of the benefits of weight loss or shape change, using CBT or other evidence-based psychotherapies. Evidence-based, manualized psychotherapy is the core of lasting treatment benefits and

involves helping the individual learn to deal with fundamental issues in living, such as identity formation, mood regulation, and harassment, as well as to gain a sense of control.

4. To reach a decision about whether the comorbid medical and psychiatric diagnoses are secondary (in which case, these conditions—for example, hypothermia—improve on their own) or primary, perhaps predisposing the patient to the disorder (e.g., preexisting depressive disorder), and whether they are long lasting, as in the case of osteoporosis caused by the eating disorder.
5. To provide treatment that prepares males for return to their gender-specific social roles as well as to work out issues in sexuality. Males especially need therapeutic work to more accurately identify and express feelings beyond anger and sexuality. An increasingly recognized truth is that the diagnosis and treatment of males with eating disorders requires an understanding of how the muscularity-oriented body image ideal leads to maladaptive attitudes and eating behaviors (Lavender, Brown, and Murray, 2017).

There is merit to considering physiologic replacement of testosterone for adolescent and adult males who have reached all or most of their axial height. For males with anorexia who have documented low levels of testosterone, there are no definitive studies regarding the specific benefits of testosterone replacement versus allowing hormone levels to return naturally. Studies of testosterone treatment on older males with low testosterone, however, have shown improvements in volumetric bone density and strength in a controlled trial (Snyder et al., 2017). If testosterone replacement is chosen during the weight restoration phase, the typical mode currently used is a topical gel, with a dose of 5 mg per day. Once full weight restoration has been attained, supplemental testosterone is discontinued.

The testosterone level is reassessed one month after attainment of a healthy weight range to determine whether a truly adequate weight has been reached for achievement of the patient's normal hormone production. Because virtually all late adolescent and adult males of normal weight should be producing normal amounts of testosterone, a continued low level usually indicates inadequate weight or persisting testicu-

lar disorder independent of weight. The therapeutic use of testosterone for males with anorexia is based on an extension of testosterone use for underweight patients with AIDS and for burn patients. Caution would be indicated for males who are not yet close to full height and maximal bone growth, because testosterone can cause premature closure of the bony growth plate. The eventual benefits versus risks of this short-term anabolic hormone need to be rigorously studied.

Males with moderate to severe eating disorders are usually treated for most of their inpatient or day hospital stay in our program in mixed-gender groups, with two important additional treatments. Our experience is that males benefit, first, from enhanced strength training, with an emphasis on strength, not appearance, and second, from participation in psychotherapeutic groups for boys and men, with their fathers and male siblings welcome. Ideally, the strength training exercises are guided by a team member trained in exercise physiology, starting "low and slow," building up to moderate levels of resistance, and emphasizing good form to enhance lean muscle development and decrease the tendency for early weight restoration to show prominently in the abdominal area. When restored weight is concentrated in the abdomen, it provokes fears of becoming fat and concerns about repeating the undesired body weight habitus that may have prompted the dieting originally. El Ghoch et al. (2017) have documented by DEXA that while men with anorexia nervosa attain normalization in lean body mass, they show greater deposition of total body fat mass in the truncal region, meaning the concern of males about not showing abdominal obesity is objective, and not only a subjective concern.

A male patient commented on follow-up regarding strength training during his treatment experience: "I must admit one thing, however. I could not have accepted my weight gains without my exercise—both the aerobic exercise and the progressive resistance training. I knew the weight I was gaining was healthy weight. The eating and the exercising complement each other. I ate healthily to exercise, and I exercised to eat healthfully. It was a very symbiotic relationship for me."

Monitoring of liver function and hemoglobin, as well as other laboratory tests, typically continues during testosterone replacement. There

is some concern about long-term testicular deficiency in male patients who have had an eating disorder, a result noted in a study showing that 2 of 11 males who returned to a healthy weight remained oligospermic or azoospermic. The physician should ask the patient about the return of normal sexual drive, fantasy, and sexual function during the weight restoration process. Often the male patient's social behavior and spontaneous discussion of sexually related topics will indicate a return to more normal hormone production. Flirting with staff members and signs of masturbatory activity are signals of improved weight and testosterone production. The addition of testosterone to the weight restoration of male patients with anorexia is not a settled issue and is not mandatory.

Groups for teen boys and young men with their fathers and brothers have, in our experience, proved beneficial to improving the distressed relationships within the family. Teen boys and young men often suffer from alexithymia, not having words for their feelings. One patient, for example, would come home fatigued from a day of work, walk past his father, and go right to his room. The father would angrily come after his son and demand to know why his son was ignoring him and would not stop to talk with him. The son would become angry in return, and a long-standing distressed relationship worsened. After participation in several groups with the patient and his father, the patient learned to come home and say to his father, "Dad, I am tired and worn out from tough customers; I'll spend some time in my room and then we can talk." As a result of disclosing his feeling to his father, the son allowed the father to relate to his son's fatigue and wait till later for a chat. A simple illustrative story we use in father-son-brother groups is that boys stop learning colors beyond those in a small crayon pack they receive in grade school. Their range of colors learned is limited to blue, red, green, yellow, brown, and black. Seldom do they identify mauve, fuchsia, salmon, or teal. The same limitation goes for feelings—anger is quickly learned and all too often may be the only feeling employed. In groups using CBT, sons learn to say: "When I come home tired, I feel tired and a little depressed; I am not sad or ignoring you. I need some space and quiet." Fathers learn to say: "Thank you for sharing your feelings. That makes

sense that you would feel that way. I know you are not ignoring me." Distressed father-son relationships improve regularly.

We often treat servicemen in their forties and fifties who have developed an eating disorder in response to being required to lose weight and to demonstrate increased fitness to pass annual physical performance and medical examinations. They have generally responded well to treatment because their jobs are on the line. Males with eating disorders and alcohol use disorders appear to respond more rapidly to treatment needs when they are required to improve to maintain a job. Building a therapeutic alliance of trust and respect is essential for successful treatment of males with eating disorders.

A group that may be overlooked is long-term incarcerated men who develop MDED to protect themselves from other, predatory prisoners or gangs, as described for one patient earlier in the chapter. Another patient, a 40-year-old incarcerated male, began each day with 900 sit-ups despite knee injuries. He also performed hundreds of push-ups each day to avoid becoming fat from his involuntary intramuscular antipsychotic medications. Some incarcerated men go on hunger strikes to achieve leniency from prison restrictions, attaining anorexic weights.

MacNeil, Hudson, and Leung (2018) have demonstrated that an active program of innovative methods to increase the engagement of males in specialized outpatient assessment and treatment can be successful. A 467% increase of men in assessment and treatment occurred after their outreach. They emphasized a specialized treatment track for men.

Determining a Healthy Weight for Males with AN or BN

In determining a healthy weight, the rule of thumb is to ask the body for the answer rather than an insurance table, although ideal body weights from standard tables may be used for the short-term goal in acute treatment. If the patient was at a stable weight and height for some time prior to the eating disorder and this was not an obese weight, then the preillness weight may be a good target for fully grown men. When an individual has grown taller during an illness of several years, the goal weight may not be obvious. Here, the Metropolitan Life Ideal Body

Weight scales or the mean matched population weight chart may be reasonable initial choices, but final weight range should be modified according to the body's messages that it is functioning in a stable range. Specific signs of a return to a healthy body weight are present when the following conditions are met:

- Full normalization of temperature control with no signs of hypothermia
- Normal patterns of hunger before meals and satiety afterward
- Restoration of normal LH, FSH, testosterone, and leptin levels
- Normalization of TSH level, if low when the individual was ill
- Normalization of T4 if found to be low in the anorexic state due to euthyroid sick syndrome

Continued mental preoccupation with weight and shape is a different issue, not yielding to the simpler measures of weight restoration but usually requiring expert psychotherapy, most clearly accomplished with CBT. As with the return of normal estrogen levels, normal testosterone is a necessary but not sufficient guideline for determining a normal weight range. It is important for patients to understand that body functioning may not be fully normal until healthy behaviors have continued and weight has been stable in a healthy range for 6–9 months or longer after acute weight restoration. A final weight appropriate for age, height, and individual physiology will be worked out during the year after initial treatment is concluded, a year of intensive relapse prevention. The patient needs to be reassured that the disproportionate concentration of some weight in the abdominal area during weight restoration is a natural occurrence; this weight will be redistributed during the year of relapse prevention and psychological growth after acute care.

The male patient with bulimia nervosa suffers medical consequences similar to those for females, with medical symptoms divided between the consequences of weight loss, if present, and the specific problems associated with the particular purging behaviors. Binges themselves are uncomfortable physically and psychologically, triggering urges to purge or otherwise compensate for the excessive calories ingested, but generally have fewer medical complications than the purges (except for the

rare gastric rupture). Hypokalemia with resulting arrhythmias is the most common serious medical complication of purging. Tender, enlarged, parotid glands may be the most obvious physical sign. If a male with bulimia is HIV positive (usually resulting from comorbid substance abuse or unprotected sex, often due to an impulsive personality or bipolar II mood disorder), then appropriate treatment should be offered according to the best recent studies.

When binge eating disorder is diagnosed in an obese male, treatments are directed first toward improvement of the BED through a combination of nutritional guidance, CBT, and often, prescription of an SSRI, typically fluoxetine, sertraline, escitalopram, bupropion, or lisdexamfetamine. Once the binge eating behaviors are improved, the second phase of treatment involves directing attention to the overweight or frank obesity, deciding whether it should be treated because of medical complications or whether the patient should be encouraged to develop overall cardiovascular fitness and attain greater lean muscle mass while accepting a higher weight. Evidence is good that a BMI from 25 to 30, when combined with good cardiovascular fitness, low body fat, and absence of hypertension or diabetes, may be compatible with excellent health and is best accepted, rather than seeking a so-called normal BMI. For males with BED who try to attain a "normal" BMI, the effort may be associated with hypothermia, relentless hunger, and a return to binge eating. Interestingly, in one study, older males with a BMI of less than 22.7 had the highest mortality (Breeze et al., 2006). Simply telling a patient in a weight range with BMI of 26–30 to lose weight is easy for physicians to recommend but much harder to carry out, and sometimes it is not necessary or is unhealthy if fitness goals are otherwise attained.

Recognition of BED is increasingly made by bariatric surgery clinics. Evidence has been accumulating that bariatric surgery appears to be related to later lower all-cause mortality in obese patients, compared to treatment as usual. Most bariatric surgeons will request that an obese male with BED first show cessation of binge activity for 6 to 12 months before being accepted for surgical weight loss.

If weight loss is sought without surgery, the clinician runs into the several problems of trying to trick the body into attaining and, more

importantly, maintaining a lower weight than the body had become accustomed to during the active binge eating disorder. It is sometimes possible to obtain moderate, long-lasting changes in the "set point" of an individual's weight through improved choices of healthy foods, with the emphasis on choosing balanced food groups rather than limiting calories; the addition of moderate, regular exercise; and improvement of stress management. The role of alcohol use, either solitary drinking or in a social context, in disinhibiting eating behavior in males must be explored and discussed. The increased eating associated with alcohol binges (the latter present in 60%–70% of first-year college students) is often passed off as normal behavior but may be a substantial contributor to undesired weight increase.

Finally, males with eating disorders who have had any sustained underweight for 6 months or more should be tested for bone mineral density with a DEXA scan. A clinician is less likely to think of osteoporosis in association with a male patient, but males with anorexia experience more osteoporosis than do females (Andersen, Watson, and Schlechte, 2000). The contributing factors to the pathogenesis of osteoporosis in men appear to include low testosterone and, as in females, diminished calcium intake, lowered body weight, elevated cortisol, and possible other factors. The best current treatments for osteoporosis in male patients include moderate exercise through low-impact weight-bearing activities (avoiding high-impact sports, which increase fracture risk), prompt restoration to normal body weight, restoration of natural production of testosterone, and adequate calcium (1,500 mg/day) and vitamin D (800–1,000 IU/day) intake. (Use of bisphosphonates for osteoporosis in men is not yet validated but may be considered in older males.) A return to normal weight, involvement in low-impact weight-bearing activities, and normal testosterone production are the major contributing factors to improved bone mineral density.

Treatment Outcome: Concerning News and Good News

Richardson and Paslakis (2020) note that men need to be recognized as stakeholders in health care related to eating disorders. Men are often re-

luctant to recognize their eating disorders, face minimization of their symptoms by health professionals, and have difficulty finding treatments tailored to their unique needs and preferences as men. Kinnaird, Norton, and Tchanturia (2018) studied treatment adaptions for men with eating disorders. Males responded best to male-friendly environments that included an increased focus on muscularity and methods for males to learn to express a wide range of emotions, emphasizing the availability of male-only groups and male clinicians.

The prognosis for males with anorexia nervosa is the same as or better than that for females (Strober et al., 2006). Any beliefs that maleness is by itself an adverse risk factor for recovery from eating disorders need to be updated by evidence-based studies supporting the fact that males have an excellent prognosis if treated promptly and fully. Individuals with eating disorders can be better off after remission through successful treatment than if they had never had the disorder (although it is a tough way to go) because they will have learned to deal more effectively with the sociocultural forces that overvalue weight and shape changes.

Fichter and colleagues have contributed substantially to understanding the long-term outcome of males treated for AN and BN, studying gender-matched samples (Strobel et al., 2019). Males with AN had a similar rate of remission (40% of males vs. 41%of females). On follow-up for AN, there was no BED, and obesity was rare. Remission rates for BN were similar: 44% of males and 50% of females. Males with AN scored lower than females at follow-up on most subscales of the Eating Disorders Inventory. Lower scores on standardized testing has also been documented by Smith et al. (2016). Males with BN scored lower on perfectionism and higher on interpersonal distrust at follow-up. Researchers also documented that being male and having AN increased the risk of premature death (Fichter et al., 2020). Males with AN died sooner after onset of their eating disorder than males with BN or EDNOS (eating disorders not otherwise specified; Quadflieg et al., 2019).

The possible role of feminism in the lack of recognition of males with eating disorders and for the paucity of specific treatment foci for males with eating disorders is complex. In groups of females with eating disorders, the admission of males may be threatening, with the risk that

they become lightning rods for feelings of the girls and women toward males who have abused them in the past. Males may be seen as intruding into a safe space for women in treatment. Treatment for groups of patients that includes both males and females needs to be addressed up front as an issue to be worked through with benefit for both genders. We have found that allocating an important minority of total therapeutic work to male-only and female-only subgroups has led to an improved understanding of all relationships, present and past. An important goal for all treatment programs is to decrease the perceived atypicality of males with eating disorders and to make the treatment environment more male friendly (Kinnaird et al., 2019).

Some pearls of wisdom and tips about the diagnosis and treatment of eating disorders in male patients are listed in table 13.2.

Summary

Men and boys develop eating disorders more frequently than was previously believed, in a ratio of approximately one male to two or three females. Many fewer seek medical care, however, indicating multiple roadblocks for males—especially shame, clinicians' failure to recognize these disorders in male patients, nonacceptance in treatment facilities, assumptions on the part of caregivers of sexual orientation, and fear of being treated like females. Males with eating disorders have a plurality of body image choices compared with females: their options, in general, are to attain extreme thinness, become lean but muscular, or to be hugely muscular. The most common desire of males with disordered eating behaviors is to achieve lean muscularity, with minimal body fat and favoring upper body development. If lean muscularity is not chosen, then huge muscularity or extreme thinness are the next most commonly sought goals. The media have increasingly presented body image ideals for men that are as impossible to achieve as the starved images are for women. The socially and commercially mandated ideal physique for men has become more intensely promoted since about 1990. Males are more likely to develop AN for specific personal reasons rather than a sociocultural endorsement of slimness for all men. Some of the more common

TABLE 13.2. Clinical pearls of wisdom for the diagnosis and treatment of eating disorders in males

Boys and men tend to feel fat only at weights above population norms, whereas women generally feel fat even when objectively thin.
Males tend to be more concerned with shape than with weight and predominantly want to change their body waist-up rather than waist-down. The most frequently desired body image is lean muscularity; the less common alternatives are huge muscularity or extreme thinness.
Males more often diet for specific, personal reasons rather than because of a general cultural endorsement of thinness.
Older males are not exempt from developing any eating disorder. They often desire a change in weight or shape to meet vocational standards or to develop new relationships.
Males with eating disorders are less well recognized by clinicians, who may not think of the diagnosis for male patients. They also are underrecognized because of their self-stigmatization, leading to reluctance to seek diagnosis or to accept treatment.
While not a proven or necessary therapy, short-term replacement of testosterone during the weight restoration phase for males with anorexia, especially for those at or close to maximal height, merits consideration.
Although a gay orientation increases the risk for an eating disorder because of stringent sociocultural body image ideals, most males with eating disorders are not gay or bisexual. Many young males with anorexia are asexual. Minority groups are especially vulnerable.
Males with eating disorders develop osteoporosis as frequently as females and with even greater severity, most likely tied to a combination of the gender-specific contributing factor of low testosterone, as well as the gender-shared contributions of low calcium intake, low body weight, elevated cortisol, and excessive consumption of diet sodas.
Males especially benefit from resistance training exercises during weight restoration; these exercises improve morale and provide a sense that the individual is making a personal contribution toward a desired body shape, resulting in an increased percentage of lean muscle mass and less abdominal weight deposition, helping to decrease relapse, and providing a setting in which to work out with other males.
Males may have reverse anorexia, in which they exercise relentlessly, eat abnormally, and attempt to increase their body weight because of the perception that they are never big enough, even though they may objectively be very muscular with extreme definition. In contrast to the more common drive for thinness, these males experience a drive for great muscularity. A newer term for reverse anorexia nervosa is muscularity-driven eating disorder (MDED).
Males with MDED are liable to use anabolic-androgenic steroids to attain the "bigness" they desire: extreme muscularity with little body fat. This syndrome overlaps with muscle dysmorphia, which does not have disordered eating in the diagnostic criteria.
Males with diminished bone density should avoid high-impact sports activities until their bone density improves. After attaining a healthy weight, abstaining from binge-purge behaviors, and developing a healthy attitude toward food, weight, and shape for several months, they can return to their chosen sports. Some males need to avoid wrestling, figure skating, modeling, or other activities that promote weight loss if they continue with a high drive for thinness.
Wherever possible, it is beneficial to include male-specific components in an eating disorders program. These components include "guys-only" groups (comparable to women-only rape and assault survivor groups), consideration of testosterone replacement, strength training programs, and individual therapy that addresses issues of gender orientation, decisions about sports and vocational goals, and, especially, improved relationships with fathers and peers, as well as return to a chosen gendered lifestyle.

reasons for developing eating disorders in boys and men are to avoid being teased for obesity as they were in childhood, to improve sports performance, to avoid medical illnesses, to improve a gay relationship, and for older men, to develop new relationships or to attain workplace goals. Males are often more concerned with shape than weight. Males in higher socioeconomic levels are generally very avoidant of any trace of obesity.

Screening tests used to identify males with eating disorders often underscore or omit male symptoms. A brief informed clinical evaluation suffices. Diagnosis is similar for male and female patients with anorexia nervosa or bulimia nervosa, but generally only males develop reverse anorexia, a condition in which fear of smallness dominates despite huge muscular development, predisposing these patients to use of anabolic-androgenic steroids and to disordered eating. A new category to replace reverse anorexia nervosa is muscularity-driven eating disorder. Specific terms to describe the psychopathology of males attempting to achieve overly defined muscularity with low body fat, despite perceptions of being still too small and underdeveloped, are macromusculophilia and microsomatophobia: a strong desire for huge muscles associated with a fear of having a small body. Treatment of males with eating disorders requires an appreciation of male concerns, including establishing support groups for male patients, striving for improved relationships with fathers, implementing strength training, and in selected individuals, using testosterone supplementation for those with low weight. Gay males are about five times more likely to develop an eating disorder than the general population, but they still constitute a minority of cases. Sexual minority males have a higher-than-average vulnerability to developing eating disorders. Male patients have a prognosis as good as or better than that for females. Steps to address prevention of eating disorders in males are essential but often overlooked even more than for females (Cohn et al., 2016).

Nine More Truths about Eating Disorders: Boys and Men

The Academy for Eating Disorders has recently published a helpful set of summary points about males with eating disorders, entitled "Nine

More Truths about Eating Disorders: Boys and Men," produced in collaboration with Jason M. Lavender, PhD.

1. The eating disorders field historically developed through a female-centric lens.
2. Boys and men with eating disorders remain underrecognized and underserved.
3. Eating disorders can, but do not necessarily, present differently in boys and men than in girls and women.
4. Medical complications of eating disorders in boys and men can be severe, and mortality is elevated.
5. Seeking treatment for an eating disorder can be especially challenging for boys and men.
6. Boys and men can face additional eating disorder treatment barriers.
7. Eating disorders can affect boys and men across the lifespan.
8. Eating disorders can affect cisgender and transgender boys and men of all sexual orientations, races/ethnicities, cultures, socioeconomic backgrounds, and body shapes and weights.
9. Body ideal in boys and men are diverse and can be influenced by many factors.

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14

Special Considerations for Eating Disorders in Children and Young Adolescents

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COMMON QUESTIONS

What is the epidemiology of eating disorders in children and young adolescents?

When evaluating children and young adolescents, what are some of the differences in screening questions, presenting symptoms, and differential diagnosis compared to older adolescents and adults?

What should providers include in the medical assessment when they are concerned a child or young adolescent may have an eating disorder?

What medical complications are less likely to exist at presentation in younger patients?

Do children have the same risk of refeeding syndrome as adults?

What are the unique effects of eating disorders on growth and development in young adolescents?

What are common presentations of ARFID in children and young adolescents and how should the medical provider approach the evaluation of these patients?

How and when should providers have conversations with parents if there is a concern that the eating disorder can be affecting long-term bone health or final adult height?

How does understanding risk to final height and bone density affect the approach to child and adolescent patients before, during, and after treatment?

How are goal weights set in growing children?
How can providers be reassured that children and young adolescents in treatment are on track for their genetic growth potential?
What clinical issues are important during maintenance of remission for young adolescents?
When can young adolescents be discharged from the care of a multidisciplinary eating disorders team?
What do we currently know about preventing eating disorders in children and young adolescents?
What can professionals and advocates do to help children and young adolescents get into care early and prevent irreversible medical complications?

Case Study 1

A.J. was an 11-year-old cisgendered male who presented with acute weight loss over a 3-month period. His parents noted at presentation that he had always been active and loved playing sports. Prior to the last three months, he had always eaten a good volume and variety of food. His sudden decrease in intake was concerning, and their traditional parenting methods did nothing to reverse his behaviors. His parents shared that he had always been a well-behaved child, and they were confused by his new behaviors. They had found him hiding food, throwing away food, and exercising in secret. He complained of abdominal pain and feeling cold.

On his initial evaluation, he had lost 14% of his body weight in 3 months, and his growth had slowed. He had been at the 75th percentile for height and weight prior to his behavior changes. At presentation, he was 10th percentile for weight and 65th percentile for height. He had lanugo hair growth and bradycardia on his exam.

He was admitted to a local children's hospital for acute stabilization, where he required phosphorous supplementation during initial refeeding. He transferred to a residential facility, where he restored to the 70th percentile for weight. His linear growth had not resumed, however, and he was only at the 60th percentile for height at time of

discharge. A dual-energy X-ray absorptiometry (DEXA) scan was done, and his whole-body Z-score was –1.6, and his spine Z-score was –0.8.

His midparental height predicted that he should reach the 75th percentile for his final adult height. He received ongoing support from his parents as well as from an outpatient team that included nutrition, therapy, psychiatry, and medical providers. Team goals were to coordinate care, return the patient to school full time, manage his psychiatric comorbidities, and help him reengage in sports and other extracurricular activities. His team also wanted him to reach his genetic potential for final adult height with the best possible bone density.

Although his eating disorder thoughts decreased and his hunger and fullness cues improved over the first 3 years following intensive treatment, he had inadequate appetite drive to meet his total energy needs for his rapid growth and age-appropriate activity. He required ongoing meal support from his parents and dietitian, with close monitoring of his weight and height from his medical provider. He was able to maintain at the 75th percentile for weight while growing and restore back to the 75th percentile for height in the first 3 years of his recovery.

Case Study 2

B.F. was a 12-year-old cisgendered female who presented to her local children's hospital for weight loss and bradycardia. Her presenting BMI Z-score was –2.94. A DEXA done on admission showed a whole-body Z-score of –1.8 and a spine Z-score of –0.9. She was medically stabilized and transferred to a residential facility, where she was treated for anorexia nervosa. Despite stepping directly into an intensive outpatient program (IOP) with psychiatric and medical support, she struggled to complete meals upon returning home.

She continued to complain of significant gastrointestinal symptoms, including bloating and urgency, that limited her intake. She was unable to eat many foods in her meal plan and was noted to have lack of variety in her diet, including limited intake of fruits, vegetables, and dairy. Due to these issues, she had poor weight gain and slow growth over the next several months.

Her outpatient treatment team, consisting of a therapist, dietician, psychiatrist, and pediatrician, continued to be concerned about her lack of improvement and felt that a diagnosis of avoidant/restrictive food intake disorder (ARFID) was more appropriate for her eating issues. On reflection, family and patient endorsed long-term challenges with volume and variety that predated the acute weight loss and emergent medical admission. Her "picky eating" was thought to have started during her toddlerhood.

She was evaluated and diagnosed with autism spectrum disorder and generalized anxiety disorder. Despite outpatient treatment, volume and variety behaviors remained entrenched issues and she continued to have poor linear growth. Patient and family chose to admit to an intensive partial hospitalization program (PHP) for ARFID, with a goal of improving her volume and variety of intake.

Despite these interventions, she finished growth approximately 4 inches shorter than predicted by midparental height. It was felt that her slow growth prior to initial treatment likely represented most of her height deficiency.

Background

It is a widely held tenet of pediatric medicine that "children are not little adults." In the medicine of eating disorders care, however, we have generally thought of the medical complications of eating disorders similarly in children as in adults. We have trained providers to look for abnormal vital signs and lab findings as markers of medical risk and indicators of illness severity and directions for treatment.

In the past 20 years, there have been significant treatment advances for children and young adolescents with eating disorders. The use of family-based treatment (FBT) has freed families and providers from the agonizing thought that families cause eating disorders. Parents can now be successfully incorporated into treatment for eating disorders, as we do with other common pediatric illnesses.

During this same time, however, there has been a significant shift in the demographics of eating disorders. The average age of onset has de-

creased. In addition, it is now understood that children and young adolescents are not immune from any of the *DSM-5* eating disorders diagnoses, including anorexia nervosa, atypical anorexia nervosa, ARFID, bulimia nervosa, and binge eating disorder. This shift has brought new medical concerns and new treatment considerations as younger patients are now being affected by these life-threatening illnesses.

Restrictive behaviors, such as energy restriction or overexercise, may be present in any of the eating disorders in children and young adolescents. These behaviors can lead to significant medical complications, including malnutrition and cardiovascular collapse. The Society for Adolescent Health and Medicine (SAHM) has recognized the impact of restrictive eating disorders in children and young adolescents and advocated that the medical provider play an important role in the multidisciplinary team (Golden, Katzman, Sawyer, et al., 2015a).

When eating disorders were initially conceptualized as an illness of older teens and adults, it was imperative that our primary medical concern was the recognition of life-threatening medical complications, including the risk of refeeding syndrome. *While assessing for medical instability should never be minimized, additional considerations must be made in the evaluation and treatment of children and young adolescents*.

Our youngest patients have a different way to handle the energy insufficiency that accompanies restrictive eating disorders. Children and young adolescents are growing. When faced with inadequate energy, their bodies may not immediately turn to catabolism and downregulation of critical organ systems. Instead, our youngest patients will stop gaining weight and *eventually stop growing*. In addition to loss of potential height growth, long-term development of bone density and the brain may be irreversibly affected. New research supports these critical observations and is changing the way care should be discussed and conceptualized for children and young adolescents.

This chapter will review special considerations for eating disorders in children and young adolescents and review suggested management skills for working with young patients and their families, making treatment decisions, and monitoring for successful treatment outcomes.

Epidemiology

Eating disorders are common in children and adolescents, with an estimated prevalence rate of 6% to 8% in adolescents across studies (Galmiche et al., 2019). Over the past several years, there has been a worrisome trend of eating disorders affecting increasingly younger patients (Favaro et al., 2009; Madden et al., 2009). The median age of onset is now 12.3 years of age for anorexia nervosa (AN) and 12.4 years of age for bulimia nervosa (BN) (Swanson et al., 2011). In addition, younger patients are more likely to have co-occurring mental health disorders, and males and minority patients with eating disorders are being increasingly recognized in these younger age groups (Golden, Katzman, Sawyer, et al., 2015b). In fact, in younger patients, males may represent one-fourth of the patients with eating disorders (Madden et al., 2009). Eating disorders have been described across ethnicities and socioeconomic groups in studies looking at prevalence in adolescence (Swanson et al., 2011).

New challenges come with identifying increasing numbers of younger patients with eating disorders. In the past, the diagnosis of an eating disorder may have been missed in a younger patient. As the medical complications of these disorders may present differently in children than in adults, this can create possible delays in diagnosis. *In these younger patients, medical concerns related to restriction prevail*. Changes in the *DSM-5* have helped to remove barriers (such as loss of menses) that may have hindered accurately diagnosing younger patients accurately with eating disorders. Further, additions to the *DSM-5* have led to more accurate diagnosis of eating disorders that seem to be prevalent in younger patients, especially avoidant/restrictive food intake disorder and atypical anorexia nervosa.

Patients with ARFID make up 5% to 23% of patients presenting to pediatric tertiary care eating disorders programs (Golden, Katzman, Sawyer, et al., 2015b). Patients with ARFID are distinguished from the other restrictive eating disorders in that although they have restricted intake, they report no shape or weight concerns. These patients may present with a lack of interest in eating, an avoidance of foods based on sensory characteristics, or an avoidance of food related to aversive consequences.

Adolescent patients with atypical anorexia nervosa now represent one-quarter of the total admissions for acute medical stabilization for anorexia (Peebles et al., 2017). (This increasingly concerning illness is reviewed at length in chapter 15.) Providers will need to remain vigilant for atypical anorexia and be as attentive in the future to the percentage of weight lost as they have been to the absolute presenting weight in the past.

Binge eating disorder (BED) may present during childhood or early adolescence; however, onset is more common during late adolescence. As with patients with bulimia nervosa, these patients are less likely to have medical complications that are unique to younger patients. Both BN and BED have medical complications that are directly related to duration and frequency of the behaviors. Until these behaviors are chronic, medical complications may not be readily apparent in these patients, and thus less apparent in younger patients newly engaging in these behaviors. These serious medical complications are well described elsewhere, and lack of medical findings does not diminish the need for treatment for these illnesses. Because energy deficiency is not a primary feature of the illness, these patients are less likely to present with growth or development issues unless a comorbid medical condition is present.

Presentations of Eating Disorders in Children and Young Adolescents

Diagnosing an eating disorder in a child or young adolescent may be challenging because of the differences in presentation from that in an adult. Although children and adolescents can present with similar medical concerns and symptoms as older adolescents and adults, they may present instead with concerns regarding failure to achieve appropriate increases in weight or height or delayed or interrupted puberty. These may be less obviously associated with energy insufficiency. Commonly used lists of medical symptoms of eating disorders often do not include concerns about growth or puberty—yet these are common presenting concerns in children and young adolescents and must be remembered in the evaluation of younger patients. These patients present additional barriers to diagnosis in that they may have difficulty describing medical

symptoms, and commonly used screening tools are not readily applicable to younger patients.

Most providers who treat eating disorders are familiar with the SCOFF questionnaire and other eating disorder screening tools such as Eating Disorders Examination Questionnaire (EDE-Q). For children and adolescents, the American Academy of Pediatrics (AAP) recommends a much simpler set of questions based on developmental stages (Hagan, Shaw, and Duncan, 2017). These questions are approachable for young children, such as "How do you feel about how you look?" and "What concerns do you have about your weight?" Young patients should not have concerns about their shape or weight.

The Bright Futures Guidelines were designed by the AAP to help providers screen for critical issues in a developmentally appropriate way at routine health examinations. Because eating concerns figure prominently even at young ages, the AAP has included questions starting at the 9-year-old checkup that may help providers detect an eating disorder early. These questions screen not only for risk of body image–based eating disorders such as anorexia nervosa and bulimia nervosa, but also for issues with volume and variety that may be seen in patients with ARFID (table 14.1). Providers should remember, however, that even with anorexia nervosa, younger children may present with less concerns about body image than older patients.

When an eating disorder may be present, providers will need to approach obtaining a history from a younger patient with some unique features in mind. First, some behaviors that are associated with eating disorders in older patients may have not occurred to a younger patient. Rome suggests that when "asking questions about these behaviors, motivational interviewing skills may help to avoid the development of new behaviors" (Rome and Strandjord, 2016). Additionally, younger patients may be limited in their ability to describe their emotions and may lack self-awareness of their behaviors. Interviewing parents or other caregivers may be crucial in identifying behavior patterns that illustrate the presence of a covert eating disorder.

Engaging in an exhaustive medical evaluation before diagnosing an eating disorder is not necessary and may in fact delay definitive diag-

TABLE 14.1. American Academy of Pediatrics *Bright Futures* questions for eating concerns

Middle Childhood

Ages 7 and 8

Ask the Parent

- What do you think of your child's weight and growth over the past year?
- What concerns, if any, do you have about your child's eating, such as getting him to drink enough milk and eat vegetables and fruits?
- How often do you eat together as a family?

Ask the Child

- Do you drink milk? What vegetables and fruits do you eat?
- How many times a week do you eat breakfast?

Ages 9 and 10

Ask the Parent

- Do you have any concerns about your child's weight?
- Do you have any concerns about her eating behaviors or food intake, such as getting her to drink enough milk and eat vegetables and fruits, or hearing her talk about dieting?
- How often do you have a family meal together?

Ask the Child

- What concerns do you have about your weight? How do you feel about how you look?
- How often have you cut back on how much you eat or tried a diet to lose weight?
- What vegetables and fruits did you eat yesterday?
- Did you eat breakfast this morning?

Adolescents

Ages 11 to 21

Ask the Youth

- How do you feel about the way you look?
- Do you feel that you weigh too little? Weigh too much? Just right?
- How much would you like to weigh?
- Are you teased about your weight?
- Are you doing anything to change your weight? What kinds of things are you doing?

Ask the Parent

- Do you have any questions or concerns about your child's nutrition, weight, or physical activity?
- Does he talk about getting fat or dieting to lose weight?

Source: Hagan, Shaw, and Duncan, 2017.

nosis of the disorder and the initiation of treatment. This may occur more often in younger patients as providers, and parents, may not at first be able to see the presenting concern as a consequence of disordered eating behaviors. When any clinical suspicion exists for an eating disorder in a younger patient, further evaluation for an eating disorder, not a medical condition, should take place.

The Medical Evaluation of Children and Young Adolescents

The evaluation of children and young adolescents suspected of having an eating disorder relies on a history and physical exam directed with an eye toward the most common findings of eating disorders with a few significant differences. As in adults, providers should expeditiously *rule in* an eating disorder, rather than undergo a lengthy rule-out evaluation. As emphasized by SAHM in 2015, "Timely evaluations are of critical importance as shorter duration of illness is associated with improved outcomes in children and young adolescents" (Golden, Katzman, Sawyer, et al., 2015a).

All children and young adolescents with a suspected eating disorder will need an excellent history and physical by a medical provider with attention to a few important additions to the history and physical of older patients. Providers should obtain an accurate timeline of concerns and take note of all changes in eating habits, especially when the changes observed in the child differ from those of the rest of the family. In the social history, all children should be asked about bullying, especially regarding weight, as this is often identified as a factor in eating disorder development in young patients. An important aspect of the medical history in a young patient includes questions about growth and puberty. In addition, growth and puberty in family members should be reviewed. Genetics plays an important role in determining growth and pubertal timing, and medical providers can be aware early on of signs that a patient may have deviated from expected familial norms.

A medical history for eating disorders has unique challenges in children and young adolescents. Younger patients may have a limited ability to convey concerns. Children and young adolescents may lack words or experience to describe some of the symptoms providers regularly see in eating disorder patients. Issues like slow transit constipation and functional gastroparesis secondary to weight loss may present as vague abdominal complaints or discomfort. Food may erroneously be thought to be the culprit, and often elimination diets may have been previously attempted. With restrictive eating disorders, because children often stop gaining weight rather than lose weight as an adult would do, it may not be clear that the

inadequate intake is the primary event, and the medical concerns are secondary. Questions about medical symptoms in younger patients should be explored in an age-appropriate way. For very young patients, drawing can be a helpful form of self-expression during a medical visit.

When an eating disorder is suspected, providers must ensure a preexisting medical condition does not exist. Yet, extensive or ongoing medical evaluations may fuel parental concerns that something medical is being missed, and this reinforcement may make it more difficult for the family to eventually accept an eating disorder diagnosis. This can lead to delays in diagnosis and initiation of care.

Food fears or food refusals are common in younger patients and should be explored by a comprehensive history and physical as well as a broad medical and psychiatric differential diagnosis. Abdominal pain is a common and nonspecific complaint. With younger patients, a history of a choking episode or an allergic reaction requiring medical attention may be elucidated.

In general, evaluations for food allergies or gastrointestinal conditions should be done only for specific medical concerns. Patients with food allergies typically present with a history of early anaphylactic food reactions or will have a narrow range of food avoidance but overall good intake. Patients with eosinophilic esophagitis may present with a fear of choking but will also describe episodes of choking or swallowing difficulties. The symptoms of celiac disease can be harder to define in younger patients. Those with a suspected food allergy, eosinophilic esophagitis, or celiac disease and a possible eating disorder should not be presumptively placed on an elimination diet but instead should be referred for definitive testing. Acceptable testing methods include skin prick testing for food allergies and endoscopy with biopsies for eosinophilic esophagitis and celiac disease. *No young patient should be advised to eliminate foods from their diet without subspecialist consultation and definitive testing*. Eliminating even one food in a growing child can affect growth.

At the time of the initial exam, all children and young adolescents will need an assessment for puberty in addition to the weight, height, orthostatic vital signs, and temperature typically obtained in the evaluation of all patients with eating disorders. Growth and puberty are closely

linked, and both can be affected by malnutrition. Assessing adequacy of growth and puberty in children and young adolescents can be complex and is addressed further in the following sections.

Pubertal staging is important, as timing of the onset of the eating disorder may overlap with critical junctures of puberty and growth. Pubertal staging should be done by assigning a sexual maturity rating, formerly known as Tanner staging, comprising five stages of development. Girls typically begin puberty between the ages of 8 and 13 years and have peak height velocity starting after pubic hair stage 2. Menarche occurs late in puberty, and the average age of menarche is currently 12 to 12.5 years. Boys typically begin puberty a bit later, between the ages of 9 and 14 years, and peak height velocity occurs after attainment of pubic hair stage 3 (Wolf and Long, 2016). These critical developmental milestones overlap directly with the average age of onset of anorexia nervosa and bulimia nervosa and place children and young adolescents with eating disorders at risk for unique, and potentially irreversible, medical complications related to growth and puberty.

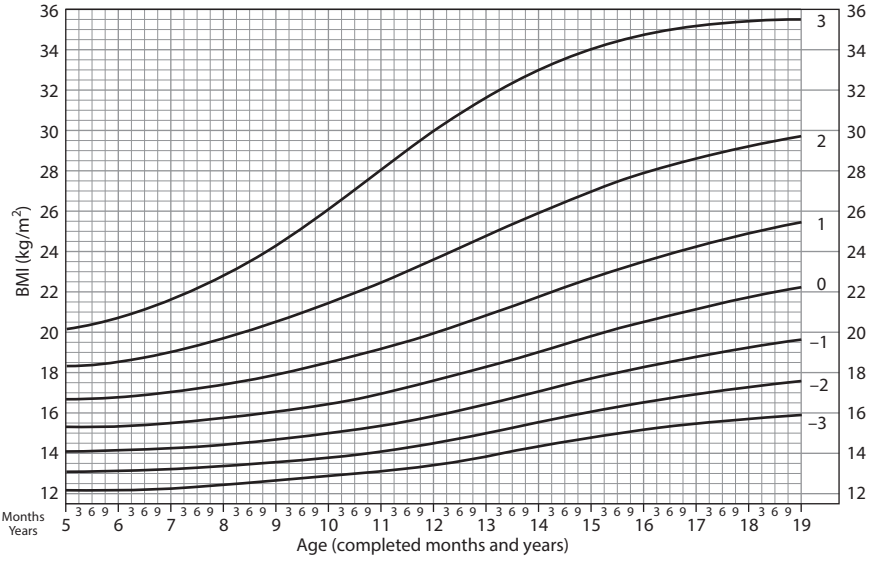
Malnutrition can delay or halt puberty. Bone age X-rays and hormone levels may be useful determinates when malnutrition has seemingly impaired growth or pubertal development as noted by history or exam. Providers should remember that growth and puberty are 99% complete at a bone age of 17 years. Since luteinizing hormone (LH) and follicle-stimulating hormone (FSH) trigger sex hormone production in the ovaries or testes, measuring these levels along with estradiol or testosterone may be useful. Adolescents with delayed puberty and an eating disorder may have hypothalamic hypogonadism, and low to normal LH and FSH levels.

Assessment of illness severity by evaluating appropriateness of weight and height for age in children and young adolescents can be complicated. Absolute numerical BMI alone is often not an adequate measure of severity of malnutrition in pediatric and adolescent patients, as it sometimes is in adults, predominantly because of the changes in rate of weight gain and linear growth seen during childhood. Degree and rate of weight suppression is important in all patients with restrictive eating disorders, but particularly so in growing children and adolescents. During times of growth, inadequate energy consumption may first present as lack of additional weight gain. *In young patients with eating disorders, failure to gain weight as expected should be considered weight suppression*. Additionally, providers

should remember that height and weight normally continue to increase throughout childhood and adolescence until age 20 years. Thus, providers may need to look at additional measures to gather a more comprehensive picture of the impact of the eating disorder on a younger patient.

Current recommendations from the AAP, the American Society for Parenteral and Enteral Nutrition, and the Academy of Nutrition and Dietetics are that the degree of malnutrition in children and adolescents should be initially assessed by a combined evaluation of percentage weight loss, percentage median BMI, BMI Z-score, and rate of weight loss (Golden, Katzman, Sawyer, et al., 2015b). Percentage weight loss is calculated simply as (initial weight – weight lost) / initial weight. Percentage median BMI (%mBMI) evaluates a patient's weight status as a percentage of the expected median BMI for adolescents of a similar age and sex. Percentage median BMI is calculated as (current BMI / 50th percentile BMI for age, gender, and height) × 100 (Le Grange et al., 2012). BMI Z-scores can be obtained from charts available from the Centers for Disease Control and Prevention (CDC) at https://www.cdc.gov/growthcharts/zscore.htm or from the World Health Organization (WHO) at https://www.who.int/tools/growth-reference-data-for-5to19-years/indicators/bmi-for-age (figure 14.1). In addition, many electronic medical record programs now calculate BMI Z-scores for all child and adolescent patients. Rate of weight loss refers to the amount of time over which weight has been lost and should be captured in the initial history. At times, prior medical records and growth charts will demonstrate weight loss over a longer period than patients or families recall.

The Society for Adolescent Health and Medicine now recommends that for children and young adolescents, terms such as ideal, expected, or mean body weight no longer be used. Instead, SAHM recommends that all younger patients with restrictive eating disorders be categorized as having mild, moderate, or severe malnutrition (Golden, Katzman, Sawyer, et al., 2015a). These proposed criteria utilize evaluation of percentage of mBMI, BMI Z-score, and percentage of weight loss (table 14.2). Pediatric patients meet criteria for mild, moderate, or severe malnutrition based on meeting any *one* of the three criteria. Thus, a pediatric patient with a percentage mBMI less than 70%, a BMI Z-score less than –3, or greater than 20% body mass loss in one year (or 10% body mass loss in 6 months) would



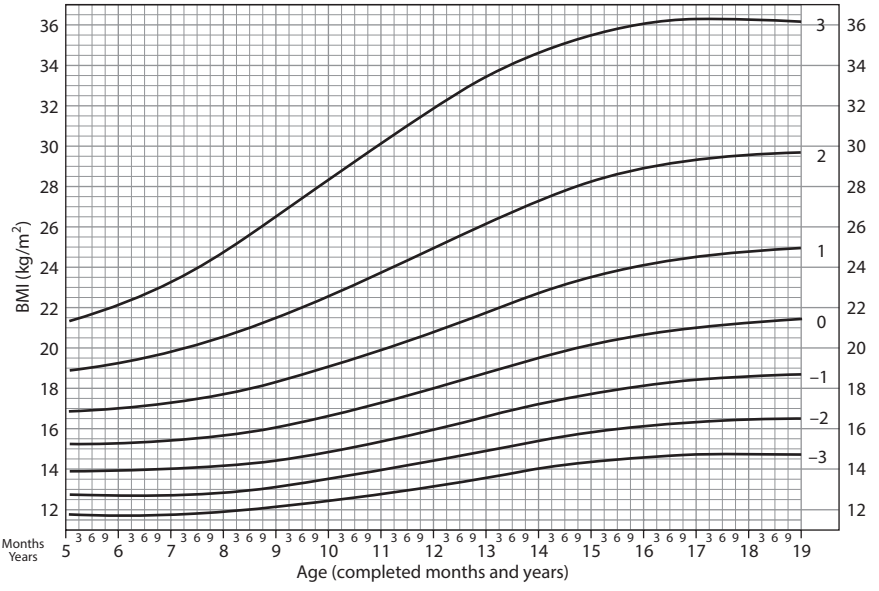


Figure 14.1. WHO BMI Z-scores: BMI-for-age boys 5 to 19 years (*top*) and BMI-for-age girls 5 to 19 years (*bottom*). World Health Organization; https://www.who.int/tools/growth-reference-data-for-5to19-years/indicators/bmi-for-age.

TABLE 14.2. SAHM proposed classification for degree of malnutrition

	Mild	Moderate	Severe
% median BMI	80%–90%	70%–79%	<70%
BMI Z-score	–1 to –1.9	–2 to –2.9	–3 or greater
Weight loss	>10% body mass loss	>15% body mass loss	>20% body mass loss in 1 year or >10% body mass loss in 6 months

Source: Golden, Katzman, Sawyer, et al., 2015.

Note: BMI = body mass index. One or more of the above-mentioned classifications would suggest mild, moderate, or severe malnutrition.

be classified as having severe malnutrition. These recommendations may more appropriately capture severity of malnutrition in children and young adolescents across the spectrum of restrictive eating disorders.

Children Should Be Growing

The primary focus of annual physical exams in children and adolescents is to track growth. Children grow in predictable ways. Although teaching resources have long espoused the importance of monitoring for growth disruptions as a sign of an eating disorder, this is often not obvious initially at the time of a well child exam. Many providers do not expect to see eating disorders in children and young adolescents. They may also be less comfortable recognizing eating disorders in males, as well as in patients of all races, ethnicities, and socioeconomic groups. Additionally, there may be less familiarity with newer diagnoses such as ARFID and atypical anorexia nervosa.

Normal growth in children follows predictable patterns. In general, after age 3 or 4, children will track along the same percentile curve for height and the same percentile curve for weight until growth is complete. Growth is charted for children and adolescents ages 2 to 20 in the United States on the 2000 CDC growth charts, available at https://www.cdc.gov/growthcharts/clinical_charts.htm. These charts are also recommended by the American Society for Parenteral and Enteral Nutrition for monitoring linear growth and weight gain (figure 14.2).

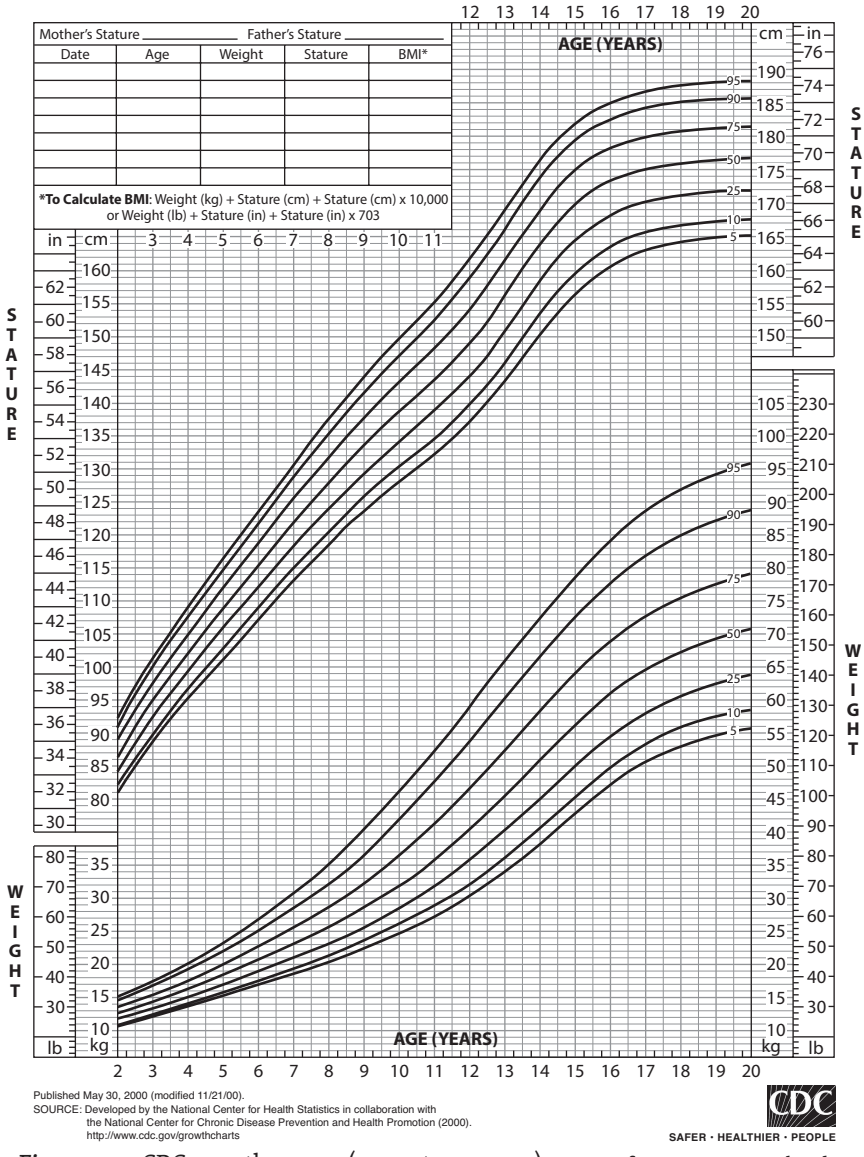
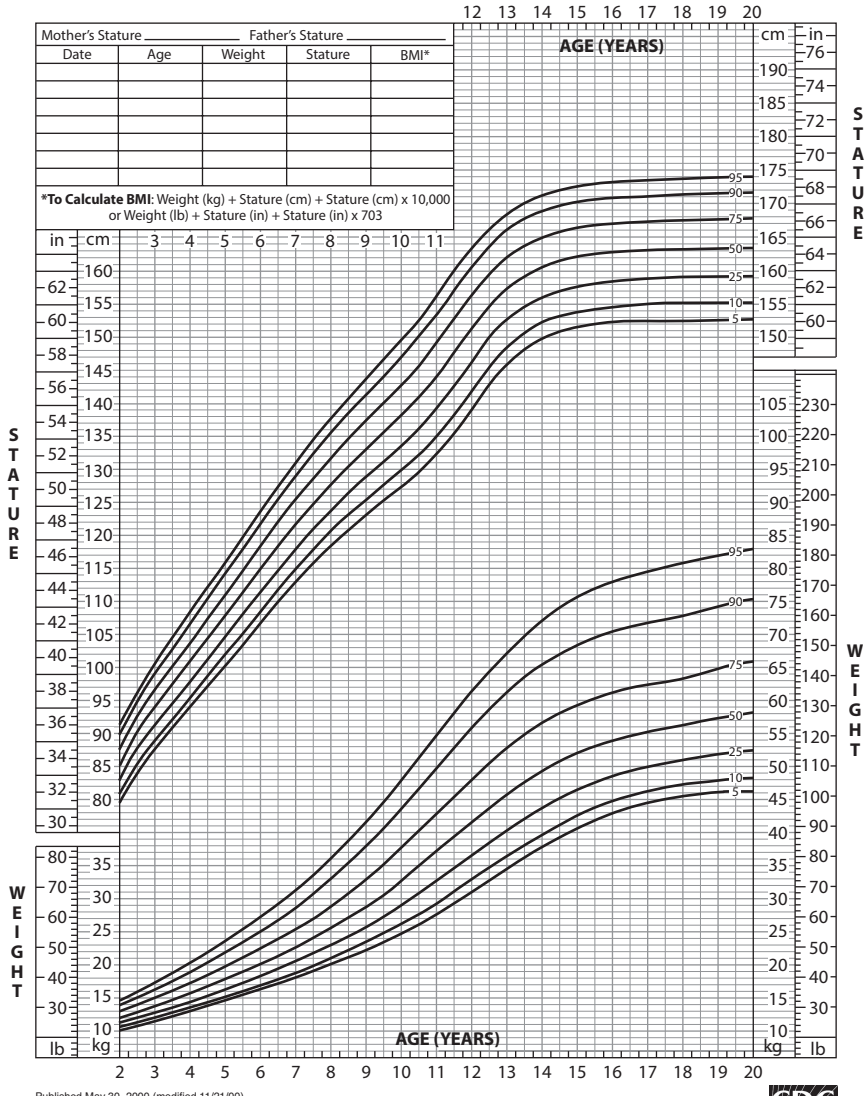


Figure 14.2. CDC growth curves (ages 2 to 20 years). Centers for Disease Control and Prevention; https://www.cdc.gov/growthcharts/clinical_charts.htm.

2 to 20 years: Girls
Stature-for-age Weight-for-age percentiles

NAME _____

RECORD # _____



Published May 30, 2000 (modified 11/21/00).

SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000). http://www.cdc.gov/growthcharts

SAFER · HEALTHIER · PEOPLE

Growth is tightly regulated, and deviations from the normal expected patterns is often "the first indication of an underlying disorder" (Weintraub, 2011). In growing children and young adolescents, shifts across 2 or more percentiles are uncommon and should be a cause for concern. In malnourished children, a percentile drop is first seen on the weight curve and, if sustained, can be followed by a percentile drop on the height curve. It is important to note that shifts across curves may occur with a slowing or cessation of weight gain; that is, *children may not present with overt weight loss*. In children and young adolescents with acute restrictive eating disorders, this pattern of a drop in weight percentile followed by a drop in height percentile is commonly observed (figure 14.3).

Patients with ARFID may be at particular risk for growth issues. These children often have dietary intakes that are limited in volume and variety beginning at an early age, at times starting in infancy or toddlerhood. Very little research exists to date on the impact of nutrition on growth in children with ARFID; however, research in children with chronic diseases has shown that limited dietary intake in chronic illnesses can affect nutrition status and cause growth failure (Sevilla, 2017). Many children with ARFID have had a failure-to-thrive work-up or have been evaluated by pediatric specialists prior to seeking eating disorders care because of this known association. If ARFID is not identified as the causative factor for slow growth, the slow growth will persist and may be assumed to be normative for this patient and family.

Genetics plays an important part in determining height potential and pubertal timing for children and adolescents. Medical providers should understand the critical importance of obtaining a family growth and pubertal history during the initial history and physical exam in all pediatric and adolescent patients with a confirmed or suspected eating disorder. Parents often present worried about the growth of their child. Sometimes parents have concerns that a younger sibling has passed the patient in growth or puberty, or that the child is not growing as the parent did at that developmental stage.

One way to approximate genetic potential for growth is to look at the midparental height (MPH). All children and young adolescents at

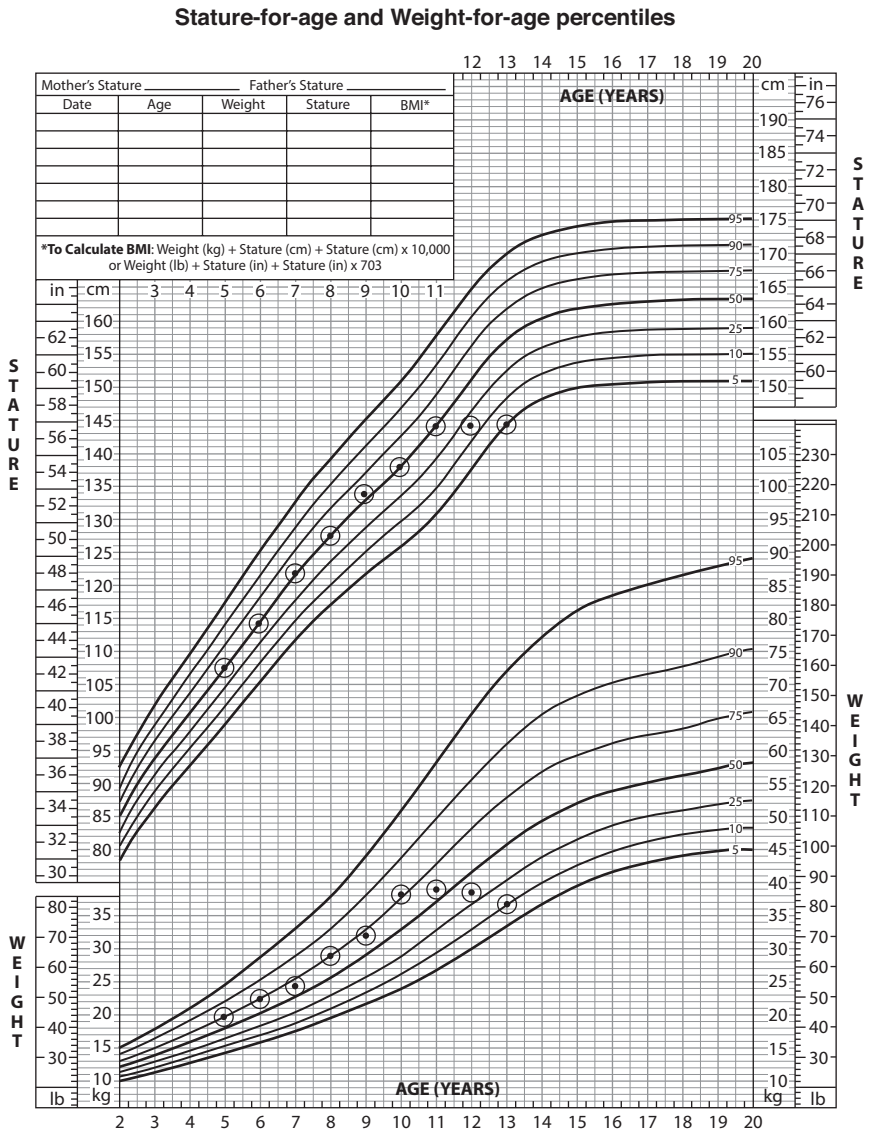


Figure 14.3. Example of patient with growth cessation following weight gain failure. Note no weight gain from age 10 to 11 years old, followed by no height gain from age 11 to 12 years old.

presentation should have growth charts reviewed and MPH calculated. To determine midparental height:

- MPH for girls = (father's height [in.] + mother's height [in.] – 5) / 2
  Or (father's height [cm] + mother's height [cm] – 13) / 2
- MPH for boys = (father's height [in.] + mother's height [in.] + 5) / 2
  Or (father's height [cm] + mother's height [cm] + 13) / 2
- Predicted range +/– 2 inches (5 cm)

For example, for a cisgendered male patient with a mother 5 feet 4 inches and a father 6 feet 1 inches, the MPH would be 5 feet 11 inches (+/– 2 inches). This would place the patient at a predicated adult height percentile of about 75%. If this patient was being examined with a suspicion for anorexia nervosa and his height growth had slowed from the 75th to the 30th percentile in the 18 months before his diagnosis, a goal would be to get him back on track as quickly as possible to attempt to reach the 75th height percentile.

For patients with ARFID and chronic slow growth, however, being off track for linear growth may be less obvious at first review of the growth charts. For patients with significant genetic height potential, growing along any curve that does not align with predicted midparental height, even if weight gain and linear height is steady, may lead to a final height that is inadequate and a reflection of long-term malnutrition. For example, a male with ARFID who grew along the 25th percentile his entire childhood since age 3 years but had a midparental height at the 75th percentile may not be recognized as being off track for growth until later and may not have adequate time for catch-up growth. Providers must remember that children and adolescents follow familial patterns for growth.

Many providers wait too long to reassess patients when the only presenting concern is lack of weight or height gain. A one-year interval between checkups is standard but can lead to significant delays in getting growth back on track. When a variance in growth is detected, this should be discussed and reviewed with the patient and family. If an eating disorder is the probable cause of growth concerns, it should be evaluated expeditiously. While no current firm recommendations exist,

we recommend closer follow-up over a period of months rather than annually for a few years, when growth is at risk. Growth curves that demonstrate a deviation from the expected pattern can be used as a tool in motivational interviewing with patients and their families hesitant to consider an eating disorder diagnosis or treatment. The primary treatment goal should always be to restore normal growth.

Adolescents Should Be in Puberty

Pubertal development, like growth, requires complex nutritional adequacy. Children and young adolescents with eating disorders may present with a slowing or cessation of puberty, as well as a slowing or cessation of growth. At the time of the initial eating disorder evaluation, all pediatric and adolescent patients should always be assessed for pubertal staging using the sexual maturity rating scale. It is important to determine the impact on pubertal development due to the eating disorder, as well as to determine risk for detrimental impact for long-term growth. Growth is influenced by the sex hormones of puberty, and the timing of the growth spurt occurs during specific pubertal and hormonal milestones previously discussed. The pubertal growth spurt is responsible for nearly 20% of final adult height (Weintraub, 2011).

Patients, parents, and providers must understand that when a restrictive eating disorder is present, recovery from altered puberty can be variable. Providers and families may believe that patients have extended time to grow because puberty has stalled. When patients achieve weight restoration and puberty resumes, however, the pace may be brisk, and patients may not have the full amount of time usually allotted for growth and development. This can lead to irreversible effects on final adult height.

Additional issues may arise with the resumption of puberty. Some patients may struggle with feelings around maturation or wellness. Pragmatically, providers should also be prepared to provide support for bothersome issues such as acne and menstrual cramps, which can be more severe when puberty moves quickly. Common therapies for these issues are usually helpful and decrease anxiety and distress regarding puberty.

Providers are familiar with the need to assess menstrual status in adolescent females who can menstruate. Providers taking care of adolescents with testicles must remember that testosterone levels can be decreased in restrictive eating disorders and assess for symptoms of low testosterone. For some patients, decreased hair growth or decreased libido can be worrisome symptoms. Discussing the relationship of these symptoms to the restriction may be beneficial in motivational interviewing and may be useful for monitoring when in recovery.

Patients who are gender diverse or present with body dysphoria due to gender identification will need additional considerations. Older teens report higher rates of eating disorder behaviors compared to cisgendered peers (Connolly et al., 2016); however, there is scant literature regarding children and younger adolescents who also have co-occurring eating disorders. With nutritional rehabilitation, secondary sex characteristics will present and progress. Support around these secondary sex characteristics will need to be provided and may need to be addressed with additional team members, such as with gender identity specialists, in addition to the eating disorders team. Providers should strive to approach patients with these medical concerns in a respectful and inclusive environment and remain mindful that research and medical recommendations continue to evolve.

Impact of Eating Disorders on Growth and Development

In children and young adolescents with eating disorders, there may be an irreversible effect on growth. A meta-analysis by Neale et al. (2020) reviewed 27 studies of growth in eating disorders. Both growth delays and pubertal delays are commonly reported in eating disorder patients at presentation. Factors that may contribute to this include hypothyroxinemia, hypercortisolemia, hypogonadism, and growth hormone resistance. The authors recommend that weight restoration be encouraged to avoid adverse effects and allow an opportunity to catch up.

Although catch-up growth is seen in some patients, it is not seen in all. In one study, female patients hospitalized with anorexia nervosa

were found to have normal heights prior to illness onset. Yet, these patients were found to have lower than expected heights at admission, discharge, and final adult height. The authors concluded that while "weight restoration is associated with catch-up growth, complete catch-up is often not achieved" (Modan-Moses, Yaroslavsky, Pinhas-Hamiel, et al., 2021).

Pubertal status at onset and diagnosis of the eating disorder plays a significant role in the impact of restrictive eating disorders on height. For adolescents, puberty, the growth spurt, and, if possible, menarche are each tied to nutritional status. If nutrition is inadequate, pubertal development slows, linear height is stunted, and menarche is delayed, with the effect on linear growth more dramatic for patients presenting earlier in their pubertal development (Swenne, 2008). Modan-Moses, Yaroslavsky, Kochavi, et al. (2012) described more compromise to height in female patients who were under 13 years old or less than one year after menarche compared to those adolescents who had already progressed further into puberty and their growth spurts prior to onset of malnutrition. Swenne (2005) found that catch-up growth in premenarchal girls is possible, but that weight gain must start before catch-up growth can begin. Postmenarchal females have largely completed growth, and restrictive eating disorders will have less impact on final adult height.

Stunting of growth is also seen in adolescent males with eating disorders, and the risk to final adult height seems also to be closely tied to pubertal stage. Adolescents with testicles (or on testosterone replacement) have their growth spurt at a later pubertal stage, which is generally represented as a later age. While this can give males in recovery from an early onset eating disorder additional time to grow in recovery, and thus additional opportunity for linear height growth, it may have a significantly negative impact if the eating disorder starts or continues into late puberty. This is borne out in the literature, as Swenne (2013) found that prepubertal boys with weight gain were able to return to their premorbid growth curve within 3 years; however, pubertal males may not have catch-up growth even with weight restoration.

Therefore, overall, younger patients are at more risk of irreversible loss of linear height. Because these patients continue to age, and once

nutritionally restored, puberty can progress faster to catch up, they are at significant risk of missing the opportunity for adequate linear growth if there are delays in diagnosing and treating the eating disorder. Modan-Moses et al.'s data showing that younger age at admission was a risk factor for poor catch-up growth in patients with eating disorders led to their conclusion that their "findings emphasize the importance of early detection and initiation of treatment of AN in adolescents." They further highlight the need for "continuous and longstanding weight gain over an extended period to achieve catch-up growth up to the full potential, and to prevent irreversible tissue damage with long-term implications" (Modan-Moses, Yaroslavsky, Pinhas-Hamiel, et al., 2021).

These studies emphasize the need for providers to help families understand the critical importance of getting back on track with growth and development as quickly as possible. Pubertal staging and time left for gains in linear height must be evaluated, considered, and discussed with patients and families as part of the medical evaluation for all children and young adolescents with eating disorders. These discussions may inform choices about treatment pace and intensity.

Additionally, growing children and adolescents will need ongoing evaluation to monitor progress with puberty and growth. Patients and families may struggle with the ongoing weight gain that accompanies growth and pubertal development. When eating disorder thoughts are present, an ever-moving weight target may be terrifying. Motivational interviewing to help patients and parents understand normal growth and development and stay on track may be a useful tool in recovery. With intensive treatment and close monitoring, excellent growth is possible (figure 14.4).

Considerations at Presentation

Considerations regarding growth and development are critical in children and adolescents with known or suspected eating disorders. In older adolescents and adults, the body does not have the option to slow or halt growth when faced with energy insufficiency; thus as starvation progresses, medical complications are seen across organ systems. Even

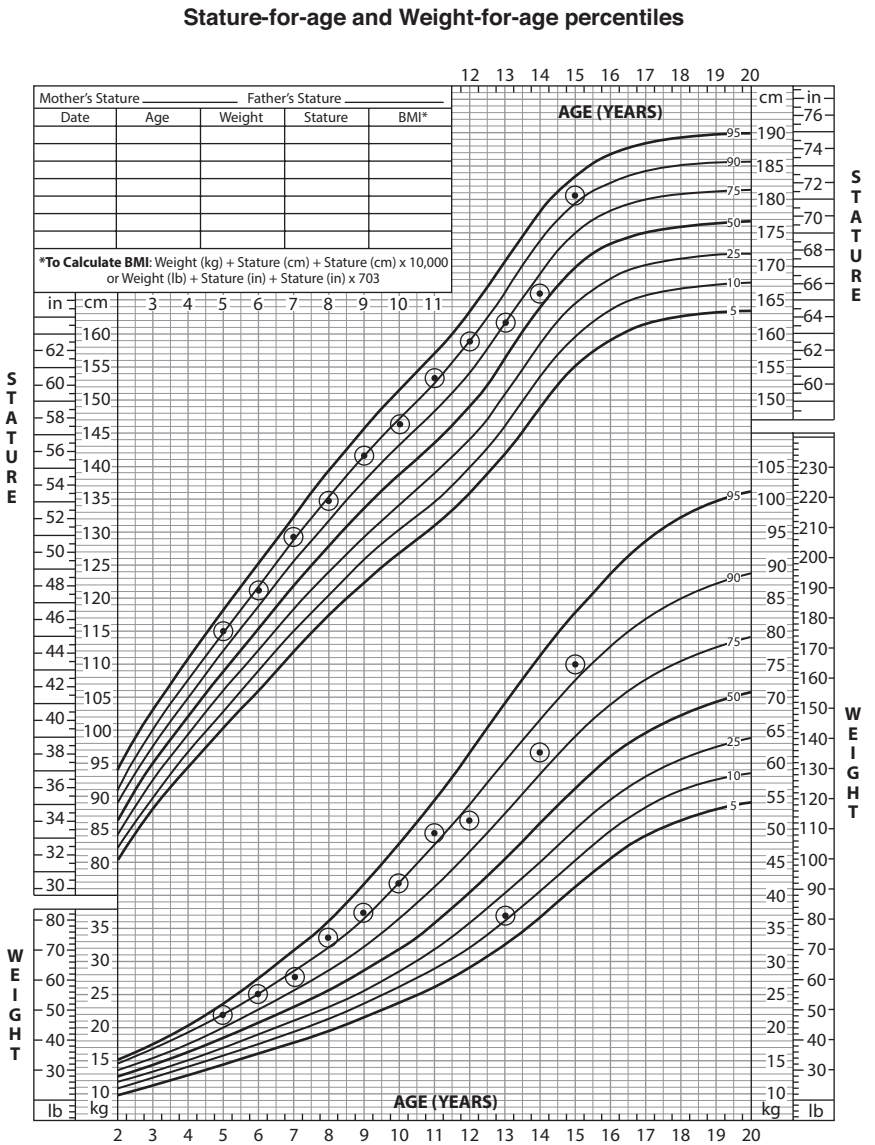


Figure 14.4. Example of weight gain needed to drive height gains in puberty

then, at presentation, normal labs and normal vital signs do not mean that the patient is not sick.

In children and young adolescents, this logic must be taken one step further. When growth is not complete, the body can first slow growth or

halt puberty to save energy. *Waiting to see signs of organ compromise in a patient who should be growing is a mistake*, as catabolism and down-regulation of organ systems are often the last resort for energy consumption.

In a 2003 position paper, SAHM recognized unique considerations when treating younger patients with eating disorders: "Because of the potentially irreversible effects of an eating disorder on physical, psychological, and emotional growth and development in adolescents, the high mortality and the evidence suggesting improved outcome with early treatment, the threshold for intervention in adolescents should be lower than in adults" (Golden, Katzman, Kreipe, et al., 2003).

Medical Intervention in Children and Young Adolescents

Beyond evaluations of growth and assessments of pubertal status, the medical evaluation process in children and young adolescents should include attention to the same physical exam findings and laboratory evaluations of eating disorders as in adults. Additionally, as in adults, the medical evaluation process may reveal significantly abnormal findings, and these may require medical admission for acute medical stabilization.

The AAP and SAHM have published criteria supporting hospitalization of adolescents with eating disorders (Golden, Katzman, Kreipe, et al., 2003; AAP, Committee on Adolescence, 2003). Although initially written based on the diagnosis of anorexia nervosa or bulimia nervosa, the criteria are now focused on identifying children and adolescents who are at risk for serious medical and psychiatric complications with any eating disorder (table 14.3). Of note, the most recent AAP and SAHM criteria include not only vital sign, EKG, and laboratory abnormalities, but also arrested growth and development, acute food refusal, and failure of outpatient treatment (Hornberger, Lane, and the Committee on Adolescence, 2021; Golden, Katzman, Sawyer, et al., 2015a). While there is no absolute BMI cutoff in children and adolescents, those below 75% median BMI for age and gender with eating disorders should be hospitalized. These additional considerations acknowledge the importance of

TABLE 14.3. SAHM indications supporting hospitalization in an adolescent with an eating disorder (2015)

One or more of the following:
1. <75% median body mass index for age and sex
2. Dehydration
3. Electrolyte disturbance (hypokalemia, hyponatremia, hypophosphatemia)
4. EKG abnormalities (e.g., prolonged QTc or severe bradycardia)
5. Physiologic instability ▪ Severe bradycardia (HR <50 beats/minute daytime, <45 beats/minute night) ▪ Hypotension (BP <90/45 mm Hg) ▪ Hypothermia (body temperature <96 F, 35.6 C) ▪ Orthostatic increase in pulse (>20 beats/min) or decrease in blood pressure (>20 mm Hg systolic or 10 mm Hg diastolic)
6. Arrested growth and development
7. Failure of outpatient treatment
8. Acute food refusal
9. Uncontrollable binging and purging
10. Acute medical complications of malnutrition (e.g., syncope, seizures, cardiac failure, pancreatitis, and so forth)
11. Comorbid psychiatric or medical condition that prohibits or limits appropriate outpatient treatment (e.g., severe depression, suicidal ideation, obsessive-compulsive disorder, type 1 diabetes mellitus)

Source: Golden, Katzman, Sawyer, et al., 2015.

considering long-term health risks (some potentially irreversible), available local measures, and family circumstances when evaluating children and adolescents. Often, the decision is multifactorial, but at times severe medical complications such as bradycardia will necessitate medical admission.

The primary goal of medical hospitalization of a child or adolescent with an eating disorder is initial medical stabilization through nutritional rehabilitation and support with interruption of eating disorder behaviors. In addition to initiating correction of the malnourished state, hospitalization should be used for promotion of healthy eating and

weight gain if indicated, evaluation for psychiatric comorbidities, and management of medical complications.

Medical Complications of Restrictive Eating Disorders in Children and Young Adolescents

As with older adolescents and adults, every organ system may be affected by starvation in children and young adolescent patients with eating disorders. Younger patients may have the same cardiovascular, gastrointestinal, hematologic, and metabolic complications from starvation as older patients. But younger patients are at risk for some unique medical complications. As described earlier, children and young adolescents with eating disorders may experience effects on growth and puberty. Assessing degree of weight suppression is critical as it is associated with significant cardiovascular risk, yet it may be more difficult to detect in growing children. Younger patients with eating disorders have additional unique and potentially irreversible concerns that include bone health, neurologic complications, and developmental issues.

In growing and developing children and young adolescents, severity of weight suppression can be difficult to detect initially but is an important determinate of medical risk. In adolescents, total weight loss and recency of weight loss, but not presenting weight itself, were associated with more physical complications. In particular, bradycardia and lower pulse rate nadirs have been shown to be associated with severity of weight suppression. To account for changes in height and to represent changes more fully across ages and sexes, however, changes in BMI Z-scores rather than absolute percentage weight lost may be a better indicator of weight suppression in children and young adolescents (Whitelaw, Lee, et al., 2018; Garber, 2018). Adolescents who go into their linear growth spurts at higher weights and then lose weight while still growing may be at particularly high risk. Providers should consider monitoring changes in BMI Z-scores closely in all children and adolescents, particularly those with weight loss, and should monitor closely for the vital sign changes associated with starvation.

Factors that influence adolescent bone health include body weight and hormone status, thus making adolescents with eating disorders at risk for decreased bone health. Reductions in bone mineral density have been described in female and male adolescents with eating disorders (Misra, Golden, and Katzman, 2016). In summary, energy insufficiency causes down-regulation of the hypothalamic-pituitary-gonadal axis. The resulting hypogonadism contributes to decreased bone accrual. Additional factors include deficiency of insulin-like growth factor 1 (IGF-1), leptin, and growth hormone, as well as excess glucocorticoids. Weight restoration and resumption of menses have been thought to be factors to improve bone health.

Younger patients with eating disorders may be more vulnerable to long-term impacts on bone health due to onset of the eating disorder overlapping with an important window for bone accrual. Adolescence is a critical time for bone deposition, with 90% of bone mass accrued by age 18. Very high rates of bone mass acquisition occur during the growth spurt. For females, peak bone mineral accretion occurs at age 12.5 years and for males at 14 years (Golden, Abrams, and the Committee on Nutrition, 2014). Overlap of an active restrictive eating disorder during this critical window can have significant and irreversible impact.

Bone health should be assessed in all children and young adolescents presenting with concerns for a possible eating disorder. A fracture history should always be obtained but may not fully reflect bone health. Measurement of bone mass is often used to assess bone health and can be measured by DEXA (Golden, Abrams, and the Committee on Nutrition, 2014). For children and adolescents, measurements should be read for the lumbar spine and whole body, not including the head. Hip measurements are not reliable in younger patients. Z-scores, rather than T-scores, should be used to interpret results as Z-scores reflect standard deviations from age-matched means (Bachrach and Gordon, 2016). In children and younger adolescents, there is some lack of consensus on definitions and terminology. Often, the diagnosis of osteopenia is not currently used, and the diagnosis of osteoporosis is made when both low Z-scores and a fracture history are present.

Maximizing bone health and determining which patients with eating disorders are at risk of bone loss or failure to gain bone mineralization should be a critical task for all medical providers caring for children and adolescents with eating disorders. The AAP has a broad recommendation to order DEXA studies in any children or adolescents with significant fractures or with medical conditions associated with increased fracture risk (Golden, Abrams, and the Committee on Nutrition, 2014). Prior studies have shown long-term fracture risk following the diagnosis of an eating disorder, especially in adults over 20 years old (Vestergaard et al., 2002). Recommendations for use of DEXA studies in patients with eating disorders continue to emphasize the diagnosis of anorexia nervosa and presence of 6 months or more of amenorrhea (Rome and Strandjord, 2016; Bachrach and Gordon, 2016). As our understanding of the impact on bone in younger patients with eating disorders improves, future recommendations on timing of DEXA studies may change.

In the past, bone density loss in eating disorders patients was thought to be fully reversible. But recent studies have shown that adolescent patients may have permanent losses. Adolescence is a critical window for bone formation. Bone mass is not accrued during the eating disorder illness, and adolescent patients may not have sufficient time to catch up. Recent studies have shown persistent negative effects on bone health despite recovery of body weight in female adolescents with anorexia nervosa (Mumford et al., 2019). Low bone mineral density has also been shown in underweight patients with ARFID (Alberts et al., 2020). Bone health risk will need to be studied as providers manage a wider variety of eating disorders across a greater range of ages, genders, and races.

Current recommendations for maximizing bone health in adolescents with eating disorders include weight restoration and normalization of hormone function (Misra, Golden and Katzman, 2016). In addition, providers should help adolescents ensure optimal calcium and vitamin D intake. For adolescents, calcium intake should be 1,300 mg/day of elemental calcium (Hornberger, Lane, and the Committee on Adolescence, 2021). The AAP recommends that vitamin D adequacy be assessed with a serum 25-hydroxy vitamin D level. Deficiency is defined as a level less than 20 ng/ml, and insufficiency is levels from 21 to 29 ng/ml. Children

and adolescents with levels less than 20 should be treated with vitamin D2 or D3, 50,000 IU weekly or 2,000 IU daily for 6 to 8 weeks. Per the AAP, once levels are above 20 ng/ml, patients can often be maintained on 600 to 1,000 IU daily (Golden, Abrams, and the Committee on Nutrition, 2014). Providers routinely caring for adolescents with eating disorders will often repeat vitamin D levels to ensure that they remain over 30 ng/ml and continue supplements of vitamin D at 1,000 or 2,000 IU units a day while patients are in eating disorders care. Compliance issues and seasonal effects can cause variations in vitamin D levels. Because of the critical importance of vitamin D on bone, providers may want to ensure that their patients sustain adequate levels to maximize bone health.

The efficacy and safety of FDA-approved treatments for osteoporosis have not been established in pediatric patients (Bachrach and Gordon, 2016). Pediatric literature continues to stress the importance of weight gain and resumption of menses. Although used in adults, bisphosphonates are not generally recommended in children because of potential adverse effects. Biphosphates are released from bone long after medication discontinuation, and studies are lacking on the long-term safety of these medications in children. At this time, the use of bisphosphonates is recommended only in select conditions with recurrent fractures, vertebral collapse, or severe pain.

Additionally, the general use of oral contraceptives is not recommended for adolescent female patients. Although some literature has shown less loss of bone mineral density in older adolescents on oral contraceptives, this effect is not completely protective, as patients still had decreases in bone mineral density (Maimoun et al., 2019). Other studies have shown that oral contraceptives do not increase bone mass in adolescents with anorexia nervosa or female athlete triad. With adolescents, providers must also remember that regular menses on oral contraceptives may provide false reassurance or mask true illness severity (Golden, Abrams, and the Committee on Nutrition, 2014). Newer studies on physiologic estradiol replacement delivered as the estradiol patch may show promise (Misra, Golden, and Katzman, 2016). Providers should continue to stress normalization of weight with resumption of normal hormonal function in adolescents with eating disorders.

Neurologic and developmental concerns represent unique complications that must also be considered in children and adolescents with eating disorders. The cortical atrophy that has been described in adult patients may have a more significant impact on adolescents. In a meta-analysis of 29 studies, adolescents with anorexia nervosa had a significantly larger reduction in gray matter than adults. Gray matter loss is correlated with cognitive deficits (Seitz, Herpertz-Dahlmann, and Konrad, 2016). No current standards exist for routine assessment of white and gray matter in young patients, and long-term studies that demonstrate improvement in gray matter in adolescents with nutritional rehabilitation are lacking. Thus, severity of impact on gray matter is not fully known at time of diagnosis or recovery for most patients. Additionally, information is lacking on younger patients and patients with other restrictive eating disorders, such as atypical anorexia nervosa or ARFID.

In addition to structural gray and white matter changes seen with eating disorders, cognition, reward, and impulse control pathways may not develop fully with disruptions to healthy reproductive and stress hormone levels. These effects on physical brain development are of increased concern when seen in the context of normal adolescent psychosocial development. From childhood to late adolescence, critical developmental tasks must occur in relation to cognitive, social, and emotional development. Adolescents become logical thinkers, set a moral compass, learn life skills, and develop an ability to sustain committed relationships.

Studies have shown considerable social impairment in adolescents with eating disorders. In one study, almost all patients with anorexia nervosa reported social impairment (88.9%) and many adolescents (11.6% with AN and 14.4% with BN) reported at least one day when they were completely unable to carry out any normal activities (Swanson et al., 2011). Interruption of this developmental trajectory by the malnutrition associated with eating disorders can be devastating. Thus, younger patients with eating disorders may have both neurologic and developmental delays that may cause many to become "stuck" in the adolescent brain.

Talking about Medical Complications with Patients and Families

As discussed, children and adolescents with restrictive eating disorders can have unique long-term, potentially irreversible medical complications. Growth, bone, and brain development are at particular risk in younger patients. Patients and families making treatment decisions should discuss both acute and long-term medical risks with their medical providers. The goal for all patients should be to reverse acute medical complications as quickly as possible and to reverse, or at least minimize, long-term complications as much as possible. Timely intervention is critical as most medical complications are reversible or preventable with appropriate treatment.

Assessing Refeeding Risk in Children and Young Adolescents

Refeeding syndrome and refeeding hypophosphatemia have been well described. Nonetheless, guidelines as to when children and young adolescents are most at risk, and how to best approach these issues in younger patients, were lacking until recent years. Previously, only general risk factors for refeeding had been identified for children and adolescents, despite rates of refeeding hypophosphatemia in hospitalized pediatric patients being shown to be up to 37% (Whitelaw, Gilbertson, et al., 2010).

A commonly used tool to assess risk are the criteria from the National Institute for Health and Care Excellence (NICE) (National Collaborating Centre for Acute Care, 2006). Since BMI changes with age, however, these criteria could not be directly applied to children and young adolescents. Additionally, focusing on presentation weight and absolute amount of weight loss can underestimate the severity and impact of weight suppression and the effects of the restricted intake. In children and young adolescents, failure to gain weight should be considered equivalent to weight loss in older patients when assessing for possible risk of refeeding complications.

Newer criteria better encompass risk of refeeding for the full range of ages and diagnoses currently affecting children and adolescents with eating disorders. Recommendations from the ASPEN consensus group consolidate current evidence and provide a separate set of criteria for pediatric patients (da Silva et al., 2020). These newer recommendations focus on BMI Z-score, percentage weight loss for expected weight gain, energy intake compared to estimated needs, and severity of lab abnormalities prior to refeeding; they can be applied to a broad range of eating disorders in children and adolescents (table 14.4).

The ASPEN criteria also help define refeeding syndrome more explicitly as a measurable decrease in serum phosphorous, potassium, or magnesium levels of 10% or greater. Severe refeeding syndrome is described as changes in any of these electrolytes greater than 30%, with end organ dysfunction from decreased electrolyte levels or clinical signs of thiamine deficiency developing in the first 5 days with the initiation of feeding.

Additionally, research has established that children and adolescents with eating disorders in inpatient care with close medical supervision may be refed at faster rates than previously considered, leading to shorter hospital stays (Garber, Michihata, et al., 2012; Golden, Keane-Miller, et al., 2013). Studies show that prophylactic phosphorous supplementation prevented refeeding syndrome in patients on higher calorie diets (Garber, Mauldin, et al., 2013), and some providers have advocated for the routine use of prophylactic phosphorous supplementation in adolescents with anorexia nervosa (Leitner, Burstein and Agostino, 2016; Norris, 2016). Providers desiring further detail regarding the acute management of nutritional rehabilitation can find additional resources in this text. A better understanding of refeeding risk in children and young adolescents will allow for safer delivery of care and more rapid recoveries, decreasing long-term medical complications.

Setting Goal Weights and Monitoring Growth in Recovery

Setting goals weights in growing children and adolescents requires a collaborative effort between nutritional and medical services. Children

TABLE 14.4. ASPEN consensus criteria identifying pediatric patients at risk for refeeding syndrome

	Mild risk: 3 risk criteria needed	Moderate risk: 2 risk criteria needed	Significant risk: 1 risk criterion needed
Weight-for-length Z-scores (1–24 months) or BMI-for-age Z-scores (2–20 years)	–1 to –1.9 Z-score that is a change from baseline	–2 to –2.9 Z-score that is a change from baseline	–3 Z-score or greater that is a change from baseline
Weight loss	<75% of norm for expected weight gain	<50% of norm for expected weight gain	<25% of norm for expected weight gain
Energy intake	3–5 consecutive days of protein or energy intake <75% of estimated need	5–7 consecutive days of protein or energy intake <75% of estimated need	>7 consecutive days of protein or energy intake <75% of estimated need
Abnormal prefeeding serum potassium, phosphorus, or magnesium concentrations*	Mildly abnormal or decreased to 25% below lower limit of normal	Moderately/significantly abnormal or down to 25%–50% below lower limit of normal	Moderately/significantly abnormal or down to 25%–50% below lower limit of normal
Higher-risk comorbidities	Mild disease	Moderate disease	Severe disease
Loss of subcutaneous fat	Evidence of mild loss **or** Mid-upper-arm circumference Z-score of –1 to –1.9	Evidence of moderate loss **or** Mid-upper-arm circumference Z-score of –2 to –2.9	Evidence of severe loss **or** Mid-upper-arm circumference Z-score of –3 or greater
Loss of muscle mass	—	Evidence of mild or moderate loss **or** Mid-upper-arm circumference Z-score of –2 to –2.9	Evidence of severe loss **or** Mid-upper-arm circumference Z-score of –3 or greater

Source: da Silva et al., 2020.

Note: Not intended for use in patients at ≤28 days of life or ≤44 weeks' corrected gestational age.

*Please note that electrolytes may be normal despite total-body deficiency, which is believed to increase risk of refeeding syndrome.

and adolescents are at risk of slowing or cessation of growth and puberty with restrictive eating disorders. All patients who have not completed growth and puberty will need an extensive evaluation at admission to eating disorders care to identify any impact of the restrictive behaviors on growth and puberty.

The initial evaluation must include a review of growth curves in all children and adolescents as well as a calculation of midparental height. Interviewing the patient and parents is often important to determine length of illness. While children and young adolescents will often report a short period of symptoms, parent reports and earlier deviations from prior growth curves may indicate longer illness impact. Length of deviation from prior growth patterns must be considered when setting initial goals. Patients with long-term eating disorders may have had consistent growth for a period but may not be on track for genetic height potential. Patients with ARFID may be at particularly high risk for this overlooked complication.

For children and adolescents who were growing normally before the onset of the eating disorder, the goal of treatment should be to get these patients back to their prior height and weight percentiles. When patients have slowed growth or stopped growing, maintenance at a slightly higher weight percentile than prior to illness may be required to initiate growth. As these patients sustain recovery, ongoing supervision will be required to ensure that they maintain adequate weight gain to continue height gains (figure 14.5). Patients may be extremely uncomfortable in early recovery with ongoing weight gain, and parents may have similar concerns. Close monitoring and review of normal growth and development may help to reassure these patients and families that the young person will "grow into" their weight. Providers should not allow a patient to stay at a weight plateau when uncomfortable. The timeframe for growth is limited, and attempts to decrease patient and caregiver anxiety should not get in the way of rapidly returning patients to their prior growth trajectory.

Further adjustments to this approach may be needed for the patient who is in puberty. Remember that adolescents can have exceptionally fast gains in linear height during the pubertal growth spurt, sometimes

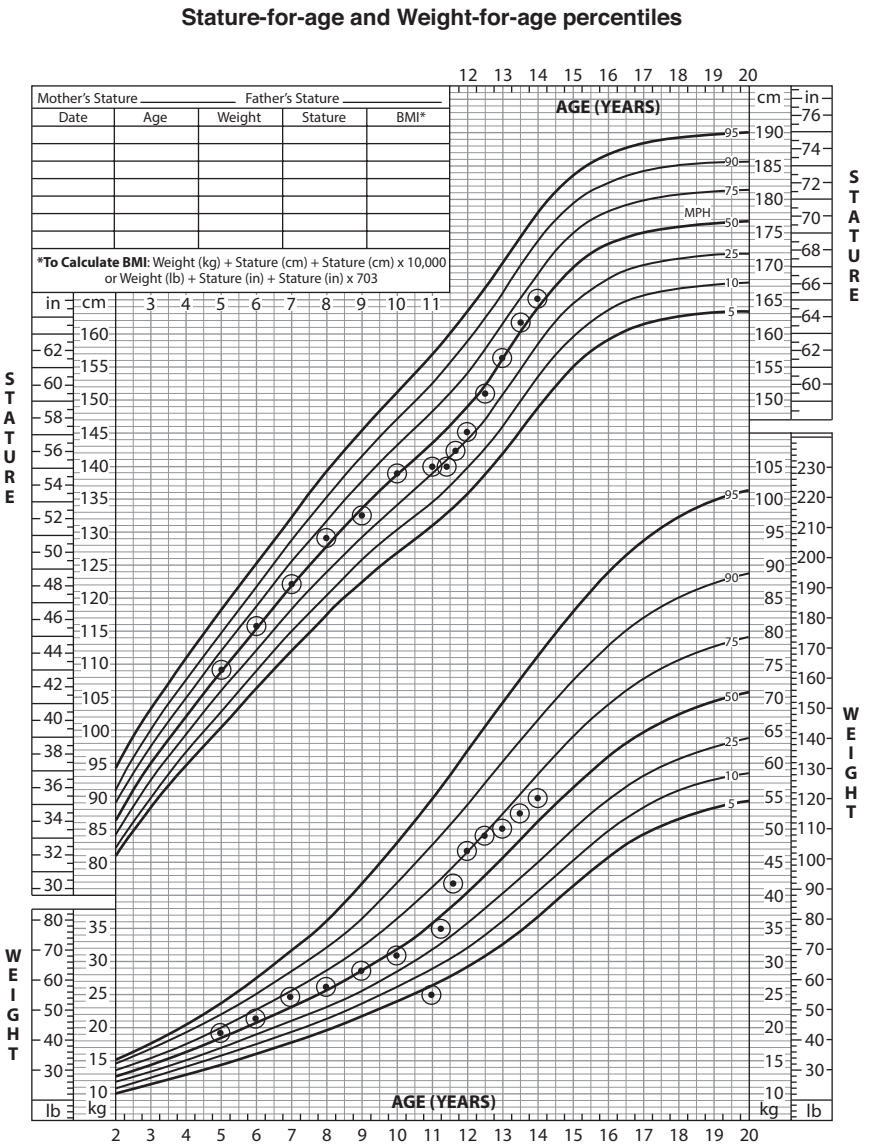


Figure 14.5. Growth curve of patient with restrictive eating disorder preillness, during illness, and through recovery. Note the increase in weight percentile prior to resumption of height gain. The patient in recovery is back on the midparental height curve.

gaining 17% of their final adult height during this time. Close supervision for adequate weight gain to support these rapid increases in linear height gains may require frequent medical visits even when the patient is doing well from an eating disorders behavior perspective. Patients who enter puberty during the illness or recovery may proceed through puberty more quickly and may have a shorter duration of the pubertal growth spurt. Bone age evaluation in these patients can be helpful to determine projections for timing and potential remaining duration of the growth spurt.

All child and young adolescent patients will need close monitoring of growth in recovery as the initial goal weight of treatment is not the final endpoint. Katzman and colleagues (2013) remind providers to discuss with parents and patients that goal weights during adolescence are "moving targets that will change with growth . . . and development." According to SAHM, "During a period of growth, the individual's treatment goal weight should be reassessed every 3–6 months" (Golden, Katzman, Sawyer, et al., 2015a).

Growth is inexpensive to measure and should be done often and for as long as needed. Evidence from Neale et al. (2020) that losses in height attainment may be irreversible can be constantly reinforced with patients and families to support ongoing care and supervision. Providers can use FBT principles to help patients and families understand that restoration of physical health is not negotiable. For children and adolescents, growth can be one of the most important markers of good health.

Studies of long-term outcomes in eating disorders currently focus on remission of disordered thoughts and weight restoration. With a newer understanding of the potential long-term risk to linear growth and bone density, future studies should include examination outcomes with an eye to prevention of irreversible medical complications in children and young adolescents.

Approaching ARFID in Children and Young Adolescents

Patients with avoidant/restrictive food intake disorder, a diagnosis newly included in the *DSM-5*, present unique challenges to medical providers

for diagnosis and management. Patients with ARFID are unable to meet their nutritional or energy needs as manifested by one or more of the following: significant weight loss (or failure to make expected gains in weight or growth), significant nutritional deficiencies, a dependence on oral supplements or enteral feeds, and/or marked interference in psychosocial functioning. Patients with ARFID do not have body image disturbances. The diagnosis of ARFID replaced a prior feeding disorder diagnosis and now includes older patients and patients with more psychosocial issues. Subtypes will likely continue to be differentiated as research on this new diagnosis progresses (Sharp and Stubbs, 2019).

Patients with ARFID will present with concerns about adverse consequences related to eating, an apparent lack of interest in eating or food, or an avoidance of food based on sensory characteristics. Because of these issues, patients with ARFID all have trouble meeting appropriate nutritional needs and should be considered to have the same potential medical risks as children and adolescents with other restrictive eating disorders. Patients with ARFID may have serious medical complications that are often unrecognized.

Patients with ARFID made up approximately 14% of new patients presenting for eating disorders evaluation at institutions with adolescent medicine specialists (Ornstein et al., 2013). Compared to other eating disorder patients, these patients are often younger, have had longer durations of illness prior to presentation, and are more likely to be male. Patients with ARFID have high rates of comorbid psychiatric conditions in addition to medical symptoms (Fisher et al., 2014). Common comorbidities include anxiety disorders, OCD, learning disabilities, and autism spectrum disorders.

Although often dismissed initially as "just picky eating," patients with ARFID do not improve by middle childhood, as do children with childhood selective eating. Many patients with ARFID have only a few foods that they can consume, and they will present eating the same foods meal after meal and day after day. Brand selectivity is common, as is avoidance of certain food based on colors, smells, or textures. For many of these patients, the eating issues begin very early. Parents will often describe issues with volume and/or variety starting during infancy or

childhood. The long-term impact of such chronic malnutrition is still poorly understood.

A subset of ARFID patients present with acute fears of adverse events related to food or eating. Common fears are choking, vomiting, or illness. Often, these presentations are more acute and may surface following a frightening event. At times, however, no precipitating event is identified. Patients with an acute onset and a sudden reduction in oral intake are at risk for the same medical complications of restriction as those seen in children and adolescents with anorexia nervosa. In general, however, patients with ARFID are less likely to present with acute weight loss and associated symptoms such as bradycardia (Strandjord et al., 2015). Because they do not have body image disturbances and because their nutrition issues are often chronic, these patients may avoid medical detection until late in the illness.

Many patients with ARFID have associated somatic symptoms such as abdominal pain (Zucker et al., 2019). Somatic complaints may lead to extensive medical testing if the primary issue of ARFID is not identified. Patients with ARFID do not have concerns about shape or weight. In fact, patients with ARFID often want to gain weight but struggle to take in adequate volume or variety of food. Nutritional concerns may intensify during times of increased energy needs such as with growth spurts or increases in activity. Many patients with ARFID have had failure-to-thrive evaluations and seen specialists in gastroenterology or endocrinology and may have worked with feeding specialists previously.

The disgust response has been well described in many patients with ARFID and may serve to maintain selective eating (Harris et al., 2019; Menzel et al., 2019). Understanding the implications of the disgust response and impairments in psychosocial functioning are critical to successful treatment. These patients are often unable to attend to normal developmental milestones that relate to food or eating. Because they can be uncomfortable eating in public, or with family or friends, they may miss important developmental opportunities. Many patients with ARFID have been unable to enjoy important childhood experiences such as family meals, sleepovers, school lunches, outings with friends, summer camps, and travel. Because the disgust response may not extinguish with

exposure, older patients with ARFID often need a highly valued motivation to do the necessary exposure work and may need to start treatment with the expectation that disgust may not change. Relating this understanding and these expectations to the final desired social outcomes can result in more effective treatment participation.

Patients with ARFID and higher levels of severity or medical complexity will need multidisciplinary team involvement and engagement of caregiver and family support. Eddy and Thomas (2019) note that psychoeducation about ARFID, exposure therapy, and structured mealtimes are all important components of treatment. Patients with ARFID will need to achieve a healthy weight, correct nutritional deficiencies, increase amount and variety of foods eaten, and learn skills to identify and manage emotions and feel more comfortable in social situations. Working with a team of seasoned eating disorders professionals with experience with ARFID will allow this work to progress in a coordinated manner.

Since patients with ARFID can have various symptomatic presentations, medical complications may also vary widely. The authors recommend that providers evaluate for medical complications based on duration of ARFID symptoms and identification of intake concerns as primarily related to volume or variety. Providers should also remember that patients may have issues with both volume and variety and may have an early onset of symptoms but present later when using this tool, so multiple domains may need to be assessed as part of the initial evaluation.

- For patients with **early onset** of difficulties with **volume**:
  - Evaluate for medical complications related to growth and bone development
  - May be at particular risk for acute medical complications of starvation at times of increased energy needs (e.g., growth spurts, illness, change in physical activity)
- For patients with **early onset** of difficulties with **variety**:
  - May be at risk for less common nutritional deficiencies (e.g., scurvy or rickets)

    - May be at risk for social concerns, primarily stemming from difficulties engaging in normalized eating patterns with peers and/or family
- For patients with **late onset** of difficulties with **volume**:
    - Evaluate for acute medical complications of starvation
    - May be at risk of refeeding syndrome if associated with sudden weight suppression
- For patients with **late onset** of difficulties with **variety**:
    - May be at risk for social concerns, primarily stemming from difficulties engaging in normalized eating patterns with peers and/or family
    - May be at risk for less common nutritional deficiencies (e.g., scurvy or rickets)

Any child or adolescent with acute volume difficulties or acute weight suppression must be evaluated under the AAP and SAHM guidelines for acute medical stabilization. These patients can be at risk for the same complications of starvation as any restrictive eating disorder.

As with any other children or young adolescents with an eating disorder and failure to make expected gains in height and weight, patients with ARFID may be at risk for long-term irreversible impact on final adult height and bone density, although studies are lacking due to the novelty of this diagnostic categorization. All patients with ARFID should have an assessment for growth compared to genetic potential as evaluated by MPH (figure 14.6). For patients who are behind on growth, providers may need to ensure adequate nutrition to get weight and linear growth back on track. Although the use of supplements is a defining abnormal characteristic of ARFID, patients may require nutritional supplements to maintain adequate growth while beginning work on issues of volume and variety.

The Role of the Medical Provider in the Care of Children and Young Adolescents with Eating Disorders

The medical provider holds a critical role in identifying eating disorders, ensuring patient safety, and excluding other medical causes for presenting

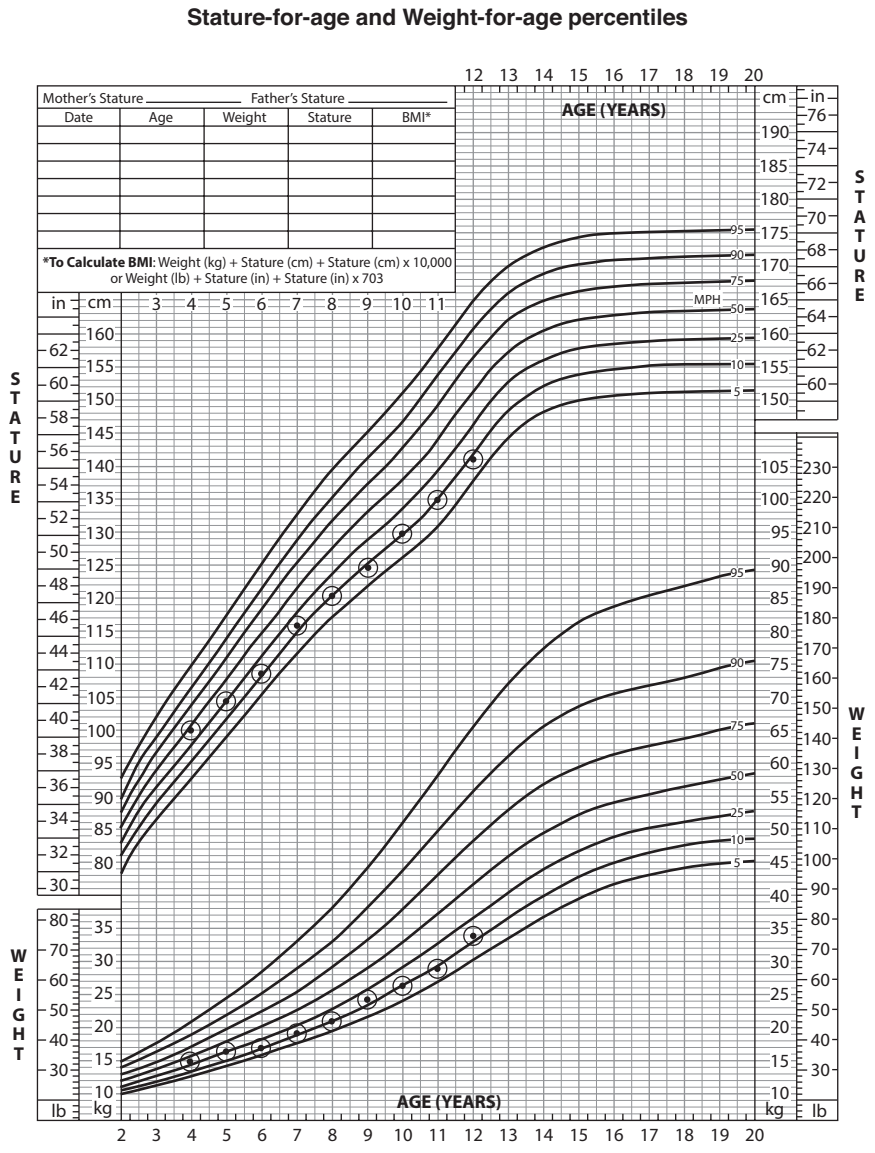


Figure 14.6. Growth stunting in a patient with avoidant/restrictive food intake disorder. The patient is not growing along the midparental height curve.

symptoms. For many patients, the first provider to identify the eating disorder will be a primary care provider. Patients of higher severity will require a multidisciplinary eating disorders team (Golden, Katzman, Sawyer, et al., 2015a). The primary care provider should be involved in these cases and will need to be familiar with issues related to medical monitoring, especially regarding growth and bone health in children and young adolescents. Primary care providers may feel comfortable in this role or may prefer to refer the patient to specialized eating disorders providers if available (Hornberger, Lane, and the Committee on Adolescence, 2021).

Ongoing improvements in early detection and early intervention will require increased recognition of the newer eating disorder diagnoses such as ARFID and atypical anorexia nervosa. Medical providers must move past any prior biases and recognize that eating disorders can present in any age, gender, race, socioeconomic group, or body size. When concerned, patients should be quickly evaluated and referred for treatment. Shorter durations of illness are associated with better outcomes.

The nutritional restoration process should begin immediately, using medical hospitalization if indicated. Physical and psychological complications of malnutrition and refeeding should be identified and managed. The appropriate initial level of treatment should be determined by the needs of the individual patient. Medical providers should assist in the initial determination of the correct level of care and provide regular medical monitoring to ensure safety and progress. The family will need ongoing support and guidance as well.

Because shame and stigma still surround eating disorder diagnoses, families may at first be reluctant to accept an eating disorders diagnosis. In addition, medical providers may be reluctant to make this diagnosis and may inadvertently create delays in initiating care, especially in younger patients. Providers must remember that delays in care can cause irreversible medical complications—especially loss of linear height and bone density. Balancing kindness and compassion with firmness regarding concerns can effectively help families and patients engage in the next steps that can lead to lasting recovery.

Prevention of Eating Disorders in Children and Adolescents

Both the AAP and SAHM have issued statements regarding the prevention of eating disorders in children and adolescents.

Some key points for providers from the AAP (Golden, Schneider, et al., 2016):

- Discourage dieting, skipping meals, or using diet pills.
- Promote positive body image—do not encourage body dissatisfaction.
- Encourage more frequent family meals.
- Encourage families not to talk about weight, but to talk about healthy eating and being active to stay healthy.
- For overweight or obese teenagers, ask about a history of mistreatment or bullying.
- Carefully monitor weight loss in adolescents who medically need to lose weight, ensuring that they do not develop the medical complications of semistarvation.

In addition, SAHM stated that medical providers can do several things to decrease the risk of eating disorders (Kumar et al., 2018). These included reminding adolescents that there are no "good foods" or "bad foods," explaining that nutritional patterns are more important than individual foods eaten, encouraging adolescents to critically evaluate nutrition claims, and screening adolescents for exclusionary diets and discouraging these diets unless there is a medical, cultural, or religious necessity.

Talking to Children and Families

The diagnosis of an eating disorder can be disruptive and difficult for young patients and their families. Families and patients may wrestle with the shame and stigma regarding eating disorders that continue to permeate our society. Early on, providers can reinforce an important message that families do not cause eating disorders, and that these are unbidden psychiatric illnesses.

At times, children may present as acutely medically unstable, and families will have to manage an unexpected medical emergency in addition to a new psychiatric diagnosis. Children and young adolescents may become medically unstable more quickly than adult patients. Refeeding risk may be underrecognized. Younger patients may present with significant loss of bone density or linear height. Walking families through these challenges is crucial in the early management and stabilization of younger patients with eating disorders.

Families may face difficult decisions when weighing options. Treatment considerations for children must balance family involvement, local options, distance to treatment, severity of illness, acute medical risk, and long-term risk of irreversible medical complications. Even without a comorbid diagnosis of an anxiety disorder, being ill in childhood is disruptive and can create anxiety. Young patients need family support, and options that underscore the importance of family involvement are critical.

Early detection, early intervention, and weight restoration are key components to improving eating disorder outcomes in children and adolescents (Rome et al., 2003). Medical providers can ensure that patients remain on track, and that there is adequate pace of progress, as treatment decisions are made. Using all available local and national resources, providers should help guide families and patients to access best practice treatment. Providers can remind patients and families that with help, children and young adolescents with eating disorders can and do get better.

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15

Atypical Anorexia Nervosa

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COMMON QUESTIONS

What are the differences in diagnostic criteria between anorexia nervosa and atypical anorexia nervosa?
Are there differences in epidemiology and clinical presentation between the two conditions?
Are there medical risks associated with atypical anorexia nervosa?
How do you treat atypical anorexia nervosa?
How does a provider determine a healthy body weight or treatment goal weight for someone with atypical anorexia nervosa?
How do you define clinical recovery in patients with atypical anorexia nervosa?

Case Study 1

E.E. is a 17-year-old female who presented to a tertiary care eating disorders program because of bradycardia (HR of 40 bpm) and orthostasis after losing 45 pounds over one year. At a previous routine health maintenance examination, she had been told by her physician that "it was a good idea to lose weight." In the past she had weighed 185 pounds, with a BMI of 29.9 kg/m^2 (>95th percentile for age). She lost the weight by dietary restriction to 600 to 800 kcal/day and an exercise regime that included running 7–8 miles 3–4 times a week,

swimming 150 laps 3–4 times a week, biking for 11 miles 2–3 times a week, and participating in a Zumba class for one hour once a week. Her last menstrual period was 6 months ago. Her parents became concerned after she developed social isolation, increased irritability, and anxiety around eating. On admission her weight was 140 pounds (75th percentile for age, 104% of median BMI), height 66.5 inches (82nd percentile for age) with a BMI of 23.2 (71st percentile). Her resting pulse was 40 bpm and went up to 70 on standing. Her BMI chart is shown in figure 15.1.

Case Study 2

D.J., a 14-year-old male who was previously overweight (220 lbs.), was referred by his pediatrician to a specialized eating disorder program because of bradycardia and orthostasis in the context of severe dietary restriction and loss of 74 pounds over 6 months (33% of his premorbid body weight). There was a strong family history of hypertension and type 2 diabetes, and he wanted to be healthy. Initially he decided to cut out junk food, soda, and high fat foods. Eventually he was eating only one small meal a day. His mother took him to the pediatrician because of abdominal pain and constipation. She was also concerned about behavioral changes. He kept to himself and no longer interacted with his friends or siblings. He had not had a bowel movement for more than 7 days. On examination, his weight was 146 pounds (91st percentile for age), height 69 inches (93rd percentile), and his BMI was 22 (82nd percentile). He was 114.9% of median BMI. His resting pulse was 42 bpm and increased to 83 on standing (pulse difference of 41). Overnight, on cardiac telemetry, his pulse dropped to 34 bpm. His electrocardiogram revealed sinus bradycardia, ST elevation, and a prolonged corrected QT interval of 481 ms.

Background

Obesity, a major health problem worldwide, is associated with significant medical and psychologic morbidity. Over the past 30 years, the rate of childhood obesity has more than doubled, and the rate of adolescent

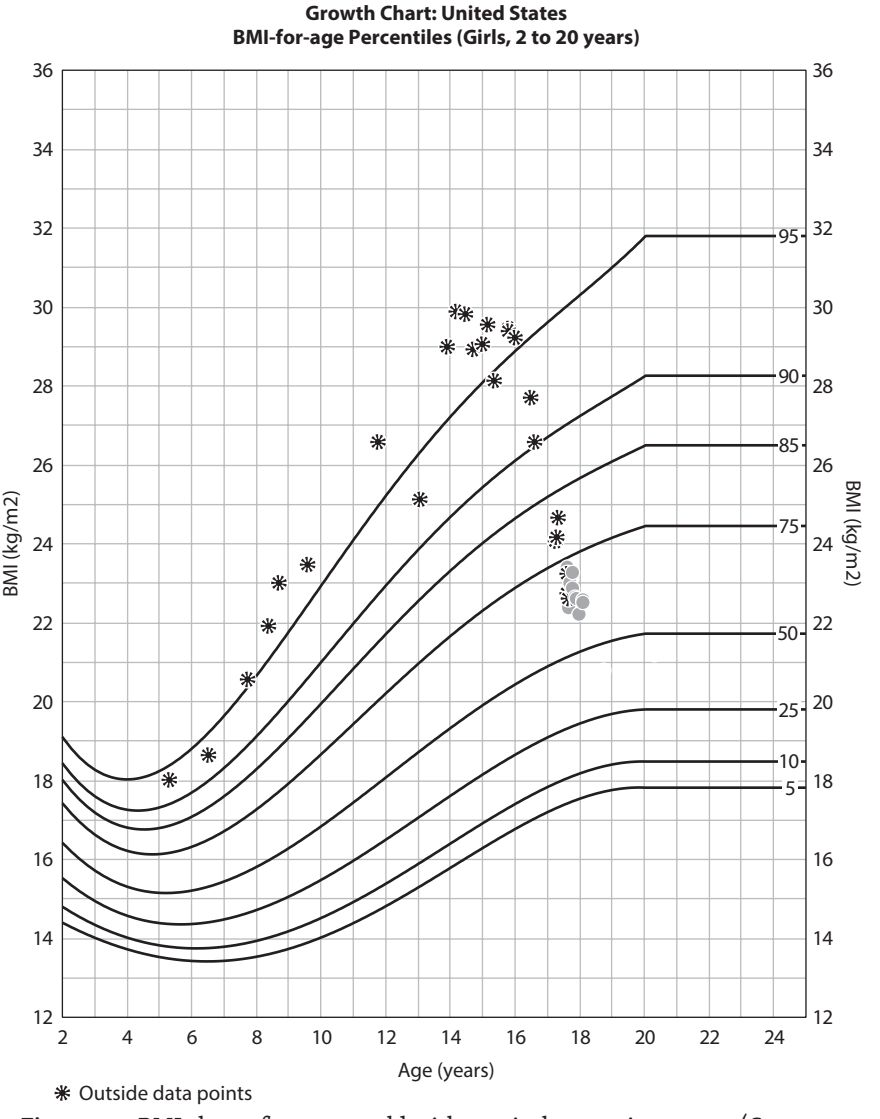

Figure 15.1. BMI chart of a 17-year-old with atypical anorexia nervosa (Case Study 1). At age 14 years, BMI was >95th percentile for age. At presentation at age 17, after losing 45 pounds, BMI was in the normal range.

obesity has quadrupled (Skinner et al., 2018; Golden, Schneider, and Wood, 2016). The prevalence of adult obesity and severe obesity is also increasing, and in the United States 42.4% of adults were obese in 2017–2018 (Hales et al., 2020). Individuals who are obese are at increased risk

of developing multiple medical and psychological sequelae, including hypertension, type 2 diabetes, coronary artery disease, end-stage renal disease, nonalcoholic fatty liver disease, gallstones, obstructive sleep apnea, bone and joint disorders, as well as increased rates of depression and poor quality of life. Most patients who are overweight and try to lose weight for health reasons do not develop eating disorders, but some, as in the two cases described above, may develop atypical eating disorders that can be as severe as the classic disorders.

Historically, obesity and eating disorders have functioned as distinct fields of study with little interaction or collaboration among scientific communities. An emerging body of literature, however, has argued for a more integrated approach to prevention of both eating disorders and obesity (Golden, Schneider, and Wood, 2016; Neumark-Sztainer and Loth, 2018). Neumark-Sztainer and colleagues demonstrated that patients who are obese and those who have eating disorders share similar risk and protective factors (Neumark-Sztainer, Wall, et al., 2007). Youth who have been teased about their weight are more likely to develop body dissatisfaction and disordered eating (Neumark-Sztainer, Falkner, et al., 2002), and disordered eating occurs more frequently among adolescents who are overweight than among their normal-weight peers (Field et al., 1999; Neumark-Sztainer and Hannan, 2000; Rodgers et al., 2017). Furthermore, longitudinal observational studies have shown that engaging in disordered eating behaviors during adolescence predicts a higher BMI 15 years later (Yoon et al., 2020).

Prevention and treatment of obesity is complex, requiring a multisystem approach. Individuals who are overweight or obese are being encouraged to lose weight, but frequently without clear guidance on how to do so, and with little medical monitoring. Some adolescents and young adults who were previously overweight engage in unhealthy weight loss behaviors, such as extreme dieting, excessive exercise, skipping of meals, self-induced vomiting, or use of laxatives, diuretics, or diet pills to lose weight, and may be at risk for developing an eating disorder.

Atypical anorexia nervosa (AAN) describes those individuals who meet all the criteria for anorexia nervosa (AN) except that, despite significant weight loss, their weight remains in the normal or above nor-

mal range. Patients with AAN are being referred to specialized eating disorders programs with increasing frequency. Most individuals with AAN were previously overweight or obese and frequently go unrecognized because weight loss is usually seen as beneficial in this population, and the diagnosis of an eating disorder is not suspected because weight is still in the normal range. As a result, they present for treatment after a longer duration of illness (Lebow, Sim, and Kransdorf, 2015). Because AAN is classified under the category of other eating disorders, many providers perceive AAN to be less severe than AN. On the contrary, even though their weight may be in the normal or above normal range, patients with AAN can have just as severe or more severe medical complications as those with classic AN and can have even greater eating disorder psychopathology (Sawyer et al., 2016; Garber et al., 2019). The aim of this chapter is to review the epidemiology, etiology, diagnostic criteria, clinical presentation, medical complications, psychological morbidity, and treatment of adolescents and young adults with AAN.

Epidemiology

The proportion of patients with a history of overweight and obesity presenting to specialized eating disorders programs has increased dramatically, often accounting for 25% to 45% of patients admitted to inpatient units (Lebow, Sim, and Kransdorf, 2015; Sim, Lebow, and Billings, 2013; Whitelaw, Gilbertson, et al., 2014; Sawyer et al., 2016; Garber et al., 2019; Kennedy et al., 2017). The number of such patients presenting to one tertiary care inpatient service increased fivefold over a six-year period (Whitelaw, Gilbertson, et al., 2014). Patients with AAN have different demographic characteristics from those with AN. One study of 552 adolescents and young adults with restrictive eating disorders found a higher prevalence of males in the AAN group (18.8%) compared to the AN group (10.5%) and a higher prevalence of patients from racial minorities (Kennedy et al., 2017).

Limited data exist on the population prevalence of AAN in community samples. In adolescents, using a community sample of 496 adolescent girls followed for 8 years, the lifetime prevalence for AAN by age 20 years

was 2.8%, compared with 0.8% for AN. The cumulative incidence over the 8 years of follow-up was 14% with an estimated incidence of 366 per 100,000 persons (Stice, Marti, and Rohde, 2013). Prevalence and incidence rates for adolescent boys are not known. In adults, a community sample of 1,640 women and 794 men attending a private university revealed a point prevalence of 2.8% in women and 2.0% in men when using a cutoff of >15% weight loss to define AAN (Forney et al., 2017).

Etiology

It is not known whether AAN and AN are similar conditions on the same spectrum, or entirely different clinical conditions. The classic starvation experiments of Ansel Keys demonstrated that experimental semistarvation in previously healthy volunteers can lead to many of the medical and psychological findings seen in patients with AN, including bradycardia, orthostasis, hypotension, reduced bone mineral density, as well as preoccupation with food, disordered eating behaviors, and comorbid depression (Keys et al., 1950). The etiology of classic AN is multifactorial, and the biopsychosocial model of etiology describes predisposing, precipitating, and perpetuating factors contributing to the etiology. We do not know whether these same factors apply to AAN. For example, a biologic predisposition has been identified for patients with AN, and genome-wide association studies (GWAS) have demonstrated a region of interest on chromosome 12 (Duncan et al., 2017). It is not known whether patients with AAN share this same genetic predisposition. In addition, the proportion of males with AAN is higher than that for AN, and the premorbid personality type described for AN (perfectionistic, obsessional, harm avoidant) may not apply to patients with AAN.

Diagnosis

Atypical anorexia nervosa is a new diagnostic category in the 5th edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*) and falls under the category of other specified feeding or eating disorders (OSFED) (American Psychiatric Association, 2013). It describes

individuals who meet all criteria for AN except that, despite significant weight loss, weight is within the normal or above normal range. These individuals report an intense fear of gaining weight or becoming overweight, with persistent behaviors that interfere with weight gain. It is not clear whether they have a distorted body image and perceive themselves to be more overweight than their actual body weight suggests. While the introduction of this diagnostic category has improved the clinical utility of the diagnostic categories, certain challenges remain. For example, it is not clear what constitutes "significant weight loss" (Forney et al., 2017), nor over how short a period said excessive weight loss needs to occur. Forney and coauthors demonstrated that loss of even 5% of premorbid weight with cognitive concerns can be associated with significant disordered eating and psychological distress, while other individuals can lose a great deal of weight with little or no psychological distress. The diagnosis of AAN requires both significant weight loss and cognitive concerns.

Low body weight is a key clinical feature of AN and differentiates AN from AAN. In adults, a BMI <18.5 kg/m^2 indicates low body weight, but the interpretation of what constitutes low body weight in adolescents remains problematic because of changing height, weight, and body composition during normal linear growth and pubertal development. The suggested weight cutoff of <85% expected body weight to diagnose AN in *DSM-IV* was eliminated in *DSM-5*. An unintended consequence of eliminating a low-weight threshold for AN is that currently there is considerable diagnostic overlap between AN and AAN. A study of 14 adolescent medicine-based eating disorder programs, which applied *DSM-5* criteria to 700 adolescents with eating disorders, found that 14% of patients diagnosed with AN were ≥ 90% median BMI, and 27% of patients diagnosed with AAN were <90% median BMI (Forman et al., 2014). In clinical research studying patients with AAN, some investigators have used a weight ≥90% of median BMI to indicate a normal or above normal weight (Sawyer et al., 2016; Stice, Marti, and Rohde, 2013), while others have used a weight ≥85% median BMI (Whitelaw, Lee, et al., 2018; Garber et al., 2019).

Clinical Presentation

In one study, 25% of women and 40% of men with AAN were overweight or obese at presentation (Forney et al., 2017). Compared with patients with classic AN, patients with AAN present for treatment after a longer duration of illness, have higher premorbid weight, and have lost a greater amount of weight (Sawyer et al., 2016). As expected, they present at a higher body weight, higher BMI, and higher percentage of median BMI. Weight suppression, defined as the difference between the highest weight and presentation weight, has been found to be an important determinant of outcome in patients with AN (Wildes and Marcus, 2012). In patients with AAN, weight suppression is greater than in those with classic AN (Lebow, Sim, and Kransdorf, 2015).

The two cases outlined at the beginning of this chapter describe the classic clinical presentation of patients with AAN. Usually the patient is or was overweight or obese. Weight loss is frequently recommended by a health care provider, and the patient starts off eating "healthily" or "clean," with a slow increase in physical activity. Initial weight loss is viewed favorably by both the provider and family members. Over time, continued weight loss progresses to become obsessional, and weight control behaviors become more rigid and extreme and may be accompanied by social isolation and depression.

Weight history at clinical presentation is associated with objective measures of illness severity. Whitelaw and coauthors showed that greater total weight loss and the amount of recent weight loss (within the prior 3 months) each predicted both incidence and severity of bradycardia (Whitelaw, Lee, et al., 2018). Similarly, Garber et al. (2019) demonstrated that faster weight loss and a longer duration of weight loss were associated with lower heart rate on admission, and a greater amount of weight loss was associated with lower admission serum phosphorus levels. Taken together, these studies demonstrate that significant weight loss, over a relatively short period, can lead to life-threatening complications even at a normal or above normal weight.

Medical Complications

The medical complications from weight loss in patients with AAN are similar to those seen in classic AN. Peebles et al. (2010) found that patients who had lost >25% of their premorbid weight were more medically compromised than the AN group. Specifically, they are more likely to have bradycardia and orthostasis by heart rate and were more likely to meet medical admission criteria.

The medical complications of malnutrition found in patients with AAN are shown in table 15.1. Studies have revealed no differences in the severity of the acute medical complications between AAN and AN. Specifically, there were no differences in the lowest pulse rate, lowest body temperature, frequency of bradycardia, or incidence of hypophosphatemia (Sawyer et al., 2016; Whitelaw, Gilbertson, et al., 2014). Lowest lying systolic blood pressure, however, was higher in those with AAN (Whitelaw, Gilbertson, et al., 2014). At presentation to one tertiary care center, 24% of adolescents with AAN had bradycardia, 43% had orthostatic changes in pulse or BP, and 33% of girls not on oral contraceptives had secondary amenorrhea (Sawyer et al., 2016).

The major electrolyte disturbances seen in patients with eating disorders are hypokalemia, hypomagnesemia, and hypophosphatemia, and occur secondary to purging behaviors such as vomiting and diuretic or laxative abuse, or develop during nutritional rehabilitation as part of refeeding syndrome. Rates of hypokalemia, hypophosphatemia, and hypomagnesemia in hospitalized patients with AAN are similar to those in patients with AN (Garber et al., 2019).

Bradycardia develops as an adaptive response to an energy deficit, mediated via increased vagal tone. It is not known whether patients with AAN develop the other cardiac complications seen in AN, such as mitral valve prolapse or reduced left ventricular wall thickness.

Bloating, early satiety, abdominal discomfort after meals, and constipation are common symptoms in patients with eating disorders, including those with AAN, and reflect delayed gastric emptying and decreased intestinal motility. Rapid weight loss can be associated with gallstone

TABLE 15.1. Medical complications of atypical anorexia nervosa

Fluid and Electrolytes	Dehydration Hypokalemia Hyponatremia Hypophosphatemia Hypomagnesemia
Cardiovascular	Bradycardia Hypotension
Gastrointestinal	Bloating and constipation Delayed gastric emptying Elevated liver enzymes Gallstones Gastroesophageal reflux Esophagitis Barrett's esophagus Bloody diarrhea Rectal prolapse
Endocrine	Low estrogen (girls) or low testosterone (boys) Primary or secondary amenorrhea (girls) Low T3 syndrome
Hematologic	Anemia Leukopenia Thrombocytopenia
Skeletal	Low bone mineral density Possible increased bone fragility
Neurologic	Syncope Seizures

formation. Recurrent vomiting can lead to parotid and submandibular gland enlargement, loss of the gag reflex, dental enamel erosion, gastroesophageal reflux, esophagitis, elevations in serum amylase, and Mallory-Weiss tears. Prolonged vomiting causes Barrett's esophagus, precancerous changes in the epithelium of the distal esophagus caused by recurrent exposure to acidic stomach contents. Laxative abuse can cause profuse bloody diarrhea and rectal prolapse. Elevation of liver transaminases occurs on presentation in approximately 20% of patients with AAN (Garber et al., 2019).

Chronic energy restriction leads to suppression of the hypothalamic-pituitary-gonadal axis by altering the pulsatility of gonadotropic-releasing hormone secretion, mediated in part by leptin and low triiodothyronine (T3) levels. This results in amenorrhea in girls, with low levels of luteinizing hormone, follicle-stimulating hormone, and estradiol, and in boys, low testosterone levels and decreased libido. Multiple studies have shown that both the prevalence of amenorrhea and its duration in girls with AAN are similar to those with AN (Garber et al., 2019; Lebow, Sim, and Kransdorf, 2015; Rastogi et al., 2020).

Chronic malnutrition in AN can lead to bone marrow suppression resulting in anemia, leukopenia, and thrombocytopenia. Nutritional deficiency of iron, folate, and vitamin B_{12} can aggravate the anemia. Bone marrow suppression has not been studied in AAN.

Reduced bone mineral density with increased bone fragility are serious long-term complications of AN in both male and female patients. Contributing factors include low body weight, low levels of sex hormones, and hypercortisolemia. In females, prolonged amenorrhea is associated with the degree of reduction of bone mineral density. Similar to patients with AN, those with AAN can also have reduction in bone mineral density, but to a lesser degree (Bachmann et al., 2017; Schorr et al., 2017). Body weight is an important determinant of bone strength, and in general, the higher the body weight, the greater the biomechanical loading forces exerted on the skeleton during weight-bearing activities. These forces exerted on the skeleton promote bone formation and are protective of bone health. In AAN, low levels of estrogen and testosterone contribute to low bone mineral density despite the higher weight of patients with AAN. We do not yet have data on fracture risk in patients with AAN, but a bone densitometry (DEXA) scan can help to quantify the amount of bone mineral loss.

The major neurologic findings in patients with eating disorders are structural brain changes, syncope, and seizures. While structural brain changes, such as gray and white matter atrophy, have been demonstrated in AN, no such changes have been identified in patients with AAN (Olivo, Solstrand Dahlberg, et al., 2018; Olivo, Swenne, et al., 2019). Severe orthostasis can result in syncope, and seizures can develop

secondary to electrolyte disturbances associated with disordered eating behaviors.

Psychological Morbidity

In a community sample of 496 adolescent girls, Stice, Marti, and Rohde (2013) showed that adolescent girls with AAN reported significantly greater functional impairment, emotional distress, and suicidality than girls without an eating disorder. Two studies have demonstrated that patients with AAN have higher distress related to eating and body image than those with AN (Sawyer et al., 2016; Garber et al., 2019). Sawyer et al. (2016) found that adolescents with AAN scored higher on all subscales of the EDE (Restraint $p = .03$, Eating Concerns $p = .009$, Shape Concerns $p < .001$, Weight Concerns $p < .001$, and Global Score $p < .001$). They found no significant differences in rates of psychiatric comorbidity, use of psychotropic medication, severity of depression, or rate of suicidal ideation. Similar to AN, the most common psychiatric comorbidities were depression, anxiety disorders, and obsessive-compulsive disorder (Sawyer et al., 2016). Both Sawyer et al. (2016) and Monge et al. (2015) found similar prevalence of comorbid depression.

Treatment and Outcome

Most of the research on treatment of eating disorders is limited to underweight patients, and there are few data to guide management of previously overweight patients with eating disorders (Rastogi and Rome, 2020). Until new evidence emerges, the current approach to management of a patient with AAN is similar to that of patients with other eating disorders, but there are specific challenges, for example, how to determine the amount of weight gain required for clinical recovery. Similar to other eating disorders, a multidisciplinary team approach is recommended (Society for Adolescent Health and Medicine et al., 2015).

The medical goals of treatment are nutritional rehabilitation, metabolic recovery, and resolution of the medical complications. For those

presenting with unstable vital signs, brief medical hospitalization may be required for medical stabilization (Society for Adolescent Health and Medicine et al., 2015). Those who have been severely restricting their dietary intake are at risk of developing refeeding syndrome on reintroduction of nutrition, so careful monitoring of serum phosphorus during the early stages of refeeding is recommended. Weight gain is not necessarily one of the primary medical goals of treatment, but some weight gain may be necessary for metabolic recovery. The patient's greatest fear is rapid weight gain to premorbid weight levels. The medical provider can play an important role in reassuring the patient that this is not the goal of treatment.

The patient's prior growth trajectory can help guide determination of treatment goal weights. Someone who was previously at the 75th percentile may need to get back close to that percentile for medical and psychological recovery. For those whose premorbid BMI was >95th percentile, a BMI closer to the 75th or 80th percentile might be more appropriate for recovery. In females, resumption of menses can be used as one objective marker of biological recovery, and serial estradiol levels obtained every three months could be used to predict an individualized weight that would indicate restoration of normal hypothalamic-pituitary-gonadal function and imminent resumption of menses. In patients with AN, Golden and colleagues showed that a serum estradiol level ≥30 pg/mL is associated with resumption of menses (RR 4.6; 95% CI 1.9–11.2) within 3–6 months (Golden, Jacobson, et al., 1997). A serum estradiol <30 pg/mL would suggest that further weight gain is required, but a level >30 pg/mL would suggest that the patient's current weight is at a level that would support resumption of menses, and maintenance of weight at that level might be more appropriate than continued weight gain. Seetharaman et al. (2017) demonstrated that in females with AAN, some weight gain is required for resumption of menses, and patients with AAN resume menses at approximately 106.1% ± 11.7% median BMI, a weight higher than their previously normal-weight peers with AN, 94.2% ± 8.9% median BMI. As demonstrated by Rastogi et al. (2020), however, patients with AAN do not necessarily need to be restored to premorbid maximum weight to resume menses.

A registered dietician can help the patient create a structured meal plan incorporating three meals and one to two snacks a day and provide nutritional counseling regarding healthy food choices that are sustainable. Counseling is critical to reassure the patient that the goal is to improve nutrition and reverse the medical complications of malnutrition and not necessarily to return to premorbid weight levels. Once vital signs have stabilized, moderate physical activity can be introduced and slowly advanced to improve psychological well-being. Physical activity will also help with long-term weight management.

Preliminary studies suggest that family-based therapy (FBT) may be effective in adolescents with AAN. A case series of 42 adolescents with AAN demonstrated high engagement, high completion rates, and reduction in eating disorder symptoms (Hughes et al., 2017). These findings suggest that despite the emphasis on weight gain in classic FBT for AN, this outpatient psychological treatment may have a role in the treatment of AAN, but randomized controlled trials are lacking. Determination of clinical recovery remains challenging. By definition, patients are already at or above normal weight, so weight gain alone cannot be used to define clinical recovery. Resolution of medical complications (such as amenorrhea in females) can be used as one biological marker of recovery, but objective biological measures of recovery other than weight for males are lacking. In both females and males, objective measures of improvement in psychological functioning, such as normalization of EDE-Q scores, can be used to guide clinical recovery.

Given that AAN is a relatively new diagnostic category, there are limited data on outcome. One study found that approximately 71% of adolescents with AAN showed remission within 1 year (Stice, Marti, and Rohde, 2013).

Summary

In summary, eating disorders can occur at various weights, and patients with AAN can be just as ill medically and even more ill psychologically as those with other eating disorders. After recommending weight loss for an overweight patient, medical practitioners should carefully monitor

their patient to ensure they do not develop an eating disorder. Those who have lost a large amount of weight or have lost weight rapidly should be asked about disordered eating behaviors. Although we do not yet know much about outcome, early intervention is likely to be associated with improvement. Focus of treatment in patients with AAN should be on metabolic recovery and improvement in psychological functioning along with some degree of judicious weight restoration. Some weight gain is necessary for metabolic recovery, but those who were previously obese do not necessarily need to return to premorbid weight.

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16

Eating Disorders in Transgender Individuals

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COMMON QUESTIONS

What does the term transgender mean?
How are sex and gender different?
How common is it to self-identify as transgender? Compared to other sexual minorities?
What social and emotional challenges do transgender individuals often face?
What are some of the medical challenges faced by transgender persons?
What constitutes necessary medical care of transgender individuals?
How do eating disorders affect transgender individuals?
How are eating disorders in transgender patients recognized?
How are eating disorders in transgender persons treated?
What other mental health diagnoses do transgender patients face?
What is the outcome of mental health treatment of eating disorders in transgender patients?

Case Study

V.N. is a 39-year-old transgender female of Mexican American background who presented to the eating disorders clinic for evaluation of abnormal eating after bariatric surgery in November 2018. Her history of struggles with weight and gender dysphoria began in her

preschool years and early grade school, both in New Mexico. She was encouraged to develop ability in sports, including a muscular body, but this only resulted in overeating, weight gain, and distress at her body size and shape. By her preteen years, she was binging and purging regularly. A social worker made a diagnosis of bulimia nervosa but never referred her to treatment. Her many diets only worsened her binge-purge behaviors and caused increasing weight. She experienced a severe distortion of body image, resulting in guilt, self-blame, and loathing. Her high drive for thinness and her morbid fear of fatness dominated her life. In her twenties she was employed in a bakery and a hardware store. She enjoyed food shopping and cooking but would not eat her gourmet meals.

She was convinced by age 4 that she was the wrong gender. She experienced physical, emotional, and sexual abuse during her developmental years, substantially due to her gender distress and behaviors. Because of her rebellion and cross-dressing, she was kicked out of home at age 16. During her first year of college, she married her first wife but soon separated. V.N. later married her current wife and enjoyed a blended family of her two children and three of her wife's children. They remained sexually active. She came out to her bisexual wife two years before this evaluation and began medically transitioning. Her lowest adult weight was 250 pounds in college. From that time on, her weight only increased, reaching 410 pounds (BMI 58.9) at the time of her bariatric surgery.

Despite multiple medical complications, the bariatric surgery was overall successful.

V.N. went beyond the recommendations of the surgical team and restricted her food intake to 150–200 calories per day, except when having small binges, losing almost 170 pounds, presenting to the eating disorders clinic at 239 pounds (BMI 33.4). She experienced many of the psychological symptoms and physical signs and symptoms of anorexia nervosa. She felt cold all the time, wearing multiple layers of clothing at the evaluation. Her hair fell out in clumps. She was severely orthostatic. Her constipation was intractable. Her feet were edematous. She had become more isolative.

Her history included persisting depressive symptoms. She made four suicide attempts, by overdose, hanging, and wrist cutting, all interrupted by friends or family. She abused opiate medications prescribed after multiple past surgeries for her back, hernia repair, cholecystectomy, rotator cuff repair, and vasectomy; she stopped her opiate abuse cold turkey despite great distress. She had been diagnosed in the past with PTSD, schizoaffective disorder, bipolar disorder, and major depressive disorder. Past medication trials were of limited benefit. She had one brief inpatient psychiatric hospitalization.

Her medical symptoms included spinal stenosis, morbid obesity, hypertension, obstructive sleep apnea, chest pain of unknown origin, GERD, left ventricular enlargement, and chronic pain.

On her mental status examination, she was casually dressed in androgynous clothing. Her hands showed acrocyanosis but no swelling in the parotid area. She was calm, cooperative, and established good rapport. Despite being tearful at times, she was generally euthymic. She had no psychotic features or suicidal or homicidal ideation. She was oriented with normal memory. Her insight was fair in general but limited in terms of recognizing her disordered eating symptomology. Regarding her transgender status and her journey of partial medical/surgical transitioning, she reported that she was happier than at any other time in her life. She endorsed a high fear of fatness, a relentless drive for thinness, and body image distortion, thinking of herself as still morbidly obese. She had an experience akin to phantom limb, perceiving herself so overweight that she entered the door sideways.

Her PHQ-9 for depressive symptoms was 20, severe. Her GAD-7 for anxiety was 10, moderate.

The tentative diagnoses made at the evaluation were:

1. Anorexia nervosa, binge-purge subtype
2. Persistent depressive disorder
3. History of gender dysphoria

Her formulation noted that she had most of the typical features of anorexia nervosa (AN) resulting from inducing a severe decrement in body weight to the point where she manifested the psychological,

physical, and social features of typical AN. Others might have diagnosed her with atypical anorexia nervosa, noting that her weight at presentation was not very thin. This case illustrates how the diagnosis of anorexia nervosa is best understood as a condition in which a person achieves a substantial reduction in weight so that they manifest the medical, psychological, and social features of a state of starvation not necessarily having achieved a "thin" body. Her body, by the brain's response, was exceedingly "thin" and starved. She was referred to inpatient hospitalization with a plan to transition to partial hospital after stabilization of her eating pattern, trials of appropriate medications, and an introduction to cognitive behavioral psychotherapy. Each patient's journey with eating disorders and gender dysphoria, including medical/surgical transitioning, is unique but does have shared common features.

Introduction and Terminology

The subject of transgender people has increasingly become a concern to mental health professionals and medical providers during the past decade, as well as becoming a subject of public awareness. The term *gender* has been increasingly differentiated from the term *sex*. Gender now generally means the personal and often public lived-in role as woman or man, girl or boy. *Sex* generally refers to the biological characteristics of a person, including external and internal genitalia, sex chromosomes, and sex hormones. *Gender assignment* refers to the gender given to children at birth, hence the term *natal gender*. The term *transgender* refers to individuals whose perception of gender differs from their biological sex or natal gender. *Gender dysphoria* is now a diagnostic category in *DSM-5* and refers to the presence of emotional and cognitive distress related to the difference between an individual's assigned birth sex and their self-perception of gender. Gender dysphoria may be long term or may be a transient experience, especially in younger individuals. *Transgender men or boys* identify as male in gender but were assigned female at birth. *Transgender women or girls*, conversely, were assigned at birth as male but identify in gender as female.

Nonbinary individuals may identify as neither female nor male, or as having features of both. How a person communicates their gender identity refers to *gender expression*. Surgical and medical interventions to align the physical and physiological aspects of gender with the assigned sex may be referred to as *transitioning*, *gender confirmation*, or *gender affirmation*. ICD-11 is rewording the diagnosis of *transsexualism* from the mental health section of ICD-10 and using the term *Gender Incongruence of Adolescence and Adulthood*. *Cisgender* is sometimes used to identify individuals with congruence between their assigned sex and self-identified self-perceived gender. *Sexual orientation* (hetero, gay, lesbian, bisexual, asexual, unsure) refers to a continuing pattern of sexual or romantic attraction to persons of the same sex or gender, the opposite sex or gender, or to both sexes and more than one gender.

Prevalence of Transgender Individuals

Estimates for the number of individuals self-classifying as transgender vary from about 0.17% to 0.5%, but slightly higher and much lower estimates of prevalence have been found. As a rough guide, about one-third of transgender persons seek both medical and surgical transitioning; about one-third seek only medical hormone treatment; and about one-third do not seek either medical or surgical intervention. More precise estimates await continued surveys of large populations. Nieder, Eyssel, and Köhler (2019) describe the characteristics of being trans without seeking gender-affirmative medical interventions. These individuals were more likely to identify with a nonbinary gender. They were significantly older. The main reasons for declining gender-affirming medical intervention were to avoid suffering from the transition process and being comfortable in the psychological and social transgender role without interventions.

Young people differ from adults in several ways. "Most children who exhibit gender nonconforming behaviors or who have a diagnosis of gender dysphoria in childhood do not continue on to be transgender adults" (Weiselberg, Shadianloo, and Fisher, 2019). The estimate for birth-assigned males who are transgender adults has been estimated to

be between 2% and 30%. For birth-assigned females, the estimates vary from 12% to 50%. Great caution is indicated in moving teenagers with gender dysphoria, and especially children, to medical or surgical interventions to achieve congruence with a gender state opposite to natal sex, which may well be transient.

Eating disorders in these children or teens may be used to delay puberty or to decrease pubertal changes from their natal sex development to their preferred gender, albeit a temporary preference for many. Weiselberg, Shadianloo, and Fisher (2019) give an overview of diagnosis and general psychological care and management for transgender children and youth.

Medical Issues

Medical issues in the care of transgender individuals arise from several sources. First, disordered eating practices and behaviors prior to transitioning may lead to some or many of the typical medical complications of eating disorders, including self-starvation, binge-purge behaviors, extremes of exercise, use of appetite-suppressing medications, and anabolic steroids to build muscle in males.

Second, gender-affirming medical and surgical interventions each carry substantial risk as well as potential benefit. "Care of Transgender Persons," by Safer and Tangpricha (2019) in the *New England Journal of Medicine*, provides a comprehensive guide to the medical components of transitioning as well as aftercare. They estimate that 0.6% of adults identify as transgender. The goals of transfeminine hormone treatment include the development of breasts, the reduction of facial hair, and the redistribution of body fat to a more feminine pattern. Height and voice pitch are not very responsive to hormones. With adequate hormone treatment, testosterone decreases from a male range of 300–1000 ng/dL to the typical female range, or less than 50 ng/dL. Estradiol increases to a goal range of 100 to 20 pg/mL. Androgen levels may be decreased by the diuretic spironolactone, the progestin cyproterone acetate, or gonadotropin-releasing hormone agonists. Contraindications to transgender female hormones include a history of cardiovascular disease,

cerebrovascular disease, thromboembolic disease, or breast cancer. Transgender women receiving hormone therapy suffer an increased risk for pulmonary embolism, stroke, heart attacks, and deep venous thrombosis compared with cisgender persons. Estrogen and antiandrogen treatments are started at the same time. Removal of testes will of course decrease androgen production. Beaty, Trees, and Mehler (2017) reported persistent hypophosphatemia in a male-to-female transgender patient with anorexia nervosa.

Hormone therapy to achieve a transmasculine status is accomplished medically by administration of testosterone by skin patches or subcutaneous injections to achieve a testosterone level of 300–1000 ng/dL. The goals of transmasculine hormone therapy include a deepening of the voice, the development of facial and body hair, the elimination of menses, additional muscle mass, and an increased sexual drive. Possible complications include polycythemia from erythropoiesis, pertinent in persons at risk for an elevated hematocrit, such as sleep apnea patients. Evidence is not yet adequate to advise transmasculine men whether they may suffer from increased cardiovascular disease and whether hysterectomy will avoid endometrial cancer. In addition to testosterone's effect on typical masculine anatomic features, its administration to trans men results in functional and structural changes in the brain regarding body perception (Burke et al., 2018). Nonbinary individuals may choose lower hormone levels, but it is not yet clear whether lower hormone levels predispose to bone mineral loss.

Third, aftercare for transgender individuals after transitioning requires a combination of the routine care for persons of similar age and gender, but in addition, careful attention to monitoring hormone levels and hormone effects related to transitioning. Each change in the dose of hormones needs to be followed by clinic visits to assess the results of the dosage change. The authors (Safer and Tangpricha 2019) recommend deferring transition-related surgeries except for transmasculine chest surgery until at least a year after hormone treatment. The endocrine treatment of gender-dysphoric/gender-incongruent persons has been summarized by Hembree et al. (2017). Haseen et al. (2020) express concern that transgender men who have a vagina, cervix, and/or uterus

have routine gynecological screenings to avoid marginalization in their health care, creating a more inclusive space.

Fourth, comorbid diagnoses, especially substance use, are not uncommon in transgender persons and are an additional medical and psychological burden. Virtually all programs for surgical transitioning now require abstinence from tobacco use or inhaled substances for about 6 months before acceptance. Approximately 29% of Hispanic transgender individuals reported illicit substance use (Yockey, Vidourek, and King, 2020). Transgender women and others who identify along the transfeminine gender spectrum are more likely to report use of illicit substances compared to their cisgender peers (Restar et al., 2020).

Eating Disorders in Transgender Individuals

In addition to the challenges of living and development that are associated with the onset and perpetuation of eating disorders in cisgender heterosexual or gay individuals, transgender individuals or persons with gender dysphoria appear to be subject to an increased vulnerability to developing eating disorders. The causes are certainly multiple contributing factors, which may include

1. Public discrimination, including rejection, misunderstanding, negative attitudes, abuse, violence, decreased life satisfaction, stigmatization, and denial of or barriers to medical and other services (Anderssen et al., 2020). There is some correlation between the development of eating disorders and the sum of adverse life events, comorbidity, and trauma.
2. Intrapsychic pretransition experiences of depression, loneliness, and anxiety.
3. Body image distress concerning their pretransitioning body size and shape, which may be a major factor in seeking to transition.
4. Idealized internalized expectations for what the post-transitioning body should be like, leading to disordered eating behaviors after transitioning to achieve and accentuate this body image.

Multiple studies have found an increase in eating disorders in transgender individuals. Diemer et al. (2015), based on a large study of almost 300,000 students, found that a disproportionate percentage of transgender college students (15.82%) had received an eating disorder diagnosis in the past year. In contrast, 0.55% of cisgender heterosexual men had received an eating disorder diagnosis. In between these extremes were cisgender sexual minority men (2.06%), cisgender unsure men (3.66%), cisgender sexual minority women (3.52%), cisgender unsure women (2.97%), and cisgender heterosexual women (1.85%). Coelho et al. (2019) confirmed this finding and noted that "a consistent theme across case studies was engagement in food restriction and/or compensatory eating behaviors to prevent puberty onset or progression, suggesting that for some transgender youth, these behaviors may be understood as a means of coping with gender-related distress."

Witcomb et al. (2015) also found that trans individuals had greater body dissatisfaction than controls. Trans males were comparable in body dissatisfaction to males with diagnosed eating disorders. The Drive for Thinness on the EDI-2 was greater in both cis and trans females compared to males. They noted that "both trans males and females reported greatest dissatisfaction not only for gender-identifying body parts but also for body shape and weight."

The relation of assigned sex and natal gender with later chosen opposite gender in relation to eating disorders remains complex. Vulnerability to eating disorders based on body dissatisfaction with natal gender appears to be a major factor in seeking transition to a preferred gender. In addition to body dissatisfaction being a factor in seeking transition, the transition-seeking individual may also desire the social role associated with the preferred gender. The internalized images of both body appearance and social role patterns of the desired gender may play a role in seeking transition.

Autism

An intriguing but unexplained finding has documented that individuals meeting criteria for autism spectrum disorder appear to have a higher

than matched population average of experiencing gender dysphoria. Fielding and Bass (2018) found that 8% of patients seeking transition intervention fell on the autism spectrum. Coleman-Smith et al. (2020) reported evidence of a higher prevalence of autism among people attending gender identity clinics. They noted sympathetically that these individuals were prone to suffering from feeling different and were fearful of hostility because of "having two stigmatized identities." Seeking gender transition may have been an attempt to provide congruence with one identity.

Case Reports

While large studies are ideal in understanding any psychological or medical issue, at times even small case reports may be instructive (Murray, Boon, and Touyz, 2012). One intriguing report documents how a trans female experienced a high drive for thinness with self-starvation and excessive exercise to achieve an idealized female body. This same individual, when identifying with a masculine gender, undertook intense muscle development to achieve an idealized muscular body. Similarly, a second trans female, during a time of identifying with the female gender, began to severely restrict food intake and began a rigorous exercise program to further lose weight. When this person reaffirmed his natal sex, he ceased the striving for emaciation and began an intense muscle-building program, with commensurate increase in food intake. The authors posit sagely that disordered eating psychopathology is concordant with an individual's preferred gender identity, "suggesting that the construction of one's gender identity and the construction of one's body may be interrelated . . . masculinity is linked with the drive for muscularity." In westernized societies, gender and body image appear to be closely related and associated with different but specific changes in eating behavior.

Most of the professional publications addressing the presence of eating disorders in transgender individuals reported on one to several cases. As the field expanded, larger studies were carried out. Watson, Veale, and Saewyc (2016) surveyed 923 transgender youth across Canada. They found that *enacted stigma* (the higher rates of harassment and

discrimination sexual minority youth experience) was linked to higher odds of reported past-year binge eating and fasting or vomiting to lose weight. They also noted, helpfully, that protective factors (connectedness with family and school, caring friends, and social support) were linked to lower odds of disordered eating. The largest study to date, as noted previously, involved 289,024 students and is discussed above (Diemer et al., 2015). An additional large study was carried out in New Zealand (Lucassen et al., 2019) with 7,769 students. They found that 9% of students self-designated as sexual and gender minority students (SGM), including gay, lesbian, and transgender youths. Male SGM students were significantly more likely to try to lose weight, engage in unhealthy weight control, consume fast food and takeout, and be physically inactive; they were less likely to participate in a school sports team. Female SGM students were significantly more likely to engage in unhealthy weight control, be overweight or obese, and consume fast food and takeout; they were also less likely to participate in a school sports team. SGM students were more likely to experience socioeconomic deprivation and to develop an eating disorder. Two hundred gay, lesbian, and/or transgender/nonconforming (TGNC) adults were evaluated by Bell, Rieger, and Hirsch (2018) in a study to determine mediational factors between sexual identity and eating disorders. Proneness to eating disorders in TGNC persons was related to low self-compassion, perceived stigma, and thwarted belongingness. Handler et al. (2019) reported on 417 transgender and gender-nonconforming pediatric patients. Most referrals were for requests for cross-sex hormones and/or blockers (34%), gender-affirming surgery (32%), and mental health (27%). Older patients requested hormonal and surgical services while younger patients sought out mental health services. The most frequent surgery was mastectomy.

Screening

Screening for eating disorders in trans and gender-nonconforming persons is an essential component of overall care. Avila, Golden, and Ave (2019) screened 106 young people aged 13–22 years in a gender clinic with the EDE-Q questionnaire. Fifteen percent had elevated scores. The ma-

jority (63%) were manipulating weight for gender affirmation, while 11% of natal females manipulated weight for menstrual suppression. Thapliyal, Hay, and Conti (2018) document that when gender issues were ignored by treatment providers, the result was nondisclosure of gender identity. Men were especially less likely to be assessed for an eating disorder, hindering referral to specialized treatment. Gender issues clearly affect the experience of eating disorders.

Gender-Affirming Medical/Surgical Intervention in Changing Eating Disorders in Transgender Individuals

The most common overall finding of studies on gender-affirming interventions is that cross-sex hormones may decrease body image dissatisfaction compared to the absence of such treatment. Jones et al. (2018) documented that cross-sex hormones can alleviate body dissatisfaction and thereby may alleviate eating disorder psychopathology. Van de Grift, Elaut, et al. (2017) found that hormone-based interventions and surgery were followed by improvements in body satisfaction. People without intervention continued to be significantly dissatisfied with their body compared to people who received either hormone therapy or hormone therapy and surgery. Van de Grift, Elfering, et al. (2018) documented that subcutaneous mastectomy improves satisfaction with the person's body and psychosocial functioning. Mofradidoost and Abolghasemi (2019) stress the important clinical point that "hormonal and surgical treatments in transgender persons only can eliminate body dissatisfaction to a certain extent . . . [I]t may be harder for transwomen to believe they are perceived as female by others and their bodies are adequate. Changing cultural beliefs, social values, and increasing social acceptance with the help of the media may play an important role in improving body image in the future." Duffy, Henkel, and Joiner (2019) found that 74.8% of transgender individuals with eating disorders experienced nonsuicidal self-injury; 75.2% had suicidal ideation; 74.8% attempted suicide in a one-year period. Zavlin et al. (2019) found, encouragingly, that preoperative depressive symptoms disappeared after gender-affirming surgery in the 40 patients they studied.

Summary

Individuals experiencing gender dysphoria are often misunderstood, subjected to social isolation, and may be overlooked by medical professionals in evaluations of health care, especially in their mental health needs. In addition to an increased probability of meeting criteria for an eating disorder, transgender individuals frequently suffer from self-injurious thoughts and behaviors, depression, loneliness, and self-stigmatization. As a result, the presence of eating disorders in individuals experiencing gender dysphoria may be less regularly detected or ignored. Body dissatisfaction often plays a prominent role in gender dysphoria. Eating disorders may also be a coping mechanism for other, additional stresses transgender individuals face. It is possible that gender dysphoria prior to seeking transition may have a contribution from additional sources besides body dissatisfaction, such as an envisioned social role in the desired gender. The results of body dissatisfaction often include disordered eating behaviors to change the body size or shape of the unwanted natal sex assignment before considering medical and/or surgical transitioning. Disordered eating practices constitute a method to suppress the features of the biological sex and to enhance features of the desired gender. The percentage of individuals seeking transition appears to vary according to age, desire for partial versus complete transitioning, and cultural factors such as social acceptance of living in a chosen gender role without physical transitioning. Transitioning for many, but not all, persons with gender dysphoria appears to result in less body dissatisfaction and a more comfortable social role. The possibility of significant medical and/or surgical complications as a result of transitioning needs to be well understood by mental health clinicians, general medical providers, and the transgender individual. Expert medical and psychological care before, during, and after transitioning is essential. Great caution is indicated in advancing children and teens to transitioning medically and/or surgically until a stable gender identity is fully settled in view of the high probability that gender dissonance during early years will revert to the natal gender.

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17

Psychotherapeutic Use of Medical Information

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COMMON QUESTIONS

What is psychoeducation?
How can clinicians use medical information psychotherapeutically with patients who have eating disorders?
Can you scare patients into healthy behavior?
Will medical information, if accurately presented, demoralize patients who have eating disorders?
Which medical tests offer the best possibility for psychotherapeutic use?
When is medical information not appropriate for psychotherapeutic feedback?
What information is helpful to families?
How can new genetic information help families understand eating disorders and decrease guilt?
How do patients with binge eating disorder benefit from psychoeducation?

Case Study 1

C.Y. was a 47-year-old surgeon whose wife persuaded him to come for evaluation for an eating disorder because of compulsive exercising and weight loss. The patient insisted that he was overweight at 185 pounds, 5 feet 10 inches tall, before his dieting began. Three years

before evaluation, he had begun a strenuous exercise program of long-distance running, swimming, and bicycling. His father's death at about that time had caused him to fear that he might, like his father, be at risk of death from cardiac disease. Also, his older daughter was preparing to leave for college. His weight decreased to 130 pounds. He counted calories and limited fat to 10 grams a day, including monosaturated lipids. Despite feeling cold, becoming irritable at suggestions to decrease his hours of daily exercise, and suffering two ankle sprains and knee pain, he continued his dieting and extreme exercise.

At evaluation, the surgeon insisted he was only practicing healthy behaviors. He met criteria for anorexia nervosa in a male but would not accept the diagnosis until his DEXA scan and testosterone blood levels were shared with him. He met the criteria for osteoporosis with a femoral neck bone mineral density of T = –2.7 standard deviations below normal and an L1–5 finding of T = –2.3 standard deviations. His testosterone was 180 ng/dL (normal 340–800). Gentle confrontation with these data allowed him to accept treatment, which included moderate exercise but at a much more reasonable level, along with a gradual increase in weight to 165 pounds. Through his psychotherapy, he came to terms with his fears of mortality and established a more adult-to-adult relationship with his daughter, including acceptance of her transition to becoming a college student. His marriage improved, including his ability to communicate with his wife, as well as their marital relations. Providing him with information about bone density loss in males with anorexia nervosa (Andersen, Watson, and Schlechte, 2000) was helpful, as he was a clinical researcher as well as a surgeon.

Case Study 2

S.J. was a 20-year-old engineering student at the top of her class. In the course of her eating disorder, in addition to restricting her food intake, she began to abuse laxatives. She developed significant electrolyte imbalance, which was corrected in treatment. She was presented with information in a published study that 80% of calories

ingested are absorbed despite laxative use that was excessive enough to lose 6 liters of diarrhea fluid a day (Bo-Linn et al., 1983). When she saw this information, she said, "It doesn't make sense to do things that don't work" and stopped using laxatives. She went on to recover her weight and ceased all purging behavior, as well as changing her core beliefs that thinness was essential for her sense of control and effectiveness. Medical information produced a helpful psychotherapeutic response.

Background

Eating disorders are characterized, according to Hilde Bruch (1979), by denial of thinness, denial of illness, and denial of sexuality. Patients with eating disorders, especially anorexia nervosa (AN), often live an intellectualized life, existing largely from the neck up, with little attention to the reality of what is happening to their bodies. They often appear to produce "insightful" but only intellectualized responses in psychotherapy without any resulting changes in illness behaviors or illness-based thinking. Clinicians can use medical tests that document significant changes in body function in several different ways when interacting with patients who have eating disorders. First, medical information may simply be screened from the patient and never presented (the paternalistic style). Second, information may be presented in a neutral manner with neither psychotherapeutic discussion nor blame (the *Dragnet* style: "Just the facts, ma'am"). Third, information may, unfortunately, be used to blame or scare patients (club the victim). Scaring patients does not last for long, certainly not long enough for motivation and engagement in successful treatment. Last, and preferably, information can be used psychotherapeutically (the ideal use). Psychoeducation means using information about the nature of the disorder and the medical test results in a manner that respects the patient, empowers them to become a partner in decision making, and helps draw out the implications of accepting versus refusing treatment. Accurate and therapeutically presented information is vital for families to become cotherapists, and to relieve their apprehension, guilt, and self-blame. With psychotherapeutic

information, they come to understand that eating disorders are medical and psychological illnesses with genetic contributions.

Using Medical Tests Psychotherapeutically

The goal of using medical information psychotherapeutically is to form a therapeutic alliance with the healthy side of the patient to overcome the patient's denial of illness, or denial of the seriousness of the illness. It is an attempt to form a working partnership with patients to help them interrupt the belief that they do not have an illness or that it has no significant consequences. Blaming patients for medical consequences is inappropriate because often they have already been blaming themselves. Scaring patients doesn't work; at best, it produces a short-term alteration in behavior, and at times despair or hopelessness.

The following are suggestions for possible uses of medical information based on experience but not on any established studies with statistical confirmation. A good background reading for the clinician is the chapter on psychoeducation in Garner et al. (1985), which has stood the test of time. Presenting accurate medical information, both from current medical testing and from expected symptoms during recovery, will reassure the anxious, fearful patient. It will also increase respect for the staff through patient awareness that this illness is understandable and predictable to the staff, as well as treatable. The following examples of medical information can be used therapeutically (Yager and Andersen, 2005).

Vital Signs and Acute Medical Instability

When a patient is evaluated for diagnosis and found to have electrolyte abnormalities with associated EKG changes, information about these test results may help the patient accept the diagnosis as a serious concern as well as accept the need to begin treatment; or, if the patient is already admitted, this information can help them accept the need for team-directed treatment instead of self-directed efforts. This information generally is best presented by using the laboratory reports themselves, which show the normal levels as well as the patient's abnormal

results. Along with presenting medical information, explaining their significance and cause is helpful so that the prescribed treatment will make sense. Unfortunately, many patients with restricting AN have normal laboratory values despite marked cachexia. Fairburn (1995) reviewed the physiology of anorexia nervosa. AN patients may die with relatively normal labs.

Bone Mineral Density by DEXA Scan

A dual-energy X-ray absorptiometry (DEXA) scan often provides graphic visual evidence that the patient has a serious medical problem. This information is usually first received by the clinician in the form of the patient's findings on a graph that compares the patient's results with those of normal, same-gender individuals at different ages. It is easy to show patients with AN, when their findings are traced across the graph, that they have the same bone density as 80-year-old individuals. A typical response is, "Oh, my goodness. I never realized that this was happening," or, "I didn't know it was this serious." The patient can be reassured that the problem, while possibly not completely corrected by treatment, will be treated with increasingly effective methods. Some of the facts to present include the following explanations: because lifetime maximum bone density is usually reached, at the latest, at some time in a person's twenties (some recent studies suggest by age 17 or 18), with the maximum rate of accumulation in the midteens, there is urgency to building a healthy "bone bank for after retirement." Active research is taking place to address the problem, with assurance that most young patients with deficient bone mineral density still have several years to correct the deficiency, which responds slowly to treatment. Incidentally, recent studies have demonstrated that estrogens are relatively ineffective in the starved AN patient (Mehler and MacKenzie, 2009), another reason for restoration of full body weight and low-impact weight-bearing exercise. The DEXA scan, as noted in previous chapters, is widely available, moderate in cost, accurate (Winston, Alwazeer, and Bankhart, 2008), and low in radiation (one-tenth that of a chest X-ray).

Magnetic Resonance Imaging

While a brain MRI scan is not indicated in all patients, eating disorder patients with atypical features or a history of seizure or brain injury require an MRI for differential diagnosis and to assess the contribution of structural brain change to mental functioning. When an MRI is obtained for an AN patient, it often shows enlarged ventricles and decreased brain volume. Patients may be astounded to see that their MRI is similar to that of a patient who has Alzheimer's disease, which also is often associated with enlarged ventricles and decreased brain substance. Assurance can be given that most recent studies demonstrate recovery of brain size in anorexia nervosa with effective treatment including weight restoration, but it is not certain that all brain abnormalities are eventually completely improved (Swayze et al., 2003).

Gastrointestinal Changes

Patients can be shown published studies (Waldholtz and Andersen, 1990, for example) showing that abdominal distress is improved with renutrition and supportive medical care in most patients with AN without medication or GI studies. Patients find it difficult to concentrate during therapy when there is significant ongoing abdominal distress. Published information about the return to normal gastric emptying, decreased bloating, and normalization of the speed of GI transit time helps the patient tolerate the temporary refeeding symptomatology affecting the GI tract because of the knowledge that it is temporary, will improve, and is a diminishing consequence of the illness. Anorexia nervosa doubles the transit time of the gastrointestinal tract.

Signs of Medical Starvation

Discussion of the signs and symptoms of medical starvation may help the patient accept the fact they are medically starved and not simply healthy in a way others should envy. Demonstrations of low T3, the presence of

increased reverse T3, signs of acrocyanosis, and small, shrunken ovaries on sonogram are all evidence that the body is truly starved. It may help to refer to the studies of Keys et al. (1950) in *The Biology of Human Starvation*, showing that many of the psychological changes and much of the social isolation and altered eating behavior associated with some forms of eating disorders come from medical starvation and are not the core psychopathology of anorexia. "Marcie, that slowness of thinking and the difficulty in finishing the personal journaling assignment come from the effects of starvation on the brain. They will improve soon." Most of the medical symptoms in anorexia nervosa are understandable as the biology of starvation.

Status of Reproductive Hormones

The presentation of information documenting low estrogen in the female or low testosterone in the male and comparing these results to normal values may help individuals accept the fact that they have an illness. Even though patients with AN often avoid discussions about sexuality, many want to have families in the future. Information about their low reproductive hormone levels may help them accept necessary weight restoration. The subsequent demonstration that these values usually become normal with return to healthy weight will often reassure patients and especially parents that no permanent damage has occurred. "There is nothing broken. Your reproductive hormones have gone back to a prepubertal state, and they will almost always return to normal when your weight is fully normal, when your eating pattern is normal, and when your stress levels are lower. The body has mechanisms for dealing with famine from humankind's long experience with shortages of food. The body thinks you are experiencing a famine. When you let it know that the 'famine' is over, the hormones will return to normal over several months." It is important for women who anticipate being sexually active to know that they may become pregnant before their period resumes because the ovum is shed before that time, and that they therefore should take appropriate precautions.

Allergies and Complaints of Lactose Intolerance

Nutritional rehabilitation is substantially interfered with or resisted if patients have significant food allergies and/or lactose intolerance. Some patients complain that virtually all foods they eat cause allergic responses. Others complain of GI symptoms after eating dairy products, denying themselves their beneficial protein and calcium content. Refeeding will often be greatly assisted by requesting a formal consultation early in treatment for breath hydrogen testing to confirm or rule out lactose intolerance and allergies to specific foods. About 80% of patients complaining of lactose intolerance do not have confirmation on objective testing. Similarly, many fears of a generalized allergic response to foods or additives are also not sustained on medical testing. Normal responses on these tests will allow nutritional rehabilitation to proceed with as few limitations as possible and will generally decrease the phobic quality of these foods in the patient's mind.

Abuse of Laxatives

As noted in the second case study in this chapter, information about the ineffectiveness of laxatives may help patients undergo a program of cessation or tapering of laxatives by recognizing their ineffectiveness in producing the desired goal (see Bo-Linn et al., 1983).

Night Sweats

The first patient in whom night sweats were noted in our service was a pathologist whose occupation brought her in contact with various potentially fatal fungal disorders associated with a symptom of night sweats. An extensive evaluation was undertaken in this patient for TB and other fungi, but all results were negative. There was no evidence of any medical disorder besides the medical starvation of anorexia nervosa. When this pattern was asked about in subsequent AN patients undergoing weight restoration, they all acknowledged night sweats as a phenomenon.

Night sweats should be discussed with patients because when its normality as a phenomenon is not recognized, it can worry patients or families, may result in iatrogenic and unnecessary tests to be ordered, or may be brushed off as psychosomatic. Medically starved patients can be alerted in advance to expect, usually within two to three weeks of being hospitalized, that their feelings of coldness and objective hypothermia will improve in a sequence: first, they will experience night sweats, sometimes drenching and requiring a change of night clothing, usually starting about two or three weeks into treatment; then there will be a leveling off of the sweating gradually over the next two weeks; and, finally, there will be a return to a state of normal temperature control without subjective coldness. Patients sometimes give increased respect to staff in place of skepticism when the prediction of night sweats is confirmed by experience. Interestingly, this phenomenon is observed in males as well as females, so increases in estrogen may not be involved; rather, some aspect of hypothalamic regulation in the setting of increased nutrition, including change in thyroid hormones or receptors, are the likely cause.

The Patient with a Binge Eating Disorder

Patients with binge eating disorders may also benefit from psychotherapeutic presentation of medical information. Foremost, they need to understand the benefits, risks, and alternatives to no treatment versus behavioral treatments. They may or may not understand the multitude of medical risks associated with any accompanying obesity, which is frequently present. When morbid obesity is present, it is helpful to present patients with the pros and cons of bariatric surgery. Recent studies have demonstrated a decrease in all-cause, long-term mortality that occurs after morbidly obese patients undergo successful surgery (Carlsson, Sjöholm, Jacobson, et al., 2020).

Many patients with a combination of binge eating disorder and morbid obesity are already aware of the health hazards of their combined disorders. The second most important reason for seeking surgery, after improvement in health, is to improve appearance, a reasonable expectation,

with the exception of a problematic redundant panniculus. Information about subsequent surgery to remove excess abdominal skin can be reassuring.

Collaboration between medical providers and the behavioral health team is essential, but too often not practiced. Generally, bariatric surgery will not be approved until a patient ceases binge eating. Teaching a patient suffering from binge eating associated with severe or morbid obesity about the potential benefits of treatment with a combination of psychotropic medications and psychotherapy to achieve interruption of the abnormal eating pattern will often alleviate the discouragement of not being immediately accepted for surgery.

It is essential for patients undergoing bariatric surgery who have ceased in their binge eating pattern to form a long-term alliance with a behavioral health clinician for aftercare to avoid the recurrence of post-surgery binge eating, which will diminish or abolish the benefits of the bariatric surgery. Pinto-Bastos, de Lourdes, Brandão, et al. (2019) documented that higher postoperative scores on the Eating Disorders Examination Questionnaire and grazing behavior were associated with increased weight regain. At times, the new body image and decreased BMI of a patient, often half of the previous body weight, requires some psychotherapeutic work for the patient to become comfortable in a new social and intrapsychic role.

When Is It Not Helpful to Give Medical Information to a Person with an Eating Disorder?

Clinical experience suggests that in several situations, sharing medical information is not completely appropriate. Very young patients may simply not be able to understand the nature of medical data. If they are too young or too starved, or have substantial deficits in intellect, making it difficult to form a working therapeutic relationship especially for cognitive behavioral work, then medical information may produce only fear and no therapeutic response, or a negative preoccupation. Some patients with vulnerable personality traits may internalize medical information to increase self-blame, but this can be anticipated and prevented by the

manner of sharing information. Information sharing with patients who are self-critical or depressed at the time the medical information is available may need to wait until the depression is improved and/or a psychotherapeutic relationship has been established. The goal of sharing medical information is not to frighten patients or increase their self-blame but to form an alliance with the patient against the illness.

One form of medical information, in our experience, is not helpful to most patients with anorexia nervosa: daily information about the patient's weight during intensive inpatient programs, and sometimes not even in partial hospital or outpatient programs. A preoccupation with daily weight may result in learning to "eat their way out of hospital" by complying with the behavioral goals but not changing core beliefs; weight change without psychotherapy is not adequate therapy. Not everyone agrees with this advice, but in our experience, daily sharing of the patient's body weight, which is more akin to daily confrontation, has more drawbacks than benefits. A focus on weight obscures the fact that eating disorders have essentially nothing to do with food, weight, or shape, but are instead strategies to deal with issues in living, mood regulation, relationships, an adequate sense of control in life, and low self-esteem.

Obviously, a time comes when patients need to know their weight. We usually tell them their weight and their goal weight range when they are in a healthy target range or have transitioned from inpatient treatment to partial hospital treatment at 85%–90% of healthy weight. At that time, they are taught the rationale for a goal of a 4- to 5-pound weight range, rather than a single number—for example, 120–25 pounds for a 25-year-old woman who is 5 feet 3.5 inches tall. They are told that it is normal to experience fluctuation within that 4- to 5-pound range, but not to alter their nutritional pattern. They are asked before that time to concentrate on understanding themselves and changing their thinking and behaviors that form the core of their eating disorders while they trust the team to prescribe nutrition as medication. We promise them, based on more than 40 years of experience, that we will not let them become overweight. At times, a patient with a personal and family history of obesity may need to have a target weight 10–15 pounds above the so-called ideal weight. Weight, like height, is a bell-shaped curve, and someone has

to hold down the higher standard deviation above the average, although few women accept this fact. It is an important therapeutic goal to persuade the patient that the true goal weight will be determined during the year after intensive treatment, when the patient and therapist will both be determining where the body functions well, using information on labs, return of normal temperature regulation, return of normal hunger and satiety, regular menses, and return of normal sexual drive and function.

The Role of Parents

Appropriate medical information may also reassure parents that the physical symptoms of the eating disorder are being addressed and that their daughter or son will not die. Parents often fear the worst. If the situation is desperate medically, certainly the possibility of a fatal outcome should not be withheld, but often parents' fears are greater than the situation warrants. Parents appreciate learning the difference between starvation-induced, reversible symptoms versus serious, short-term or long-term complications that need to be separately addressed with specific medical treatments. They may need the appropriate medical information if they, too, are doubtful of the need for treatment and have their own form of denial of illness. Parents are often reassured to know that few, if any, medical consequences are permanent. They usually welcome information on the increasing data concerning the genetic contribution to the predisposition to eating disorders. This information allows them to consider the eating disorder as a true illness, and not the fault of the family or the patient. At times, parents may disclose that they or another family member have struggled with an eating disorder or with other illnesses in the spectrum of eating disorders vulnerability, such as depression, anxiety disorders, or OCD.

The Method of Presenting Medical Information

Medical information is psychotherapeutic when it is presented as part of a planned approach aimed at forming a therapeutic alliance to defeat

the denial of illness that is characteristic of eating disorders. Information is most convincing when it is shown on a formal report in which the patients themselves can see the results and, especially, when they can compare their results to normal values. Visual appreciation by the patient tends to break down the intellectual denial of illness. It may also help patients understand why they have difficulties with mental concentration and abdominal discomfort, or why the treatment team or clinician has prescribed limitations on high-impact activities and so on. Information on the reversibility of reproductive hormone abnormalities may reassure patients who wish to have a family later. Medical information presented therapeutically rather than in a critical or even neutral manner may enable patients to give themselves permission to accept the refeeding process rather than being fearful or hostile recipients of nutritional rehabilitation.

Accurate medical information also increases respect for staff knowledge when the patient is told about upcoming symptoms that probably will occur during treatment, how they will be treated, and their transience. This includes information about possible night sweats during the refeeding process, gastrointestinal discomfort, refeeding edema, changes in mental concentration, and progressive increases in energy, to name but a few.

Summary

Conveying medical information about the physical consequences of eating disorders to the patient is important so that the treatment team can give good care. In certain situations, or if the information is not presented properly, some vulnerable patients may receive the information critically, with negative effects on the therapeutic relationship. But if the information is presented as we recommend, psychotherapeutically, it can be used to form an alliance with the patient, to establish the reality of illness, and to diminish denial of the illness or denial of the potential seriousness. The psychotherapeutic use of medical information in ARFID patients, considering the variety of causes and the large age range, is not well documented but may reasonably be inferred.

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18

Ethical and Medico-Legal Considerations in Treating Patients with Eating Disorders

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COMMON QUESTIONS

What bioethical principles guide care of patients with eating disorders?
What is coercion?
What is the difference between certification and guardianship?
Why is it harder to certify a patient with anorexia nervosa than a patient with other psychiatric illnesses?
Are there specific criteria that can be used to certify a patient with anorexia nervosa?
Do patients with anorexia nervosa who are treated involuntarily do as well as patients treated voluntarily?
What is harm reduction?
What is the difference between palliative care and hospice care?
Does a patient with a severe eating disorder have to be competent to make the decision to reject treatment and begin hospice care?

Case Study 1

K.A. is a 35-year-old woman diagnosed with anorexia nervosa binge/purge subtype. She was first diagnosed with an eating disorder in her early teens. She was treated on seven occasions and was eventually able to live and function albeit at a low weight. She maintained this weight for eight years. She then began having abdominal pain. K.A.

was diagnosed with gallstone pancreatitis. She underwent multiple abdominal procedures and surgeries, as a result of which she began to use pain medications excessively. She also suffered from depression and anxiety but had a supportive family who set strong boundaries regarding the need for treatment. In addition, her family controlled her finances. K.A. was admitted to an inpatient level of care. At the time of admission, she weighed approximately 65 pounds (her height was 63 inches; thus she was at 54% of ideal body weight). She refused to eat and would not accept nasogastric (NG) feeding. Her treating psychiatrist petitioned for inpatient certification and requested court-ordered medications and involuntary tube feeding. K.A. was subsequently certified, and the court approved the petition for court-ordered medications and tube feeds. She required a physical hold to insert the tube and initiate feeding on several occasions. The certification was renewed at three months, uncontested by the patient. During the weight restoration process, she was transitioned to 100% oral nutrition and restored to her goal weight. She was discharged to outpatient treatment. Four months later, K.A. returned for a short hospital stay as a voluntary patient after having lost 10 pounds. She was again readmitted approximately a year thereafter, severely ill. Following a prolonged hospital stay, which included certification, she was discharged to outpatient care, maintained her goal weight, and was engaged in gainful employment.

While this patient had a long eating disorder history and several protracted hospitalizations, she was nonetheless eventually able to achieve and maintain a reasonable level of recovery. Certification was helpful in that it kept her in treatment when her body weight was so low that her cognitive distortions did not allow her to restore weight. In addition, having family who were able to maintain strong boundaries with her and support the treatment team was important in helping her through treatment as, even if she had eloped from treatment to escape the certification, her family would not have continued to support her financially, and she would have had few options other than to return to treatment.

Case Study 2

Y.D. is an 18-year-old female diagnosed with anorexia nervosa, restricting subtype. She was transferred to an inpatient eating disorder treatment program following treatment at a medical unit, where she had been admitted weighing 74 pounds. During her stay on the medical unit, she was fed via nasogastric tube under guardianship laws. Y.D. had been treated on 11 prior occasions since age 11. She had required forced tube feeding in the past and had eloped from prior treatment facilities. She was diagnosed with comorbid post-traumatic stress disorder, depression, and conduct disorder and transferred from the medical unit to the inpatient eating disorder treatment unit on a mental health hold. The treating psychiatrist petitioned for certification. Y.D. was certified, and the petitions for involuntary medications and NG tube feeds were granted. During her course of inpatient eating disorder treatment, she removed her NG tube on multiple occasions, purged via the tube, and had several episodes of syncope. Y.D. refused to transition to an oral diet, despite the promise of more privileges on the unit, with a chance to transition to a lower level of care and go on outings off the unit. She "cheeked" the olanzapine and lorazepam she was prescribed and saved these medications, which she then took all at once. She told emergency room staff that her motivation for the overdose was that she was hoping to be discharged from eating disorder treatment. Emergency room staff opined that Y.D. was too ill from an eating disorder standpoint to be admitted to an acute psychiatric unit, a procedure that would normally be considered after a deliberate drug overdose. Following her return to the inpatient eating disorder unit, she continued to repeatedly remove the NG tube (to such a point that her nostrils became inflamed and further NG tube insertion was considered harmful). In terms of her willingness to engage in recovery, Y.D. remained amotivated. As her weight was no longer critical (she had gained more than 20 pounds through tube feeding), the team agreed she had reached maximum benefit under certification and petitioned the court to drop the certification. Y.D. agreed to minimal oral intake (just enough to ensure her weight and vital signs remained

stable while disposition was arranged). She was discharged home at 85% IBW (106 lb.) and has not returned for further treatment to the facility that certified her.

While this patient certainly had the potential to recover, her comorbid diagnosis of conduct disorder led to her flouting rules on every occasion she could, increasing her resistance to treatment and decreasing her compliance with the treatment team. In addition, her parents differed on how best to proceed with treatment, and differences of opinion between the parents (and between parents and treatment team) greatly affected her care. The treatment team terminated her care when she was medically stable enough to leave and it would have been more harmful than helpful to continue NG feeding against her will.

The Principles of Modern Biomedical Ethics

Four guiding principles form the basis of modern biomedical ethics:

1. Respect for autonomy: The patient's informed choices take priority.
2. Beneficence: We as medical professionals should do what we think is best for patients.
3. Nonmaleficence: First, do no harm—*primum non nocere*.
4. Justice: Balance individual and social costs, benefits, and risks.

Nonmaleficence and beneficence have historically been honored. In recent decades, respect for autonomy has become of increasing importance in medical practice as the narrowing of socioeconomic and educational gaps between physicians and patients have altered the doctor-patient relationship to more of an egalitarian model (Strous, Ulman, and Kotler, 2006). Social justice is the concept of fair, equitable, and appropriate treatment in light of what is expected or owed to members of society.

These four guiding principles are applicable in most situations involving patient care. Nonetheless, there are conflicts within and between these principles that are relatively specific for anorexia nervosa (AN) because of the unique features of this psychiatric illness:

1. the interrelationship between effects of medical starvation on cognition and the behavioral consequences of overvalued beliefs underlying the disorder
2. the perception that AN is a voluntary disorder responsive to self-determination in both the development of the illness and the process of regaining health
3. the moralizing that disorders of eating behavior and weight result from personal failure
4. the common stereotype of AN as an illness that affects only teenage Caucasian females
5. the relatively robust appearance and behavior of many patients even with serious AN
6. the multifactorial concept of etiology that generates competing approaches to treatment
7. the reality of living in a society with increasing health care costs and the need to use health care resources wisely

The following sections weigh these guiding principles against the reality of treating eating disorders.

Respect for Autonomy versus Beneficence

To respect autonomy, one must acknowledge that patients have a right to hold views, make choices, and take actions based on their values and beliefs. Two concepts are essential for autonomy: liberty (independence from controlling influence) and agency (capacity for intentional action). Strictly speaking, competence may be determined only by a judge, and the word *capacity* is used in the clinical context. Yet, that distinction is not supported by terms used in the statute or in court decisions across the United States—thus the words *competence* and *capacity* can be used interchangeably. Capacity may vary over the course of a patient's lifetime or illness. The desire of patients to receive information and make decisions is not uniform across cultures but influenced by their culture and community. It should not be assumed that all patients want the same amount of information regarding their condition, or the same amount

of autonomous choice. Respect for autonomy does not extend to persons who cannot act in an autonomous manner: for example, patients who are immature in age, incapacitated, and suicidal.

Marceta (2020) wrote that "the relevant problem in practical bioethics is not to determine what is authentic but to determine what is inauthentic" and argued that it is justified to deem a desire inauthentic to the extent that it is due to causal factors that are alien to the person and deviates from that person's usual identity. Regarding anorexia nervosa, an individual's desire to harm himself or herself that is borne out of the illness itself is an alien, inauthentic, and pathological desire that thwarts the liberty and authentic values of that individual. Thus, it is reasonable to question whether persons with AN are truly autonomous in this state.

Individuals who have severe anorexia nervosa often appear more authentic than they really are, making promises they are unable to keep. A frequent statement made by individuals with AN is, "If you don't make me do inpatient treatment, I promise I will gain weight at home." Those with AN are intelligent and articulate and often able to convince relatives as well as treatment providers that they are doing quite well when, in fact, they are not. According to Appelbaum and Rumpf (1998), emphasis should be placed on patients' actions rather than their intentions. Patients with severe anorexia nervosa give the illusion of sanity even when they are driven by deadly irrationality, failing to appreciate that AN has the second highest death rate of all psychiatric illnesses after opioid abuse.

The principle of beneficence is the moral obligation to act for the benefit of another. Beneficence entails protecting and defending others, preventing harm from occurring to others, and removing conditions that will lead to harm. These rules present positive requirements for action. The idea that beneficence is the primary obligation in health care dates to the time of Hippocrates, and physicians have historically relied mostly on their own judgments about what is in the best interests of the patient. In many cases physicians override the wishes of the patient and justify this action by citing the goal of benefiting or reducing harm to the patient. This is termed *paternalism*. As modern medicine has led to an emphasis on a patient's rights to receive information and make in-

dependent judgments, however, paternalism is being questioned to a greater extent (Strous, Ulman, and Kotler, 2006).

Carefully delineated respect for autonomy combined with experienced assessment of mental and behavioral functioning in anorexia nervosa will determine when beneficence trumps autonomy.

Authentic Beneficence versus Authoritarian or Pseudo-Beneficence

True beneficence does not imply a patronizing or an authoritarian approach. On one hand, an out-of-date form of beneficence fails to discuss with the patient the diagnosis, treatment options, prognosis, benefits, and risks of having treatment or not. The "doctor knows best" attitude falls into this category, as does limiting the treatment options to overly narrow options, consisting only of the skill set of the clinician, even when not best suited to the needs of the patient. On the other hand, a false view of updated beneficence presents treatment options via a "cafeteria approach," with no guidance or specific recommendations. "Whatever the patient wants" is pseudo-beneficence and not in the best interests of the patient. Ethical clinicians give patients a clear understanding of the diagnosis, treatment, and prognosis of anorexia nervosa and offer tailored recommendations based on their experience and evidence from sound studies and clinical practice. Clinicians should also acknowledge limitations in their training and accede to a request for a second opinion or a referral when appropriate. Not every clinician who can competently diagnose eating disorders can also treat all forms of eating disorder at every level of severity. Recognizing one's limitations is essential and referring to a higher level of care when needed is in the patient's best interests.

Authentic beneficence grows out of empathy and appreciates the patient's struggles with giving up the perceived benefits of illness (anorexia is often ego-syntonic) for what seems to be perceived as a loss of control resulting from becoming healthy. Beneficence will acknowledge that although the patient's physical concerns (hair falling out, dry skin) may seem to be clinically less important than the threat of death or

chronic illness, these concerns need to be incorporated into a treatment plan. Authentic beneficence always includes a biopsychosocial formulation that integrates palpable concern for patient suffering with the best available interventions, treating the medical, intrapsychic, and interpersonal aspects of the disorder. Authentic beneficence also takes into account that inauthentic or pathologic values due to AN may be taking hold and exerting undue influence on an individual. This may lend itself to paternalistic intervention in the form of medical professionals restricting the present autonomy of an individual with AN to promote their future autonomy.

Nonmaleficence versus (Unintended) Maleficence

The principle of nonmaleficence obligates us to abstain from causing harm to others. In addition to the obligation of beneficence, the Hippocratic Oath incorporates the obligation of *primum non nocere*—first, do no harm. The rules of nonmaleficence are negative prohibitions of action (they tell us what *not* to do) and provide a moral basis for legal prohibitions of certain forms of conduct. As medical care has become more sophisticated, so has our thinking regarding when it is appropriate to override the prima facie obligation to treat. Examples of when we override the obligation to treat are when treatment is futile, or when the costs or burdens of treatment clearly outweigh the benefits. Much controversy still exists in this domain, however, and determinations regarding what is considered active killing (versus letting die) continue to evolve. Active and passive forms of harm may occur in several other ways. An example of active harm is inappropriate refeeding (e.g., refeeding too quickly, albeit well intended). Another example of unintended maleficence in the active form may be deciding to certify and force-feed a patient with severe and enduring anorexia nervosa without fully considering the patient's treatment history, the wishes of the patient and family, and whether pursuing the goal of full weight restoration will have a reasonable probability of achieving remission. Conversely, consideration of withdrawal of active treatment and deeming care for an

individual with an eating disorder "futile" may not be an appropriate response to adolescent AN, given that a large majority of such individuals can be expected to recover (Forbes, 2020).

Social Justice versus Business as Usual

Social justice views the patient's disorder as a tapestry in which individual and societal costs, needs, and benefits are interwoven. The term *distributive justice* refers to fair, equitable, and appropriate distribution of benefits as determined by the norms of society. The patient is a de facto part of a broad social network of family members, caregivers, communities, governmental regulators, third-party payers, legislators, and judges. While each component of this social network in theory has the patient's welfare at its center, in practice, each has its own self-serving goals, which may conflict with what is in the patient's best interests.

Legislatures have previously given lip service to parity, but seldom has this resulted in an impartial approach to all illnesses as worthy of comparable levels of care based on intensity of illness. Psychiatric disorders have more limited health care insurance benefits than "medical disorders," and eating disorders specifically have seen the most stringent limitations in coverage. In contrast, "medical disorders" may have almost unlimited benefits. The result has been shorter inpatient treatments for AN despite evidence-based studies documenting that inpatient treatment short of full weight restoration leads to the propensity for a patient to relapse and be readmitted to inpatient care (Steinhausen et al., 2008). Repeat hospitalizations are considered "business as usual," while adequate length of stay is considered a money-losing proposition, although it may end up costing less over the course of illness.

True social justice does not tolerate disparities in service based on race, gender, or sexual orientation. Failure to reach out to diverse ethnic and racial communities, which data increasingly demonstrate have a high and increasing prevalence of eating disorders, represents an injustice.

Advocacy for social justice includes a call for comprehensive treatment programs and adequate reimbursement of care, as well as a call for

funding of both clinical and translational research studies. Increased knowledge of anorexia nervosa will lead to clinical advances, community education programs, screening for early diagnosis, and preventive intervention in demonstrated areas of effectiveness. Social justice advocacy also calls for an end to stigma regarding eating disorders, interaction with legislative bodies for further actualizing parity, and a willingness to appear before administrative justices and other personnel within the justice system to plead the patient's case for funding. States that currently do not support involuntary treatments for patients with severe anorexia nervosa, even when clearly necessary in life-threatening cases, need to remediate this deficit, in view of evidence that selective involuntary admission and treatment can be beneficial as well as lifesaving (Elzakkers et al., 2014).

Huskamp et al. (2018) examined mental health service use from 2007 to 2012 after federal parity implementation among individuals diagnosed with eating disorders for whom financial access to care has often been limited. The study examined whether Mental Health Parity and Addiction Equity Act (MHPAEA) implementation was associated with changes in mental health utilization and spending in this specific population. The study concluded that federal parity law's implementation was associated with increased intensity of outpatient mental health service use with individuals diagnosed with ED, but no increase in out-of-pocket expenditures, suggesting improvements at some level of financial protection. Results of this study show that the vast majority (85%–90%) of the sample was female and 13%–29% were under the age of 25 (Huskamp et al., 2018). Between 37% and 44% of individuals had a diagnosis of co-occurring mental health or substance use disorder. Huskamp et al. (2018) found that parity was associated with increases in the total number of outpatient mental health service users, but there was no association between MHPAEA and the number of inpatient mental health days or outpatient medication management visits.

Whiteford and Striegel Weissman (2017) explored six factors that have been identified as commonly playing a role in health-financing decisions and discussed these factors as they apply to eating disorders. The six factors were (1) size of health burden arising from disease or

health condition, (2) availability of cost-effective treatments, (3) ability of private markets to efficiently deliver health services for the condition, (4) catastrophic costs associated with treating the condition, (5) negative externalities associated with nontreatment, and (6) the "rule of rescue." In the Global Burden of Disease 2015 studies, mental disorders account for 7% of total global disability adjusted life year (Whiteford and Striegel Weissman, 2017). The combined burden from AN and BN made eating disorders the 12th leading cause of physical and mental health–related illness. There are thus compelling reasons for government involvement in mental health care. The burden of mental disorders is high, and there is increasing evidence that cost-effective interventions can be delivered. Whiteford and Striegel Weissman also concluded that the private market without regulation does not deliver mental health services equitably, and the costs of accessing treatment can be debilitating to patients and their families. Without services, costs to the community and predicted outcome for the individual is increased disability and premature death.

Involuntary Treatment

Coercion

Coercion, in medical care, is the practice of persuasion by using force or threats. It may exist informally (e.g., treatment contracts, achievement of certain privileges if a patient demonstrates compliance with treatment) or be achieved through legal measures (i.e., involuntary treatment). Although patients may not outright refuse to enter treatment, many patients with AN feel they are coerced (albeit not involving legal measures) to enter treatment, either by family or by outpatient treatment providers who will no longer provide care for them unless they agree to complete treatment at a higher level of care. Coercive pressure to enter treatment does not necessarily undermine clinical progress or the formation of a therapeutic alliance (Schreyer et al., 2016). In addition, within weeks of entering treatment, patients are often able to acknowledge that, in retrospect, they needed treatment, even though they felt coerced at the time of admission (Guarda et al., 2007).

Despite studies that suggest coercion may produce a favorable outcome, Kendall (2014) commented that the use of coercive strategies in patients with AN indicates that "persons with anorexia nervosa are paradoxically understood as possessing the competence to make decisions to comply with treatment but not to refuse it." Accordingly, when patients enter treatment either voluntarily or as a result of coercion, their decisional capacity is not assessed. When they refuse certain aspects of their treatment, however, their decisional capacity becomes an issue. Thus when clinicians consider involuntary treatment based on a patient's refusal of a particular treatment goal, this decision appears to be made just because the patient is not complying with the plan of the treatment team, rather than based on that patient's incompetence.

Coercive approaches also engender concern as to whether a patient's autonomy or liberty interests are being protected. If patients are compliant only to avoid involuntary treatment, their decision to comply with treatment may not be truly autonomous, and they are in effect involuntary patients without the protections of the law that are afforded to patients who are being certified.

In addition, although individuals with AN typically exhibit denial, that denial is not a constant state but a dynamic process (Túry, Szalai, and Szumska, 2019). The continuum ranges from intense denial and noncompliance early in treatment to a notable improvement in compliance as the patient's insight improves. This transition is not necessarily linear, however, and the individual may go back and forth between these stages during treatment. Túry and colleagues wrote, "If the patient denies the illness then every therapeutic intervention is subjectively perceived as compulsory; thus, a lack of insight may mean that some kind of coercion is unavoidably part of AN treatment." They also suggested that the patient's perception of coercion is complex and not necessarily associated with the degree of restriction in the patient's freedom but with the quality of relationships with the treatment team and the ability to form an alliance with that team. A coercion paradox is described as one in which an individual with AN (despite denials of needing help) essentially coerces others to coerce them into treatment (Túry, Szalai, and Szumska, 2019).

Guardianship and Certification

Guardianship and certification are legal measures that are in essence formal means of coercion. Both measures rest on the assumption that the individual involved lacks capacity. Caution should be taken to ensure that courts avoid reasoning that links treatment refusal to incapacity or anorexia to incapacity (Boyle, 2019). Boyle indicates that deficits in decision-making ability relating to a functional assessment of capacity must be identified and that if courts infer a deficit in decision-making capacity, this must be put to the patient so that they have a chance to respond (Boyle, 2019).

Compulsory treatment through guardianship or certification has come into play because patients with AN are often reluctant to seek the lifesaving treatment of weight gain. Weight gain is critical to the success of treatment, however, and is a significant predictor of the reduction in eating disorder pathology (Accurso et al., 2014). Restoration of weight is also paralleled by improvements in emotional and psychological well-being, body image perception, and coping skills, and is imperative for patients' recovery (Long, Fitzgerald, and Hollin, 2012). Early response to treatment, as measured by early weight gain during the first 6 weeks of hospitalization, is especially important in terms of symptom improvement (Accurso et al., 2014; Wales et al., 2016). Younger age when beginning treatment and shorter duration of illness are also predictors of better outcome (Le Grange et al., 2014), whereas lower body mass index (BMI) on admission and older age (e.g., midlife adults) predict a poorer prognosis (Nakamura et al., 2013; Ackard et al., 2014). Thus, hospitalization and treatment, even if involuntary, should be considered *before* patients reach a severe or chronic stage of the illness. In practice, however, those for whom involuntary treatment is considered are often those very patients with the most serious forms of the illness who are least likely to benefit from further treatment.

Conversely, misplaced respect for patients' autonomy, thereby allowing patients to continue to lose weight and either refuse care or be admitted only at a critically low BMI, may lead to a fatal outcome (Holm et al., 2012; Nakamura et al., 2013). This concern is particularly relevant

because those patients more likely to default from treatment are precisely those who have the lowest BMIs, highest impulsivity (as evidenced by suicidality and substance use), and who are more psychologically ill (Huas et al., 2011).

Despite the increased risk of the illness becoming chronic (or the patient dying from their eating disorder), the family and the treatment team are often reluctant to initiate involuntary treatment. Families, though relieved that their critically ill family member is being compelled to receive care, often bear the brunt of the patient's anger and indignation at being forced into treatment. Treatment providers worry that a push for involuntary treatment will disrupt the already fragile alliance between the patient and the treatment team, making further progress all but impossible. These concerns may be well founded in that therapeutic alliance is an important predictor of achieving a target weight and remaining in treatment (Bourion-Bedes et al., 2013; Sly et al., 2013). In addition, involuntary treatment, at a time when the patient is beginning to contemplate the need for change and recovery, may also prove harmful for the patient's engagement in treatment (Douzenis and Michopoulos, 2015). These concerns must be weighed against concerns regarding future morbidity and mortality, however, if a patient with severe anorexia nervosa continues to refuse lifesaving care. In addition, patients with AN have been found to ultimately agree with the need for compulsory treatment once weight is restored (Tan et al., 2010).

Guardianship versus Certification

A guardian is an individual tasked with exercising substituted judgment on behalf of a person determined to be incapacitated (i.e., the ward) (National Guardianship Association, 2021). Guardianship and certification differ in terms of the type of treatment that may be meted out under each statute (medical care vs. psychiatric care), as well as the venue for that treatment. Treatment under guardianship laws may occur on a medical unit or to ensure medical treatment on a psychiatric unit or as an outpatient, and the guardian has latitude as to where the ward resides while receiving care. In contrast, treatment under certification can only take place on an inpatient psychiatric unit. The certification may

be transferred to an outpatient status only once the patient no longer meets criteria for inpatient psychiatric treatment.

Significantly more has been written regarding certification than guardianship for patients with eating disorders. Nonetheless, guardianship has been successfully used to treat patients with eating disorders who are severely medically ill (Griffiths et al., 1997; Born et al., 2015). Yet, the role of the medical guardian as it relates to treatment in AN, which is considered a mental illness, is limited. Guardians are capable of making decisions regarding a ward's medical care but not for the psychiatric component of the illness (Westmoreland et al., 2016). Guardianship does not grant a guardian the power to admit the ward to a mental health facility without the ward's consent. Furthermore, once the ward is no longer medically compromised, ongoing psychiatric care and nutritional rehabilitation can only be authorized by the courts under certification laws (Westmoreland et al., 2017).

Guardianship is useful in assisting with medical treatment decisions for patients with anorexia nervosa who are critically medically ill and refusing basic medical treatment such as replacement of electrolytes and intravenous fluid or nutrition (through NG or naso-jejunal tube). Once these medical interventions have been successful and the patient is no longer in immediate medical danger, however, such interventions cannot be continued under guardianship laws. Guardianship also does not cover involuntary admission to an inpatient psychiatric unit, even an eating disorder treatment unit. Therefore, guardianship is not an adequate intervention with respect to compelling psychiatric treatment for patients with severe AN once they become medically stable, no longer at imminent risk, and are able to transfer from a medical unit to a psychiatric unit. The initiation of certification to a psychiatric unit must be made in accordance with state procedure for certification and use of emergency and involuntary psychiatric medications.

To psychiatrically hospitalize individuals involuntarily, a psychiatrist or other mental health practitioner must establish that they are a danger to themselves, a danger to others, and/or gravely disabled. The family of a patient with a severe eating disorder may contact either emergency services (such as police) or a mental health provider and request that

their family member be evaluated for placement on a mental health hold. If there are grounds for the mental health hold (e.g., danger to self or others, or, in some states, the presence of grave disability), the patient is placed on a mental health hold and transferred to an emergency room.

If emergency room staff agree that continuing the mental health hold is warranted, the patient may then be admitted to a psychiatric unit for a short period of observation (usually several days, but variable from state to state). During this time-limited observation period, the treating psychiatrist may file for certification. The psychiatrist may also allow the patient to sign into treatment voluntarily or allow the patient to leave; in both instances, the mental health hold is terminated. If the treating psychiatrist files for certification, the patient may either agree to the certification (i.e., stipulate) or contest the certification in court. If the patient is certified, the certification may not exceed a defined period (e.g., in Colorado, short-term certification is three months; Colorado Revised Statute 27-65). Thereafter, further petitions must be filed to prolong involuntary treatment. While procedures and criteria vary across jurisdictions, providing involuntary medications to mentally ill patients is, in many jurisdictions, an additional step beyond certification. Moreover, involuntary tube feeding is achieved only through an additional petition. In Colorado, involuntary tube feeding is designated as a special procedure, much like electroconvulsive therapy, and requires a separate legal hearing.

Case Law and Certification

Courts in several states have applied certification statutes to treatment of patients with eating disorders. Considering some of the decisions made by those courts is useful in analyzing the results of certifying patients with eating disorders. The courts have considered several important aspects regarding certification of patients with anorexia nervosa. These include the scope of the certifying court in involving itself in treatment decisions, whether it is in the court's purview to agree that a patient is best served by receiving treatment in another state, that accurate criteria be used when presenting an argument for certification, that the defini-

tion of *grave disability* does not necessitate that the patient be close to death, and that medications may be warranted in treating patients with eating disorders.

In the case *In the Matter of Joanne Kolodrubetz* (411 NW 2d 528 (MN Ct. App. 1987)), Ms. Kolodrubetz presented to a facility in Minnesota, where she was certified because of severe anorexia nervosa. She was so ill that her life was repeatedly endangered by her behaviors when she was not in treatment. Following certification, she did not agree with the treatment modality at the facility and pursued the administrative complaint process. After that administrative process had run its course, and the findings and recommendations were unsatisfactory to her, she petitioned the certifying court for relief. Under the Minnesota statute, the certifying court only makes the initial legal determination as to whether the patient meets statutory criteria for certification. Patients challenging the specific form of treatment at a facility must follow the Minnesota administrative review process. Indeed, the appellate court noted that the certifying court may not involve itself in treatment decisions, and that the remedy for a patient who disagreed with the findings and recommendations generated by the administrative process was for the patient to file a lawsuit seeking damages. The legal standard in such a lawsuit was whether the treatment pursued by the facility was within accepted professional standards. There was no indication that the facility's treatment of Ms. Kolodrubetz fell outside that standard.

In another Minnesota case, *In the Matter of Molly Kellor* (520 NW 2d 9 (MN Ct. App. 1994)), the People appealed a District Court case authorizing Ms. Kellor to receive treatment in an out-of-state facility. As noted above, once the Minnesota certifying court makes a judicial determination, the court will not review specific treatment modalities.

Ms. Kellor was initially committed to a hospital for a period of six months, and then for a subsequent period of 12 months, to be transferred to a psychiatric facility once she was medically stable. Following her transfer to the psychiatric facility, Ms. Kellor continued to restore weight through tube feeding but gained little if any insight into her eating disorder and had a conflictual relationship with treatment staff. She sought transfer to an out-of-state facility specializing in the treatment of eating

disorders located in Tulsa, Oklahoma. The District Court granted her request, finding no in-state facility that offered eating disorder–specific treatment in Minnesota, and that under the Minnesota statute, the patient was entitled to receive appropriate treatment in the least restrictive setting. The People appealed the trial court's decision, claiming that the trial court improperly interfered with treatment decisions, and contended that the trial court had no authority to commit Ms. Kellor to an out-of-state facility. The appellate court determined that while the trial court should not interfere with or monitor treatment decisions, there was evidence in the record supporting the trial court's determination that there was no facility in Minnesota that at the time could adequately treat Ms. Kellor's eating disorder. The appellate court held that the trial court has wide discretion in determining the least restrictive setting and agreed with transferring Ms. Kellor to an out-of-state facility.

In re S.A.M. (695 NW 2d (IA Ct. App. 2005)) is an Iowa case in which S.A.M. appealed a District Court case determining that she had met the definition of a person with a serious mental impairment and, as a result, was likely to inflict physical injury on herself or others if allowed to remain at liberty. When filing the appeal, S.A.M. admitted that she suffered from a mental illness, anorexia nervosa, and therefore did not challenge the trial court's findings that she suffered from a serious mental impairment. Instead, S.A.M. challenged the trial court's finding that there was clear and convincing evidence that she was likely to inflict physical harm to herself if she were allowed to remain at liberty. Given that S.A.M. had been able to maintain a stable body weight while in outpatient treatment just prior to being rehospitalized and had never suffered from metabolic abnormalities even at a lower body weight, the reviewing court determined that there was *not* clear and convincing evidence that it was "probable or reasonably expected" that S.A.M. was likely to inflict physical injury on herself if allowed to remain at liberty. Therefore, the trial court's ruling was reversed.

In re P.A. (12 CA 1024, 13 CA 1350, not published pursuant to C.A.R. 35(f)) relates to the Colorado statutory scheme, under which the professional person associated with a facility designated for mental health treatment initiates the involuntary treatment process by filing a Notice

of Certification for Short-Term Treatment with the District Court in the appropriate venue. In Denver, the Denver Probate Court is a District Court of limited jurisdiction, handling involuntary mental health treatment requests. Hearings involving only certification may be held before a jury of six or before the court. In 12 CA 1024 and 13 CA 1350, P.A. requested court trials. In both instances, the trial court upheld short-term certification for the statutory 3-month period, determining that P.A. was a danger to herself, as well as gravely disabled. The trial court also granted involuntary medication administration authority to the designated facility regarding the respondent, as well as authority to involuntarily place a feeding tube for the purposes of NG or naso-jejunal feeding. P.A. appealed the court's decisions, alleging that because she had partially restored weight in the facility, she was no longer gravely disabled or a danger to herself as she was no longer near death. The appellate court found that the definition of *gravely disabled* does not require that the respondent be imminently near death. Instead, the definition requires only that the respondent be in danger of serious physical harm because of an inability or failure to provide oneself with the essential human needs of food and medical care.

P.A. also appealed the order of the probate court that granted involuntary administration of medications. She asserted that the standard for involuntary medication set forth had not been met by clear and convincing evidence. The court of appeals ruled that P.A.'s mental illness had so impaired her judgment that she was incapable of participating in decisions affecting her health, and medication was needed to prevent the likelihood of her causing serious harm to herself, albeit indirectly. If she did not take medication, she would decompensate psychiatrically and (as a result) put herself in life-threatening danger by not taking in adequate nutrition. There were no viable alternatives to adequately treat her mental illness, and her psychiatrist testified that she would not be able to successfully treat P.A.'s anorexia without medication.

When P.A. appealed her certification again in 2013, this was the second appeal in a period of just over one year involving a short-term commitment based on substantially similar circumstances. The court concluded that, based on these substantially similar circumstances, that is,

her lack of sustained recovery, the issues raised in the case were moot. Under the *mootness doctrine*, an issue is moot when any judgment concerning the issue cannot have a practical effect on an existing controversy, and appellate courts will not render opinions on the merits of an appeal when the issues presented are moot because of subsequent events. Thus, the court declined to reconsider the issues presented by P.A.

In Pennsylvania, *In the interest of D.C.W.* (No. 1218 MDA 2015 (PA Sup. Ct. 2016)) involves a petition filed in 2015 by the Office of Mental Health and Intellectual Disabilities of Centre County, Pennsylvania, to extend the involuntary treatment of the appellant, D.C.W., pursuant to section 7304 of the state's Mental Health Procedures Act. At the hearing, the treating psychiatrist testified that D.C.W. had anorexia nervosa, binge/purge subtype, that he had purged to the point of severe electrolyte abnormalities and dehydration, and that his condition could lead to death, disability, or serious physical debilitation within 30 days. The appellant testified that he had been doing very well, denied that he was currently purging, and insisted that he wanted to return home. After that hearing, the mental health review officer granted the petition to extend the involuntary treatment for 90 days. D.C.W. then filed a petition for review of certification to the Centre County Court of Common Pleas. He argued that the state had failed to establish by clear and convincing evidence that he needed involuntary treatment when he was voluntarily working on techniques designed to abate his self-injurious behavior. The court disagreed, noting that for a person to be committed for an extended period of treatment, it is not necessary to show that the patient committed an overt act within 30 days of the hearing, but it was necessary for the court to find that within the patient's most recent period of institutionalization, the patient's contact demonstrated a need for continuing involuntary treatment—that is, the condition continued to show that he was a clear and present danger to himself or others. In considering the clear and convincing evidence standard, the court opined that these standards had been satisfied by the testimony of the psychiatrist, and that "it was clear to this court that appellant was still in need of involuntary mental health treatment for his anorexia nervosa and without that treatment he had a reasonable probability of death, disability or se-

rious debilitation within 30 days." The superior court thus affirmed that the trial court had properly certified the appellant's continued involuntary treatment based on the clear and present danger he presented to himself.

Certifying Patients with Eating Disorders

A meta-analysis conducted by Latzer and Zohar-Beja (2014) compares perspectives on involuntary treatment of anorexia nervosa, indicating that there may be negative effects of forcing an individual with AN to undergo treatment, but that there is evidence that not intervening leads to heightened morbidity and mortality. The frequency with which patients with AN are certified ranges from 8% to as high as 44% (Clausen and Jones, 2014; Elzakkers et al., 2014). The differences in the percentage of patients treated involuntarily may be explained by differences in patient populations as well as by differences in local regulations or procedures as well as across hospitals.

For several reasons, certification involving a patient with anorexia nervosa differs from certifying a patient with any other major psychiatric illness, such as bipolar disorder or schizophrenia. Patients with AN have been described as presenting with a "pocket of irrationality operating within a sea of personal rationality regarding all other matters" (Carney, 2014). A review on the topic of assessing subtle forms of incompetence is pertinent in this regard (Gutheil and Bursztajn, 1986). First, with anorexia nervosa, the patient's competence is usually suspect only in the narrow area of self-nutrition/body image, and patients with AN do not appear disorganized or illogical in general conversation. Second, they are usually intelligent and articulate, having a good grasp of the legal issues at hand, even when their cognition is compromised by malnutrition. Patients with anorexia nervosa are usually well groomed and dress in a manner designed to conceal their extreme emaciation. Third, because the illness has been of several months or years duration by the time they come to medical or psychiatric attention, the medical danger typically associated with anorexia nervosa is often perceived not to be imminent. Fourth, patients with eating disorders usually provide the court a rational

explanation for their behavior, and they do not usually express an intent to die. On the contrary, they usually express an intent to continue living but with a continued reduction in weight (Appelbaum and Rumpf, 1998). Finally, judges or magistrates may find it difficult to distinguish an eating disorder from culturally normative weight concerns, given media and social emphasis on thinness. Certification in patients with AN is most likely to be triggered by a patient having a longer duration of illness, a greater number of prior admissions, more comorbidities, a higher incidence of self-harm, and a greater severity of medical illness (e.g., having a very low BMI and/or high risk of refeeding syndrome) (Ayton, Keen, and Lask, 2009; Carney et al., 2008; Douzenis and Michopoulos, 2015; Clausen and Jones, 2014). Duration of illness may also be a significant predictor of involuntary treatment. The characteristics noted in the group of patients who are treated involuntarily speaks not only to the severity of their illness, but also to the complexity of their situation.

There are no definitive criteria regarding when to certify versus not certify a patient with a severe eating disorder. Criteria that have been suggested as being in favor of certification include medical comorbidities such as seizures, syncopal episodes, hypoglycemia, organic brain syndromes, cardiac arrhythmias, severe bradycardia, renal compromise, volume depletion, tetany, and rapidly diminished exercise tolerance (Holm et al., 2012). Psychiatric comorbidities such as suicidal ideation or self-harm behavior are also likely to bring about involuntary treatment.

Outcome of Certification

Patients who are treated involuntarily take longer time to restore weight, are more often hospitalized on a locked psychiatric unit, more likely to be tube fed, and may also be more prone to refeeding syndrome (Clausen and Jones, 2014). Despite beginning their course of treatment with more severe symptoms and comorbidities and a having a longer and potentially more complicated inpatient stay, patients who undergo involuntary treatment have similar BMIs at discharge compared those who are treated voluntarily (Elzakkers et al., 2014; Clausen and Jones, 2014). Yet, mortality in the five years following a compulsory admission is

higher than that of patients treated voluntarily (Ward et al., 2015). Despite this statistic, approximately 20 years following admission, this difference was attenuated and, although the standardized mortality in the compulsory treatment group remained higher, the difference between the two groups was reduced over time. Thus there was no significant difference between certified patients and those who were treated voluntarily when this was measured two decades later. This reflects prior findings that the risk of premature death among patients with anorexia peaks within the first 10 years of follow-up (Franko et al., 2013). It has also been suggested that poor outcome in some certified patients is probably not the result of certification in and of itself, but rather because these patients have other validated poor prognostic factors, such as comorbid psychiatric illnesses and older age at admission (Ramsey et al., 1999; Ayton, Keen, and Lask, 2009). Nonetheless, a 2015 article from Israel comparing voluntary versus compulsory admissions of individuals with anorexia nervosa found compulsory treatment to be as beneficial as voluntary treatment and indicated that compulsory hospitalization actually reduced some patients' feelings of guilt about accepting treatment (Latzer et al., 2015). Carney, Yager, et al. (2019) noted that studies to date suggest that, with extra time and effort, the short-term weight restoration of patients hospitalized involuntarily for AN might be comparable with that seen in voluntarily admitted patients, that a reasonable number of people might actually come around to acknowledge their need for treatment, and that some might even be thankful (or at least tell their caregivers that they were thankful) for the fact that they were treated, even against their will (Carney, Yager, et al., 2019). Yet, they also wrote that literature "contains a paucity of detailed descriptions differentiating those involuntarily treated people who benefited in both the short term and the long term from those who rapidly relapsed and even succumbed," noting gaps in knowledge suggesting that large multisite studies on treatment-refusing people with AN are needed if we are ever to be able to make clinical decisions based on concrete evidence rather than anecdotes (Carney, Yager, et al., 2019).

Túry, Szalai, and Szumska (2019) note that "as human life is a basic value and the health care system is obliged to save lives, it must be

recognized that resistance and the denial of illness are characteristic features of AN." While involuntary treatment may be lifesaving, several aspects of it unfortunately cannot be continued indefinitely without a minimal level of cooperation from the patient. Involuntary tube feeding is one such example. While beneficial in the short term, involuntary tube feeding cannot continue indefinitely. In addition, patients who require involuntary tube feeding for a prolonged amount of time are generally those who are most recalcitrant in their attitudes toward and motivation for recovery. Other situations in which involuntary treatment may not be helpful include repeated elopements from treatment centers, an increase in the use of other methods of self-harm, and the development of suicidality. It may also prove futile to treat patients who have been ill for a decade or more, have required multiple prior treatments, or have never had a sustained period of wellness despite extensive interventions. That being said, in a study of individuals with anorexia nervosa and bulimia nervosa that spanned 25 years, Eddy et al. (2017) noted that, at 22 years of follow-up, 62.8% of participants with AN and 68.2% of participants with BN recovered, compared with 31.4% of participants with AN and 68.2% of participants with BN by 9-year follow-up. Approximately half of those with anorexia nervosa who had not recovered by 9 years progressed to recovery at 22 years; thus, while recovery from bulimia nervosa happened earlier, recovery from anorexia nervosa continued over the long term, arguing against the implementation of palliative care for most individuals with eating disorders (Eddy et al., 2017).

Treatment of Severe and Enduring Eating Disorders

According to Yager (2020), a state of being "cured" from anorexia nervosa means full remission of physical and psychological symptoms and consequent recovery, which is estimated to occur for 30% to 70% of patients with anorexia nervosa (Eddy et al., 2017; Smink, van Hoeken, and Hoek, 2013). Yager (2020) also notes that after a decade or so, the odds of "cure" diminish, and 20% of patients with AN develop a severe and enduring form of the illness (SE-AN), manifested by decades-long illness. While the study by Eddy et al. (2017) provides room for optimism,

SE-AN and its more broadly encompassing relative, SEED (severe and enduring eating disorder), have historically been described as unlikely to remit (Robinson, 2009, 2014; Treasure et al., 2015; Touyz et al., 2016; Broomfield et al., 2017).

Individuals with SEEDs are a subgroup of patients whose illness is long-standing and life threatening. They have a very low likelihood of recovery, poor adaptive function, high levels of symptoms and comorbid physical and psychological ills, and require the regular attention of a multidisciplinary team (Robinson, 2009, 2014; Hay and Touyz, 2015). Patients with SEEDs present with signs and symptoms of an extreme eating disorder, such as BMI below 13 and physical sequelae of a long-standing eating disorder (e.g., osteoporosis, renal failure, bowel disease due to laxative abuse). The symptoms of their eating disorders profoundly interfere with their quality of life. Patients with SEEDs may be as impaired by symptoms of their eating disorder as individuals who suffer from other serious chronic mental illnesses such as schizophrenia. The burden on caregivers is noted to be substantially higher than that of caring for a relative with schizophrenia or bipolar disorder (Martin et al., 2015). Patients with SEEDs often suffer from depression as well as social isolation and stigmatization.

In an article entitled "Managing Patients with Severe and Enduring Anorexia Nervosa: When Is Enough, Enough?," Dr. Joel Yager (2020) noted that clinicians "must be realistic about the low odds of effecting cure and adjust treatment expectations accordingly." Thus, "managing these patients may require trying a novel treatment modality or shifting the goal of treatment from cure to harm reduction, or ultimately to palliative or hospice care. We should also strive to provide humane comfort and support, as closely aligned to patients' judicious wishes as possible" (Yager, 2020). Yager recommends striving for the to the so-called Goldilocks principle, in which balance is key. According to Goldilocks rules, clinicians must establish therapeutic goals that neither grossly overestimate what can realistically be achieved nor underestimate the patient's potential (Yager, 2020). Expectations that are unrealistically high for a patient may generate internal performance pressures and contribute to cycles of attempts at treatment and failures that produce psychological

harm, including avoidance of future treatment. Alternatively, setting expectations too low could lead clinicians to put insufficient emotional energy into treatment and inadequately challenge patients.

Novel Treatments

Evidence supporting the efficacy of pharmacotherapy in treating anorexia nervosa has been limited, and currently no medications are FDA approved for the treatment of anorexia nervosa (Brar, 2020, cited in Westmoreland, 2020). Fluoxetine has been shown to be useful in patients with bulimia (Yager, 2006), and lisdexamfetamine has been used to successfully treat binge eating disorders (Citrome, 2015). While there have been mixed results regarding the use of second-generation antipsychotics, according to studies reviewed by Brar, Wassenaar, and O'Melia (2020), olanzapine may have the best evidence overall. A small study of patients with severe, treatment-resistant AN whose body image distortion reached delusional proportions indicated that low doses of haloperidol reduced body image disturbance and the drive for thinness (Mauri et al., 2013). The QTc-prolonging effect of antipsychotics, however, should not be underestimated in individuals with a low BMI, especially those with the purging form of AN, which results in electrolyte imbalances that themselves prolong QTc interval (Westmoreland, Krantz, and Mehler, 2016). In addition to olanzapine, other medications that have been studied for individuals with SEEDs include dronabinol and duloxetine (Kotilahti et al., 2020). It has also been suggested that ketamine can produce symptom improvement during continuous use, including decreased suicidality, in patients with comorbid AN and major depressive disorder (Kotilahti et al., 2020; Wink, 2014, cited in Yager et al., 2006), although the treatment may be more effective after weight restoration (Dechant, Boyle, and Ross, 2020).

Neurostimulation—interventions intended to alter nervous system function using energy fields in the form of electricity, magnetism, or both (Dalton, 2018, cited in Brar, Wassenaar, and O'Melia, 2020)—have been considered for patients with eating disorders. While beyond the scope of this chapter, deep brain stimulation (DBS), transcranial direct

current stimulation (tDCS), transcranial magnetic stimulation (r-TMS), and electroconvulsive therapy (ECT) have all been used to treat patients with eating disorders, especially those with a severe and enduring form of the illness (Zhang et al., 2013; Oudijn et al., 2013; Sauvaget et al., 2015; McClelland et al., 2013; Coman et al., 2014; Ferguson, 1993; Andrews et al., 2014; Kotilahti 2020). These treatments are still in the early stages of use for patients with eating disorders, and there is no consensus that they will lead to lead to cure or remission. In addition, ethical concerns arise regarding whether an individual with a severe ED has the ability to consent to such treatment, especially when the treatment in question is relatively untested and invasive.

Harm Reduction

In some instances, avoiding harm caused by illness is neither possible nor realistic (Bermudez, Hay, and Touyz, 2021). The focus then shifts to mitigating the degree of harm and its impact on quality of life. Implicit in the goal of this *harm reduction* strategy is not to eradicate the disease or prevent all harm but to reduce the damage it causes and improve the life experiences and quality-of-life opportunities for individuals with the underlying illness. The decision to embark on harm reduction does not need to be irreversible; the door remains open either toward further harm reduction or toward curing or eradicating the illness as treatments advance or patients decide to accept existing or new treatments (Bermudez, Hay, and Touyz, 2021). Harm reduction is treatment directed toward enabling an individual to continue living, and being in less danger of immediate death, while conceding that the treatment is less than a full course of care and is directed only toward maintaining a minimal (albeit acceptable) level of care for the individual involved.

Harm reduction involves reducing the weight that individuals are expected to gain or maintain, as well as reducing purging symptoms. It is also a pragmatic, personalized approach targeting a reduction in negative consequences of risk behaviors (Bermudez, Hay, and Touyz, 2021), as well as a strategy to increase emphasis on quality of life and function, even though the latter may be somewhat limited (Hay and Touyz, 2015).

Russell et al. (2019) considered harm reduction an approach aiming for good enough rather than perfect, where weight, nutrition, safety, and symptomatic relief are the focus of maximizing function and quality of life. Harm reduction is also a step to be considered before palliative or hospice care or a conclusion of futility for a given patient who has received adequate and sufficient conventional treatment efforts (Westmoreland and Mehler, 2016).

The goal of the harm reduction model is to help patients attain (and then maintain) a reasonable level of functioning at a lower than optimal weight, rather than subjecting them to a full course of treatment (e.g., a prolonged hospital stay to achieve and maintain ideal body weight). Finding meaning and purpose in life are emphasized, and symptom remission (beyond that required to keep these patients from needing hospitalization) is deemphasized. Candidates for harm reduction are those patients who have endured multiple prior eating disorder treatments with minimal success, and those for whom full weight restoration has not been sustainable or their quality of life close to goal weight has been unacceptable to them. Patients who undergo a harm reduction treatment model are managed as outpatients once they have attained an agreed-on (though less than ideal) body weight at an inpatient or residential level of care, and once they are out of immediate medical danger. They are then allowed to remain at a weight that is sufficient to enable them to have a reasonable quality of life, even if they cannot work or be fully independent.

In this model, patients with a BMI under 13 are at highest risk of demise, whereas those with a BMI higher than 15 are at the lower end of the risk spectrum. Harm reduction entails monitoring and maintaining normal body temperature, hydration, and electrolyte balance; preventing self-induced or iatrogenic refeeding syndrome; and supporting heart, kidney, and brain health. No matter what the patient's BMI, however, their individual level of risk may be increased if they experience rapid weight loss or other medical problems (such as electrolyte abnormalities secondary to purging). Patients undergoing a harm reduction model of care are also at great risk of further morbidity and early mortality if they develop medical problems such as a viral illness, pneumo-

nia, or a fracture (Robinson, 2009; Robinson, 2014). These comorbidities may lead to rapid decompensation and death. Russell et al. (2019) noted that harm reduction also includes optimizing dental health through regular checkups, cleaning, and restoration, as well as use of neutralizing mouthwashes after purging.

Patients who receive treatment according to the harm reduction model must be closely monitored. They must have regular visits with their outpatient team, consisting of a psychiatrist or primary care physician, dietician, and therapist. If their weight drops below the agreed-on weight, or laboratory test results become significantly abnormal (e.g., rising transaminase levels; hypoglycemia; drop in white blood cell count; abnormalities in potassium, bicarbonate, magnesium, or sodium; changes in EKG), they must be hospitalized to restore the amount of weight they have lost and/or to normalize laboratory test results.

Patients undergoing harm reduction should also be monitored for depression and anxiety, and these concerns, along with drug and alcohol use, may require referral to specialty services (Russell et al., 2019). A history of physical or sexual trauma, compounded by the negative experiences of failed treatments, may also contribute to psychological comorbidities. Individuals with chronic eating disorders are often demoralized and develop hopelessness and suicidal ideation. It is well known that suicide attempts, with a high risk of being fatal, are made by people who are habituated to pain because of their experience with physical pain (Joiner, 2007). Patients with severe and chronic eating disorders often attempt suicide using highly lethal means because they are well accustomed to pain, given their experience of starvation, bingeing, purging, the medical complications of their eating disorder, and habituation to other forms of self-injurious behavior (e.g., cutting and substance abuse).

A harm reduction approach must take several ethical issues into account to ensure clinicians understand the needs of a patient at a given time and make clinical decisions that are clearly in that patient's best interest. Principles such as "first, do no harm" and respecting patients' right to self-determination (taking capacity for decision making into account) should be used as guides. The question of severely ill patients in

a younger age range being offered alternative approaches before their illness becomes chronic has recently been raised in the literature and poses another layer of complexity in ethical considerations (Kaplan and Strober, 2019).

It is also important to recognize situations in which harm reduction is not working. Bermudez, Hay, and Touyz (2021) noted that a lapse in a stipulated goal (e.g., weight loss below the agreed-on threshold, or more frequent use of laxatives than planned) does not necessarily mean the effort is futile. If such lapses become recurrent, however, and the weight or behavior goal appears not to be sustainable, then the validity of the harm reduction agreement would have to be reexamined. Continuing a harm reduction approach for a prolonged period without harm reduction goals is not in the patient's best interests.

Palliation

Palliative care is an approach that improves the quality of life of patients and their families facing the problems of life-threatening illness through the prevention and relief of suffering by means of early identification, assessment, and treatment of pain and other physical, psychosocial, and spiritual problems (WHO definition of palliative care cited in Lindblad, Helgesson, and Sjöstrand, 2019). Palliation means preventing or relieving suffering in addition to supporting the best possible quality of life for patients. Problems with the WHO definition of palliative care as it applies to psychiatry in general is that the patient must have a life-threatening illness (Lindblad, Helgesson, and Sjöstrand, 2019). Most psychiatric disorders, however, while conveying excess morbidity and early mortality, do not usually fit into this definition. Severe anorexia nervosa is an exception to this in that restriction and excessive purging behavior may well lead to death (Arcelus et al., 2011).

The concept of palliative psychiatry has evolved as a result of recognizing that patients with a severe and persistent mental illness require a specialized approach to treatment that improves quality of life (and may also prolong life) by eschewing treatments that have not been successful (Trachsel et al., 2016). The concept of Oyster Care, which origi-

nated in Belgium, is an example of a holistic approach. Oyster Care is based on four pillars whereby the quality of patients' lives are improved: physical care (responding to patients' somatic impairments), psychological care (focusing on mental comfort and well-being), social care (providing a structure of daily activities and social contact), and existential care (which enhances the experience of life as meaningful and valuable) (Decorte et al., 2020).

Patients with SEEDs who undergo palliative care are those for whom any further treatment (be it active treatment, with the goal of normalizing weight, or harm reduction) is unlikely to resolve or decrease their illness and suffering, and their goal is to be comfortable during the remainder of their life, albeit likely an abbreviated life. Continuing to treat these patients is more harmful than beneficial, and they are unable to accede to the terms necessary for a harm reduction model to work in their favor. Patients who elect to undergo palliative care often have untreatable comorbidities, such as renal failure, cardiac failure, severe osteoporosis, or severe major depressive disorder that does not respond to treatment (Westmoreland, Erickson, and Bermudez, 2020). Palliative care has been described as a "low-intensity treatment" for individuals with SEEDs for whom a cure is unlikely (Yager, 2020). Candidates for palliative care have usually had multiple prior treatments and no remission in symptoms or ability to sustain any weight gain. Symptomatic relief includes analgesics for pain associated with osteoporosis and stress fractures; wound care for decubitus ulcers; and medications to reduce anxiety symptoms, depression, and perseverative, obsessive thinking, as well as to improve sleep. By the time individuals with SEEDs consider the possibility of palliative care, they are likely to have not only irremediable medical comorbidities but also a fragile cognitive state. Delirium, entailing a fluctuating level of alertness, may be caused by medical complications of an ED such as renal failure, heart failure, metabolic abnormalities, or certain medications (Westmoreland, Erickson, and Bermudez, 2020). Palliative care may take place in the patient's home, a hospital, a skilled nursing unit, or, ultimately but not necessarily, in a hospice care unit.

The Academy for Eating Disorders has noted that, despite the potentially fatal consequences of an ED, full recovery is possible. Nonetheless,

it also recognizes the role of palliative care (AED Medical Care Standards Task Force, 2016).

Although the term *palliative care* (symptomatic relief from pain or physical or mental stress) is not synonymous with hospice care, these terms are frequently conflated.

Futility

A futile act is one that is considered pointless or useless. Little has been written regarding futility and psychiatric illness, and those who have considered the topic have asked if there is really a psychiatric illness for which every treatment has been tried and no hope remains. Given that anorexia nervosa has a poor prognosis in only a subset of patients, and that these patients' psychiatric condition leads directly to their demise (i.e., not eating, because of AN, leads to malnutrition and subsequent death), the question arises as to when to continue pursuing active treatment versus either engaging in harm reduction or conceding that continuing care (except that form of care directed toward a less painful death) is futile.

Case Study 3

E.M. was 56 years old when she was admitted to an inpatient eating disorder treatment unit. She had suffered from anorexia nervosa, restricting subtype, since her teens and had undergone multiple prior treatments. She had managed to function relatively well for eight years but had relapsed several times over the preceding five years. She became progressively more ill during each admission. E.M.'s eating disorder was complicated by major depressive disorder, recurrent, with poor response to medications. She underwent ECT and developed delirium, leading to its discontinuation. E.M. had two adult children with eating disorders, with whom she had a contentious relationship. She wanted to leave treatment. An ethics consultation was requested to assess decisional capacity. The consultant opined that E.M. lacked capacity but raised questions regarding her ability to recover, given the chronicity of her illness. A mental health hold was placed but dropped when E.M. contracted for weight resto-

ration to a certain point (i.e., harm reduction). But she was unable to overcome her entrenched behavior of caloric restriction and struggled to gain weight, especially after her transition to a partial hospital program (PHP). Insurance labeled E.M. as "chronic" and unlikely to benefit from continued care. Her insurance company refused to fund further PHP care as she was "too sick" for PHP, but then refused to fund inpatient care as she was "too chronic." Insurance terminated, and E.M. left treatment. She relapsed immediately following discharge and became severely depressed. She came close to committing suicide on two occasions and was readmitted to an inpatient eating disorder treatment unit, but she was so ill she required treatment on a medical unit. A capacity evaluation was performed, and E.M. was deemed to have decisional capacity. In addition, her brother had durable power of attorney and agreed with her decision to return home to hospice care. The remainder of her family struggled with this decision but eventually came to terms with it. E.M. died several weeks after arriving home.

E.M.'s lifelong battle with her eating disorder as well as her depression, which was not responsive to treatment, led to her feeling that she did not have a reasonable quality of life. While she had previously had a period of remission from eating disorder symptoms, during the latter part of her life, she was unable to return to that state. The treatment team assessed that a reasonable effort had been made to assist this patient in returning to health but noted more and more distress with each admission as she struggled to cope with immense depression and severe body image concerns; they opined that further active treatment would do more harm than good. At the time she decided to begin hospice care, she was deemed to have capacity to make this decision, and both treatment team members (and, in time, her family) were able to see that allowing her to die on her terms was ultimately better than continuing to try to force treatment or have her complete suicide in a violent manner.

Patients who are subjected to involuntary treatment are often those who are the most severely and chronically ill and who therefore have the

highest risk of death. Despite the truism of this statement, this does not necessarily mean these patients *should* be certified merely because the symptoms and signs of their clinical condition meet the burden of proof for certification, which is clear and convincing evidence in most jurisdictions. Patients who have been ill for a long period may be least likely to benefit from certification. Even though they may be more likely to be deemed incompetent regarding decisional capacity, they have a wealth of experience with their illness and may be the most capable of judging the quality of their lives and whether that quality justifies continued attempts at active treatment, harm reduction, palliative care, or hospice care.

The concept of futility as it pertains to anorexia nervosa has sparked a contentious debate. There are several arguments against futility in anorexia nervosa. First, what is meant by the term *end stage* as it applies to an eating disorder lacks clarity, even if the eating disorder becomes severe or chronic. Unlike terminal cancer, in which there may be little or no hope of cure, most medical complications of anorexia nervosa are treatable, even in their most severe form (Westmoreland and Mehler, 2016). What makes AN untreatable is not the lack of available cure, but rather the patient's inability or refusal to accept that their critically low weight is life threatening (Geppert, 2015). Patients with severe AN often say they want to live but then eschew the very treatment—eating and weight restoration—that is required to save their lives. A severely ill patient who states that they wish to live but then rejects lifesaving care raises the question as to whether this patient has the decisional capacity required to refuse treatment. That being said, John Zen Jackson noted that one should not conclude that all people with anorexia are necessarily incompetent to refuse treatment and that it is important to be open to the possibility that sufferers are as competent as anyone else to make decisions about the quality of their lives, and to assess the relative value of their lives apropos the suffering the endure (Jackson, 2017).

Decisional capacity is patients' ability to understand information regarding their condition, reason through the information needed to make a decision regarding their care, appreciate the consequences of the de-

cision they make, and communicate their choice. It is questionable whether patients with severe eating disorders have the capacity to decide that further treatment is futile when the core symptom of their illness is a cognitive distortion about nutrition and weight gain, and when they do not believe that starving themselves is life threatening. Poorer decision-making capacity in patients with anorexia nervosa often rests on intolerance of uncertainty and myopia regarding the future (Adoue et al., 2015). Seeing as cognitive distortions often normalize with weight gain, it has been asked whether treatment providers are therefore not obligated to treat patients so that their cognitive distortions resolve, and their decision making is no longer impaired (Geppert, 2015).

In two cases in which patients with SEEDs were allowed to refuse treatment, leading to their demise, both patients appear to have embarked on the path toward death despite having questionable decision-making capacity. In one of the first published cases discussing end-of-life care for a patient with anorexia nervosa, O'Neill, Crowther, and Sampson (1994) reported on a 24-year-old who had suffered from anorexia nervosa for at least 7 years and had been hospitalized on 11 occasions over 6 years before admission to a hospice unit. Prior treatment had included nasogastric feeding and psychotropic medications. At the time of her hospice admission, she was severely underweight and suffering from pressure sores, urinary incontinence, and multiple fractures. She continued to exercise despite her treating physician's concern that doing so in the setting of lumbar spine fractures could lead to paraplegia. A week after admission to the hospice unit, she developed delirium and died. While the decision to admit this patient to hospice care was apparently a joint decision shared by the psychiatric consultant, hospice medical director, the patient's family, and the patient herself, her decision-making capacity does not appear to have been evaluated prior to the decision (O'Neill, Crowther, and Sampson, 1994).

Another case dealt with a 30-year-old patient with a history of chronic anorexia nervosa. According to her treatment providers, she had reached a point in her treatment where forcing her into involuntary treatment or waiting for her to voluntarily engage in treatment were unlikely to cure her eating disorder or even afford her a decent quality of life

(Lopez, Yager, and Feinstein, 2010). Active treatment was terminated, and hospice care was instituted. She was reluctant to discuss end-of-life issues, however, and insisted that she would not die, nor did she want to die. Her health continued to decline, and she died three weeks after admission to a hospice care unit. Despite questions about her capacity, her treatment providers noted that even if she been declared legally incompetent regarding her ability to make treatment decisions, "Then what?," thus indicating that capacity may not be the final arbiter in deciding futility. Rather, they noted, patients with a poor prognosis who continue to decline physiologically and psychologically and appear to face a terminal course should be given the more humane choice of hospice care versus being forced in a treatment stay with an undoubtedly questionable prognosis (Lopez, Yager, and Feinstein, 2010).

In a more recent review on the topic, patients who chose to succumb to their illness were noted to be competent in their decision making when they elected to pursue end-of-life care (Campbell and Aulisio, 2012). These patients were older than those described by O'Neill, Crowther, and Sampson (1994) and Lopez, Yager, and Feinstein (2010); had longer periods of failed treatments; and consistently refused life-sustaining care over a longer period.

Case Law and Futility

Three cases from the United Kingdom argue opposing sides of the futility debate. In *Local Authority vs. E* (EWHC 1639 (2012)), a patient with a severe and chronic eating disorder had previously executed two advance directives refusing compulsory feeding. Her parents and physicians argued that she was comparatively well when she executed the directives and asked that she be allowed to die with dignity. Nonetheless, the judge ruled that Ms. E suffered from the same cognitive distortions at the time of the court case as she did at the time she executed the directives. The judge noted that the value of life trumps the presumption that further treatment will fail and ordered that involuntary treatment be tried in this case. Critics of this decision have suggested that supporting the will of the patient to the best possible extent, with the additional support of

the family, might have been a better course of action than the formulation of "best interests" (with the objective of saving the life of a patient who did not want to live, and whose family supported her decision to die) (Ryan and Callaghan, 2014).

In contrast, in *NHS Foundation Trust vs. Ms. X* (EWCOP 35 (2014)), the court declined to subject Ms. X, who had suffered from anorexia nervosa for 14 years, to forced feeding, even though she had been deemed to lack capacity regarding her eating disorder. The judge characterized forced feeding against her wishes as amounting to inhumane treatment interfering with her autonomy. The judge also attempted to distinguish between feeding and weight gain and psychosocial treatment, which focuses on quality of life (Mackenzie, 2015). This approach, however, fails to take into consideration that weight gain during inpatient treatment is a significant predictor of clinical outcome following discharge, even though it is unclear whether weight gain exerts a causal effect or is, instead, a marker for readiness to tolerate weight restoration and to engage in life beyond the eating disorder (Lund et al., 2009; Steinhausen et al., 2009). In addition, it has been argued that treatments seeking only to improve quality of life may be unlikely to produce lasting change unless accompanied by a reduction in eating disorder symptoms and an improvement in weight (Bamford et al., 2015).

In another case from the United Kingdom, *Betsi Cadwaladr University Local Health Board v. Miss W* (EWCOP 13 (2016)), Miss W, age 28, had been diagnosed with a SEED for 20 years and had spent a cumulative total of 10 years in inpatient care. As a result of her losing weight, the Health Board raised two contrasting proposals: (1) to refeed her under sedation, which would involve her being rendered unconscious for 6 months and fed via tube; or (2) to discharge her to her parents' home with a community support program. This was predicated on the recognition that her condition was not treatable and that acute treatment was no longer recommended (Geppert, Yager, and Kerwin, 2021). Judge Jackson, the jurist who had heard E's case (*Local Authority vs. E and Others*, EWHC 1639 (2012)), presided over Miss W's case. He opined, "After all that has happened, it now has to be accepted that it is beyond the power of doctors or family members, and certainly beyond the power of the court, to bring

about an improvement in W's circumstances or an extension of her life." He determined her discharge to the community was the "least worst option" (*Betsi Cadwaladr University Local Health Board v. Miss W*, EWCOP 13 (2016)). Thus, the same judge made opposing rulings regarding patients with AN. In contrast to the case of E, Judge Jackson believed that further treatment for Miss W would cause harm without any real chance of benefit (Geppert, Yager, and Kerwin, 2021). A similar scenario played out in two cases from the United States.

On February 20, 2017, a young woman known as A.G. died in the palliative care unit of a New Jersey hospital 3 months after a court decision that allowed her to forgo further forced treatments for her SEED (Geppert, Yager, and Kerwin, 2021). A.G. had undergone numerous treatments in various settings over the preceding 20 years, either eloping from or refusing treatment, and had no sustained periods of remission. In 2014, she was involuntarily committed to a state psychiatric hospital. During her 2-year stay in that facility, she completed a Physician Orders for Life-Sustaining Treatment (POLST) form with her clinician and stated her goals as "I want to live freely and not being bothered by anyone." The POLST included do not intubate, do not resuscitate, and do not administer artificial nutrition orders as well as a request for no further hospitalizations.

A.G.'s condition continued to worsen until her condition compelled the facility to obtain a temporary guardian and transfer A.G. to an acute care hospital in June 2016. A.G. was appointed a guardian. She was initially given parenteral nutrition, which was discontinued after she suffered congestive heart failure. Her guardian requested that she be transitioned to palliative care. The state opposed this motion. The ethics opinion recorded in A.G.'s medical record held that the most compassionate, humane, and medically appropriate care plan was allowing her to transition to palliative care with continued psychiatric treatments, medical management of her pain and other physical symptoms, and maximum psychological, emotional, and spiritual support for A.G. and her family (Geppert, 2020, cited in Westmoreland, 2020). On November 21, 2016, Judge Paul Armstrong ruled that the provision of palliative care to A.G. as incident to her best interests. A.G. was admitted to a

palliative care unit and died several months later (*In the Matter of A.G.* (2016)).

Several months later, Judge Armstrong presided over the S.A. case. S.A. had had severe AN since age 13, and in June 2017, when she was 20 years old, after she collapsed in her family home, she was admitted to an acute care hospital weighing just 60 pounds. She was stabilized and transferred to an ED center in Princeton, New Jersey, where she underwent treatment, including artificial tube feeding. Her psychiatrist and medical doctors believed that she was delusional, in denial, and did not understand her risk of dying and therefore did not have the capacity to make her own medical decisions. S.A.'s parents wished to override their daughter's refusal of treatment and provide compulsory feedings in an effort to save her life. S.A. articulated her wishes to return home and manage her own ED and described being in treatment as "torture." She said she would choose death over treatment, according to the judge.

Judge Armstrong, who also presided over the case of A.G. (*In the Matter of A.G.* (2016)), pronounced that S.A.'s parents were acting in her best interest to save her life and that S.A. had neither reached the same stage of organ failure and grave functional debility as A.G. had nor endured as many years of failed inpatient treatment (*In the Matter of S.A.* (2017)). He granted the parents guardianship over her medical decisions in the hopes that S.A. could have some beneficial outcome from further treatment. This case is arguably different from that of A.G. S.A. had not reached a stage of multiorgan failure in which recovery would be highly improbable and did not have the same chronic serious disabilities (*In the Matter of S.A.* (2017)).

Framework for End-of-Life Decisions

Eating disorder professionals are trying to strike a balance between opposing futility outright and succumbing to a slippery slope argument that would make all patients with SEED inherently eligible for end-of-life care. The need for compassion, perhaps above capacity, has been noted, so that patients are not forced into an intolerable living situation merely because they are deemed to lack capacity. On the other hand, when the

illness itself compromises a patient's ability to make a fully competent decision, overriding a patient's autonomy may be justified under the doctrine of paternalism in an effort to save that patient's life and return them to a state capable of making an informed and competent decision.

A framework that balances patients' wishes with legitimate concerns about allowing someone with impaired decision-making capacity to refuse further treatment has been proposed. In a recent editorial on this topic, McKinney suggested that treatment refusal decisions regarding further episodes of care should be made (1) at a time when the patient is competent (i.e., between episodes); (2) with the patient having a realistic expectation regarding outcome (i.e., the patient must know that refusing nutrition will lead to death); (3) based on a realistic assessment of current quality of life and the low probability that current or future treatment will succeed; and (4) when patients are consistent in communicating their desires (McKinney, 2015). Each case must be considered in a nuanced and thoughtful manner. Eating disorder professionals are justifiably reluctant to undertake any treatment that bears little hope of advancing a patient's quality of life, directly opposes the wishes of that patient and family, and simply extends a life of suffering, even if the patient has diminished capacity. Surely, as has been suggested, even if that patient does not have decision-making capacity, they are still likely capable of appraising their suffering (Kendall, 2014; Yager, 2015). Futility should therefore be considered on a case-by-case basis and applied when considering a particular treatment intervention, at a particular time, for a particular patient.

Conclusions

Ethical principles assist us in determining the best course of action for patients with anorexia nervosa. Certification and guardianship may be helpful in treating patients with AN, especially patients who are in the earlier stages of the illness. Given the poor prognosis for patients with chronic and life-threatening anorexia nervosa, the high recidivism rate of these patients, and the chronic suffering endured by patients with severe and enduring eating disorders, it is important to realistically as-

sess each patient's capacity for recovery or ability to engage in a harm reduction model. At the same time, one must remain in touch with the wishes of the patient, family, and treatment team. In addition, the burden on caregivers and stewardship in the expenditure of health care resources should also be considered when deciding whether involuntary treatment, novel treatments, harm reduction, palliative care, or end-of-life care should be recommended for a particular patient.

End-of-life care is controversial, but it may be of expanding relevance for a narrowly defined set of patients with AN whose illness is severe and enduring. Failure to consider end-of-life care as an option for patients with a chronic and severe psychiatric illness such as anorexia nervosa may perpetuate the stigma of mental illness as separate from physical illness in terms of the compassion we afford its sufferers. Thus, while it is important to adjust the usual treatment paradigm of AN to include involuntary treatment, novel treatments, harm reduction, and palliative care, there is also a need to educate ourselves regarding the possibility of futility as it pertains to patients with eating disorders, especially those with severe and enduring forms of the illness (Westmoreland and Mehler, 2016).

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19

Medical Information for Nonmedical Clinicians and Educators

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COMMON QUESTIONS

What background medical knowledge is needed for health and educational experts (psychotherapists, experiential therapists, social workers, dieticians, educators, coaches) who play a role in identifying, treating, or preventing eating disorders?

How do you approach a student, athlete, or friend you are concerned may have an eating disorder?

When do you refer a person with a possible eating disorder to a medical clinician?

What medical information does a nonmedical health professional (psychologist, nutritionist, social worker) need to know in accepting for treatment a person with an eating disorder?

What medical components of care may health professionals or educators without medical training carry out?

What are some safeguards against legal consequences for an adverse medical outcome while treating or advising a client or student with an eating disorder?

What ethical concerns are present for nonmedical clinicians and educators interacting with clients or students who have eating disorders?

What role does the educator play in the course of eating disorders?

What published resources are available to families?

Case Study 1

Mr. B was a coach for a university cheerleading club that performed at major sports events. Chad, a third-year cheerleader, was 5 feet 10 inches tall and weighed 160 pounds. He complained that he could not lift Jill, who was 5 feet 5 inches and 125 pounds, over his head in a complicated, show-stopping half-time program for the basketball season. Chad asked Mr. B to suggest to Jill that she lose 10–15 pounds of weight. After listening to Chad's concerns, Mr. B suggested that Chad would benefit from a trainer-guided program to increase lean muscle mass, especially increasing strength in the shoulders, back, and quadriceps, so that he could lift Jill in the routines required. He explained that Jill ate normally and was in a healthy, thin-normal range of body fat at 8%. He expressed concern that he might provoke disordered eating behavior in an otherwise healthy young woman by suggesting weight loss. Mr. B recognized the ethical aspects of this gender disparity in completing cheerleader routines, and rather than suggesting the usual change of having the young woman lose weight, proposed instead that the young man gain more strength in a fitness program so that both concerned could benefit with this "win-win" philosophy. Chad's frame would carry extra muscle mass well, as did many male gymnasts his height.

What Do Nonmedical Clinicians and Educators Need to Know about Eating Disorders in Clients or Students?

The Effects of Out-of-Control Dieting and Excessive Weight Loss

The most common abnormal eating pattern in high school and college students is to skip breakfast, eat a light lunch, and then have to fight hunger later in the day. Sometimes this restrained eating or chronic dieting pattern results in desired thinness through perseverance, but many times it leads to binge behavior later in the day. Most people self-regulate body weight in a narrow 4- to 5-pound range, called the *set point*. This range of weight is not fixed in stone but tends to be fairly consistent in each individual. Significant lowering of weight below this point

often leads to involuntary preoccupation with thoughts of food, decreased mental attention, becoming more isolative or irritable, feeling colder than peers, and manifesting changed eating behavior toward either eating slowly and in small bites or gulping food ravenously. The studies by Keys and others in the 1940s on volunteers who underwent experimental starvation to help prepare us to take care of prisoners of war documented these changes as being effects of starvation (Keys et al., 1950). Many of the signs and symptoms of what is called anorexia nervosa are in fact the mental and physical changes of starvation. The component that is truly at the core of the eating disorder illness is the internalized overvaluation of the benefits of slimness or shape change, and the resulting driven behaviors that are employed to achieve these ends.

The Effects of Binge and Purge Behavior

Binge behavior is driven by three factors: hunger, distressed moods, and habit patterns related to time of day or place. Initially, binges simply start as a response to food restriction, weight loss, and hunger. Gradually, binges become triggered by distressed moods, such as anxiety about a test, a low mood from a relationship upset, anger at perceived unfairness, or feelings of boredom and emptiness. Binge behavior is not an ineffective or "dumb" behavior; it helps the triggering situations, temporarily, somewhat like a drug fix—but then creates more problems than it solves.

Most binge behavior initially represents an attempt at anorexic weights that did not work because the body has refused to accept this degree of deprivation, allowing hunger to break through despite will power. Anorexia nervosa of the restricting type is possible only in individuals with the genetic endowment of strong perseverance. The diagnosis of bulimia requires binge episodes, even though people often mislabel purging behavior as bulimia. The term *bulimia* comes from the words for "ox-hunger." Yet, purging behavior takes place in 85%–90% of individuals who experience regular binge behavior; purging is initiated because of the fear of fatness from the unwanted binge calories, as well as from gastric medical distress from the binge. The 10%–15% of individ-

uals who binge but do not purge compensate in other ways: through extreme exercise or through even more severe restriction of food intake. While binges are uncomfortable, the purging usually produces the more dangerous medical symptoms, including a low serum potassium level that can cause heart irregularity, dehydration from loss of fluids, bleeding from the esophagus, chest pain, loss of dental enamel, and enlarged parotid glands (the glands that swell in front of the ears during mumps), to name but a few.

Binge eating disorders (BEDs) are increasingly recognized, most commonly in older adults, and those with BED will often interact with psychologists, social workers, dieticians, coaches, and other nonmedical professionals.

Healthy Nutrition

Educators, coaches, dieticians, psychotherapists, and experiential therapists are often asked to describe good nutritional patterns. There are so many fads and fallacies in the area of nutrition that informal though sincere psychoeducation or guidance in nutritional selection is commonly loaded with personal misinformation, bias, and economically driven misinformation about food choices. Registered dieticians are the most qualified to give accurate advice. In some states, it is illegal for anyone but a physician or a registered dietician to prescribe medical nutrition with reimbursement, although certified nutritionists may be eligible.

Many students and athletes count fat grams as much as calories. The preoccupation with fat grams, not distinguishing healthy lipids (monounsaturates, such as olive oil, fish, nuts) from unhealthy lipids (transfats, saturated fats), is a completely unscientific holdover from recent decades, when all fats were considered bad. From the 1970s through the 1990s, the average weight of US citizens increased while fat consumption dropped. The average student who is counting fat grams tries to live on 0–10 fat grams a day. A truly prudent program of nutrition would allow 25%–30% of daily energy intake as healthy fats. Attempts at 0, 5, or 10 grams of fat are unnecessary, unpalatable, and usually unsuccessful.

For male students and athletes who are wanting to bulk up with lean muscle, carbohydrates have become the recent phobia. Magazines touting muscle development hype low-carb diets as the secret to success. Here again, there is a vast difference between unrefined carbohydrates (whole grains, fruits, vegetables) and highly refined carbohydrates (white bread, sugar, processed foods). Avoiding highly refined carbohydrates is reasonable, while avoiding unrefined carbohydrates is unhealthy. There is increasing evidence that the brain connections between the prefrontal cortex, where executive judgment is centered, and the amygdala, where emotions and impulses are primarily experienced, do not have fully formed connections until about age 25 in males. While this may be an oversimplification of a profound neurodevelopmental process, this basic finding is supported by research. The implication is that many strongly desired but unwise activities during adolescence and early adulthood (driving drunk with a sense of immunity from crashes, going to war, engaging in extreme nutritional programs to bulk up) are pursued without a recognition of their dangers.

Multiple studies support the finding that short-term extreme dieting rarely works. The usual meaning of the word *diet* refers to an attempt to reduce weight quickly by a program that is overly low in calories and poor in nutritional balance. The only weight change plan that works in the long run is a lifetime nutritional plan: eating every day to stay in a healthy range, or if some weight change is medically indicated or is a reasonable personal goal, eating for attaining the desired set point, so that the program does not need to be significantly changed when the goal weight range is reached. For example, a young woman who is 5 feet 4 inches tall and weighs 140 pounds, with visible excess abdominal obesity, and wants to weigh 125 pounds should eat the number of calories (with a balance in nutrition) that would maintain a young woman at 125 pounds, so that when she has achieved that weight, she does not have to change her intake much. Most diet programs that advertise "40 pounds lost in 10 weeks," for example, never give follow-up at 1, 2, or 5 years to state how many dieters have maintained their weight loss. The behind-the-scenes answer is almost none.

Educators, nonmedical clinicians, nutritionists, and coaches should encourage students to think of *diet* as a four-letter word and instead advocate a small cluster of healthy behaviors: good balanced nutrition, regular exercise, and good stress management, along with adequate sleep. Inadequate sleep has been shown to increase snacking and overeating, only recently recognized as an important contributor to abnormal eating patterns.

Evidence is accumulating that there is no benefit from taking vitamins if you are eating a balanced nutritional program (evidence that will be disputed by economic interests). A major factor in helping students eat nutritionally is to have available attractive, tasty, healthy snacks and meals and to remove all soft drink machines from schools and sports facilities. An occasional soda pop will not hurt most people, but it is too easy to become accustomed to the taste and mouth texture of concentrated sweets and transfats (in the form of doughnuts, for example). The best defense nutritionally is a good offense. Studies in Norway have shown that simply having bowls of fruit in a classroom will increase students' likelihood of choosing fruits for the day.

Healthy Weight

Normal body weight is distributed in a bell curve just like height. Most media promote an unhealthy thinness or impossibly lean muscularity (McCabe and Ricciardelli, 2005). The narrow ranges suggested by insurance company tables or other charts for individual weights are often used inaccurately. These weights are averages, not norms. The real measures of health include a percentage of body fat that is appropriate for age and gender, the location of fat distribution (pear shape vs. apple shape), the resting heart rate, absence of signs of starvation, normal patterns of hunger and satiety, and abandonment of the idea that there are any bad foods or foods that should be phobically avoided. Too many physicians tell patients what to weigh too narrowly, basing their recommendations on average insurance table numbers for longest life span—for example, stating that a 5 foot 4 inch woman should weigh 120 pounds.

Instead, the appropriate approach is to take a full personal and family history, use multiple measures of fitness, and identify a range of healthy weight for the patient. Studies have shown that patients with anorexia nervosa will in general pursue low weight to a greater degree than profound religious goals. For example, Roman Catholic girls may skip going to mass because of the calories in the wafer (Graham, Spencer, and Andersen, 1991).

Exercise

We are as much an underfit nation as an overfat nation, as well as a nation that has irrationally invested moral and emotional meaning in artificially thin ideals for women and muscular ideals for men. With only a few exceptions, the body will self-regulate weight in a narrow range when eating is driven by normal cycles of hunger and satiety (rather than in response to emotional needs) and when daily life includes moderate energy output in any form of exercise or physical activity. In general, we encourage "couch potatoes" to get moving and do three to five moderate exercise periods a week of 30–40 minutes, such as walking, in addition to strength training, flexibility, and balance exercises; at the same time, we guide compulsive exercisers to choose moderate, prudent exercise rather than driven, excessive, inappropriate exercise. (See chapter 12 on athletes and eating disorders.)

Diagnostic Approaches

There is nothing sacred or limited to health professionals about asking screening questions to sort out whether a person may have an eating disorder. Educators, therapists, coaches, and others can use the same SCOFF questionnaire that primary care physicians use (see chapter 1), as well as the Screen for Disordered Eating (SDE). These questions should cover the following general areas: Are you extremely concerned about your weight? Does attaining a different weight or shape dominate your life? Are you dieting? Have you lost significant weight? Do you experience binge eating or other forms of eating out of control? Do you

purge after meals? Do you exercise compulsively? Is there an overvaluation of the benefits of slimness or shape change that overrides normal concerns? Some school systems have used the EAT-26 for screening for eating disorders (D'Souza, Forman, and Austin, 2005).

Risk Factors for Developing Eating Disorders

Eating disorders usually occur in individuals with known risk factors (see chapter 1). The more risk factors and the more severe they are, the higher the probability of developing an eating disorder. These risk factors include participation in sports or interest groups that encourage weight loss; a family history of obesity, anxiety, eating disorder, or depression; sensitive, self-critical, persevering personality traits; excess weight gain during childhood or during early puberty; gay orientation in males; and comments from influential individuals (teachers, coaches, and educators) promoting weight loss. Teachers may influence students in unhealthy or healthy habits.

Case Study 2

L.M., a 12-year-old female, developed pubertal changes slightly earlier than her peers. In a health class in fifth grade, she, like every other student in the class, was weighed in front of the class, with the teacher commenting on the weight status of each student. She was told that she was about 10 pounds overweight and should lose some weight. She felt ashamed and criticized. She started a program of severe dietary restriction, cutting out sweets and fats, adding exercise, and becoming preoccupied with the calorie and fat content of foods. Within 6 months she met the criteria for anorexia nervosa, including loss of menstrual periods. For the next 6 years she required continued regular care for her low weight, her morbid fear of fatness, the development of binge-purge behaviors, and overexercising. She avoided social events involving food. When she went to Europe on a student exchange program, she was referred to an experienced clinician who helped stabilize her weight, change her core psychological overvalued beliefs in the necessity of extreme thinness, and begin a pattern of

healthier weight and eating. She remains preoccupied with weight but has had return of menstrual periods and continues in psychotherapy.

This is a typical case of an eating disorder in a young person that was precipitated by an unwise educational practice. The evidence is compelling that describing the symptoms of eating disorders to students increases rather than decreases the probability that some students will develop an eating disorder, usually anorexia nervosa or bulimia nervosa, or atypical forms of these disorders.

When Should a Client or Student Be Referred to a Physician for Evaluation of an Eating Disorder?

It is good practice at the beginning of psychotherapy or nutritional therapy for any client or student with an eating disorder to have a medical evaluation (table 19.1). This referral may disclose hidden medical complications from the eating disorder, but at a minimum it offers a baseline of medical evaluation so that future symptoms can be compared with this baseline. It is helpful to have an established relationship with a primary care physician or advanced practice nurse having expertise about eating disorders in young people. This eliminates an inexperienced medical approach to individuals you may refer for evaluation and avoids unnecessary, excessive testing. Subjective complaints that

TABLE 19.1. When to refer a client or student to a primary care physician

▪ At the beginning of psychotherapy treatment, for evaluation and to establish a liaison relationship

▪ When the client or student has any of the following symptoms:
  ▪ Severe or rapid weight loss
  ▪ Dizziness, lightheadedness, fainting (hypotension)
  ▪ Uncontrolled binge-purge behavior (electrolytes often abnormal; cardiogram may be abnormal)
  ▪ Constant coldness
  ▪ Medical distress, such as chest pain (often from purging behavior) or abdominal pain

▪ For psychopharmacology augmentation (preferably by a psychiatrist) to psychotherapy
▪ Periodic checkups or weighing (often performed by physician's assistant or nurse)

should lead to prompt referral to a primary care physician include constant coldness, faintness or lightheadedness, heart palpitations or chest pain, shortness of breath, abdominal pain, or frequent purging behavior, including statements by a client about purging or using many diuretics or laxatives a day, and certainly if Ipecac, an over-the-counter medicine to induce vomiting in kids who have eaten poison, is used. (Ipecac is no longer recommended for poison response.) Common sense and professional judgment will also guide clinicians and educators to refer clients or students when they note significant weight loss; a thin, bony appearance; bluish hands; a cold handshake; or an overall appearance of bad health. Collateral information from parents or friends about excessive purging behavior should also generally lead to referral.

At times, medical referral is deferred because a patient is developing insight. It is important to realize that while developing meaningful insight is critical, there is a huge difference between purely intellectual insight and behavior-changing insight. Insight is useless unless it leads to sustained behavioral change. Valuing intellectual insight in the presence of continued severe weight loss, failure to restore weight, or a lack of change in significant binge-purge behavior is an illusion. The patient has not made progress when they gain and then lose weight repeatedly, with multiple excuses and promises to do better next session. Behavior is behavior is behavior. When a patient with an eating disorder is in regular treatment with a nonmedical professional, agreements should be made for the frequency of interaction with the medical care team.

When Should a Client Be Accepted for Therapy on Referral from a Physician?

Accepting a client from a physician should ideally involve sufficient assurance from the physician that the patient is well enough to come for nonmedical treatment, primarily psychotherapy and experiential therapy (table 19.2). It is important to record agreements regarding when the patient or client should be referred back to the physician and to set up both regular and as-needed communication patterns between the therapist or coach and the physician. It is also important to establish who

TABLE 19.2. When to accept a patient for therapy from a referring physician

▪ After medical evaluation is under way
▪ After establishing frequency-of-contact schedule and conditions for return to see physician
▪ After decisions are made on frequency of checking body weight and who will weigh patient
▪ After deciding who will instruct patient on healthy nutrition and prescribe a nutritional program (physician or registered dietician)

TABLE 19.3. How to collaborate effectively among team members

▪ Establish a regular, agreed-on communication schedule.
▪ Set clear, specific conditions for referrals (for example, to dietician, social worker).
▪ Clearly define the roles of the various team members.
▪ Share techniques guiding the overall treatment.
▪ Set goals for patient care within each discipline that are specific, time limited, achievable, and measurable.
▪ Decide who is "captain of the ship."
▪ Decide on a single consistent guiding psychotherapeutic strategy, such as cognitive behavioral therapy.

performs what roles regarding shared medical functions such as weighing the person. Thus, it is inappropriate to refer an individual for treatment without specification of who will follow weight or monitor binge-purge behavior. Agreements should be in writing and contacts recorded in the medical record. The steps of effective collaboration are summarized in table 19.3.

What Medical Responsibilities Can Be Carried out by Nonmedical Clinicians?

A medical degree or specific training is not required to weigh a patient or to encourage healthy behaviors such as good nutrition. These are common-sense practices that can be carried out by nonmedical clinicians and certainly are done frequently by coaches and educators, not only by dieticians and physicians. Coaches often monitor resting heart

rate for assessment of athleticism. The key here is to be clear on who does what, and under what conditions the client or student needs to be referred back to the physician. Some psychologically trained therapists do not like the idea of weighing a client, or carry outmoded ideas that weighing a client would somehow interfere with psychotherapy. Sometimes it is simply not practical or is too expensive for a client to return to a doctor's office weekly for weighing when it can be done in a therapist's or a nutritionist's office. Agreements need to be recorded about a weight minimum or simply the lack of progress in the weight restoration process that will prompt referral to more intensive medical treatments. A psychotherapist can ask a patient to keep a nutritional record to assess whether there is compliance with the nutritional prescription, emotionally based alterations in eating, or the presence of binge-purge behaviors. The patient can use a simple 3×5 index card or their laptop or iPad to record each day, in columns, the time of day, food eaten, emotions experienced, and events taking place. This record can form the basis for beginning the psychotherapy session.

How to Avoid Legal Problems

The key to avoiding legal problems for comprehensive care includes good record keeping, documented decisions about when to refer a patient to a physician (erring on the side of more rather than fewer referrals), documentation of regular communication with the primary care physician and other team members, and acting with knowledge and common sense. Letting a patient lose weight in front of your eyes because they are developing "insight," failing to refer someone who simply looks very ill, perpetuating irrelevant or out-of-date nutritional details about calories or fat grams in place of sound nutrition, failing to document the communications with a primary care physician—all would open one to being included in litigation if adverse medical consequences occur. This does not mean that medical referral is necessary for mild but improving starvation, occasional binge-purge behavior, and so on if a baseline medical evaluation is available and the person has a primary care clinician. It takes time, knowledge, and the development of professional

judgment to balance the intrusiveness of medical referrals versus their necessity after the initial evaluation. Working within your limits of qualified training is essential, as is having a team-oriented approach (see chapter 2). Litigation is rarely a concern when treatment is sound, adequate records are kept, and good patient-clinician relationships are maintained. When these guidelines are followed, the treatment and education of clients, students, or athletes with eating disorders will be satisfying and effective.

Information for Educators

The most common question asked by educators is how they should approach a student whom they are concerned may have an eating disorder. Seeing that someone is losing significant weight, passively watching a student consistently exiting after meals or snacks to use the restroom, or becoming aware of significant mental preoccupation with weight and shape should all be signs that alert the educator to the possibility of an eating disorder. Educators are often approached by friends of a student who tell them about behaviors that the educator may not see directly.

The best approach to a student about whom you are concerned is simply to approach the student and say that you are concerned that an eating disorder may be present and that it is essential for them to see a trained individual or clinic for an evaluation. Depending on the age of the student, the parents may be informed and their help solicited. See table 19.4 for suggestions about how to approach a student about who may have an eating disorder.

Educators have multiple important roles. One role is preventive intervention, an ideal that is seldom achieved but increasingly possible (Piran, 2004; Russell-Mayhew, Arthur, and Ewashen, 2007). Educators may attempt to prevent eating disorders by talking with students about eating disorder symptoms, but this approach has a contrary effect of increasing a tendency to eating disorders. Instead of more information about signs and symptoms, students need to know how to develop skills of healthy thinking and behavior in a media-saturated atmosphere that

TABLE 19.4. How to approach a person you are concerned may have an eating disorder

▪ Educate yourself about eating disorders from accurate sources.
▪ Be caring but firm in approaching the person.
▪ Share your concern about the possibility of an eating disorder being present. Share what you have observed kindly as evidence for the presence of a problem.
▪ Gently but firmly tell the person to schedule an appointment with a qualified health professional or eating disorders clinic for assessment, and then "I'll be off your case."
▪ Don't oversimplify the issue or assume that time will improve the disorder.
▪ Don't imply that bulimia nervosa is less serious because there is no obvious emaciation.
▪ Express your concern privately.
▪ Don't diagnose.
▪ Don't become the therapist or savior or offer short-term, oversimplified solutions.
▪ Do act in an emergency, such as the presence of chest pain, suicidal thinking, or passing out, and get help immediately.
▪ Assure the person that you will maintain friendship through the process of becoming well.
▪ Encourage the continuation of spiritual growth without "spiritualizing" or "moralizing" the problem.
▪ Be direct and nonpunitive.
▪ Avoid arguing.
▪ If there is no response, consider sharing your concern with an authority figure, such as a teacher, coach, or parent.

emphasizes thinness, shape change, or extreme muscularity with low body fat as the most important achievement in life. Runi Gresko, Norway's leading educational expert on prevention of eating disorders in schools, has taught, "Eating disorders are not prevented by talking about eating disorders" (personal communication, 1993).

Almost certainly, if primary prevention is possible, it will happen through insulating vulnerable individuals from our culture's relentless demands for thinness, shape change, or overemphasis on muscularity. That eating disorders can be prevented is only partially and selectively proved, but there are indications that the keys to successful prevention

include teaching increased assertiveness to young girls especially, helping students be critical of media claims promoting the value of thinness, increasing body self-esteem, and teaching valuation of the normal diversity of weights while strongly encouraging fitness at every set point. Girls benefit by moving from a judgmental and objectified view of their bodies to an operational, instrumental, and functional view, seeing the body in terms of how to increase its functionality, not primarily a focus on how it appears to the self or to the critical judgment of others. Setting minimum weights and BMIs below which a person may not participate in a sport or an activity (wrestling, ballet, cross-country, skating) shows some promise of decreasing the behavioral incentive to slim more.

Certainly, weighing students in schools, even when the staff are ostensibly concerned about health, has no health benefit but instead causes embarrassment, psychological distress, or humiliation in young people, as well as promoting unhealthy and unnecessary dieting. Spotlighting an individual's weight as being excessive may be the starting point for an eating disorder. Educators are role models who need to take seriously their responsibility to teach fitness behaviors in place of dieting and to stress the normality of a wide range of weights. Their personal examples of fitness behavior and healthy weight ranges, as well as their openness to discussion, are essential.

There is increasing evidence that for mild cases of eating disorders, especially bulimia nervosa, self-help modules through internet-assisted computer methods may be effective (Ljotsson et al., 2007). The internet is a medium that may offer young sufferers a helpful program for their needs. Educators can sensitize students to the perils of "pro-anorexia" websites by exploring such sites with students and teaching the students to critique and deglamorize them.

Information for Coaches

There is increasing evidence that setting lowest acceptable weights for participation in sports, and maximum rates for weight loss and body fat decreases, helps to decrease the onset or perpetuation of eating disor-

ders, especially in wrestling, ballet, and long-distance running. When there is no advantage to further weight loss in ballet or wrestling ("You will have to leave the program if you lose more weight"), then there is less incentive to keep on losing. In Wisconsin, for example, a legislative action has required high school wrestlers to limit the amount of weight lost for qualification for a wrestling weight category and has set minimum body fat levels for participation eligibility. These efforts, partially a response to several deaths in young wrestlers associated with weight loss practices in the 1990s, appear to be effective.

In sports, where weight for achievement of performance goals or appearance judgments may conflict with an individual's health needs, ethical and health decisions need to be integrated, with the athlete's best interests being the first concern. An individual whose performance in sports is maximized only when there are signs of abnormal eating behavior, or when there is distressed thinking concerning weight and eating patterns, needs to be counseled about the relative importance of health versus performance. Unfortunately, many young men and women live in a bubble of illusory invulnerability, whereby they fail to appreciate that some performance goals are simply not worth the health costs. Elite athletes are often driven internally as well as externally through a narrow focus of interest, relentless perseverance, and self-critique. Reports have documented the presence of the "elite athlete triad" of abnormal eating, weight loss, and osteoporosis in young female athletes (Sundgot-Borgen, 1994). Mild to moderate eating disorder syndromes in athletes are missed or overlooked when these athletes are performing with excellence. The case at the beginning of the chapter suggests that women often carry a disproportionate burden to lose weight in couples' sports such as cheerleading and pairs skating. The male partner could just as well be advised to get strength training. It is a personal opinion that the lower and lower weights and younger ages characteristic of girl gymnasts in recent Olympics represent an ethical concern. Bonci et al. (2008) describe how athletic trainers can help to prevent, detect, and manage athletes with eating disorders. The recent Olympics in Brazil may indicate support for a healthier body habitus for female gymnasts.

Information for Parents

Parents play major roles in shaping the course of eating disorders in their children. While too often blamed when they are in fact doing their best, parents may exert excess pressure for girls to be thin and boys to be muscular (McCabe et al., 2007). Only recently appreciated are an understanding of the stress of parents living with a child with anorexia nervosa (Kyriacou, Treasure, and Schmidt, 2008) and the needs of siblings of adolescent girls with anorexia (Honey and Halse, 2007). Treasure (1997) has written a survival guide for families, friends, and sufferers. The role of the parents varies with the age of the child with the eating disorder: the younger the child, the more involved the family must be. Families need support, information, and a sense of realistic optimism about the outcome of treatment for eating disorders. Parents also have a role in monitoring computer usage by their children, allowing appropriate changes as children reach their later teen years. Pro-anorexia sites are abysmally silly and unattractive from the viewpoint of an adult without eating disorders and good self-esteem, but they may have a strange fascination for a subgroup of teens. Talking about these issues openly from a neutral viewpoint may be helpful. Research is robust that having regular family meals most days of the week decreases the probability of a teen developing an eating disorder or drug abuse problem (Ackard and Neumark-Sztainer, 2001; Fulkerson et al., 2006). Parents benefit from knowing about the increasingly well-documented contribution to eating disorders, helping them understand these are true illnesses, not simply a lack of effort or a result of following social fads.

Summary

A combination of medical knowledge, common sense, and a good working relationship with medical professionals will allow for the shared treatment of clients and students who have eating disorders, as well as for the possibility of prevention, and, certainly, the early identification of eating disorders. Good record keeping, documenting regular communication with a medical professional, having a low threshold for refer-

ral when appropriate, and recognizing the ethical aspect of weight and shape change versus performance goals will enhance the significant role that nonmedical mental health professionals, educators, coaches, and nutritionists have in the care of young individuals. The recognition, treatment, and prevention of eating disorders involves a "village," or community of caring, concerned individuals who are knowledgeable, thoughtful, and proactive. Recent books for families and professionals follow in the references.

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20

Innovative Psychological Treatments of Eating Disorders

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COMMON QUESTIONS

How can asking patients to write a letter to their illness ("You are my friend because . . ."; "You are my enemy because . . .") increase their engagement in treatment and in becoming coinvestigators with the clinician?

How can clinicians use the power of stories to convey important psychoeducational information about eating disorders?

Can psychotherapy be prescribed analogous to the prescription of medications?

What psychotherapeutic methods have been recently introduced or expanded in the field of eating disorders?

What does *third-wave behavioral interventions* mean?

What does adjunctive psychotherapy mean?

What is CBT-E?

How can clinicians use a sequence of psychotherapeutic methods to comprehensively treat patients with eating disorders?

What are some general principles summarizing the prescription of psychotherapeutic methods for eating disorders?

Letters I Write to My Illness

Most patients have, at a minimum, ambivalence about treatment and often are in frank denial about the seriousness of their disorder. Entry into a treatment program means only that a patient is physically present. The real challenge is persuading patients to find motivation and to engage in treatment. The core treatment of eating disorders is psychotherapy. Eating one's way out of treatment results in a revolving door to readmission. Based on two seminal and heuristic publications by the London group led by Janet Treasure and Lucy Serpell and their colleagues (Serpell et al., 1999; Serpell and Treasure, 2002), writing "A Letter to My Illness" results in a patient's enhanced engagement and increased motivation. In addition, patients become partners in the journey to wellness.

What does not work? Scaring patients does not work. While they need psychoeducation about the medical, social, and developmental risks of their eating disorders, this information alone is not sufficient. Speaking in psychobabble or in other overly technical terms may result in nods but little internal understanding. Authoritarian approaches may work in some surgical specialties but not with eating disorders. Authoritative, yes; authoritarian, no.

Why are engagement and motivation challenging issues for patients with eating disorders? Anorexia nervosa is almost always ego-syntonic. Patients with anorexia nervosa do not see themselves as ill, or if they grudgingly acknowledge their illness, they have enormous fear of becoming fat and losing control. Patients with bulimia nervosa most often have a strong sense of shame and are filled with guilt. In contrast to other psychiatric illnesses, such as schizophrenia or major depressive disorders, eating disorders are functional and adaptive, giving patients rules and routines, rituals and methods of dealing with various issues, including existential fears of maturation, self-treatment for dysphoria, strategies for controlling families, and multiple other age- and gender-specific issues.

How do patients with eating disorders present to the therapist? They often present with arms folded, prepared to resist, feeling cornered and

threatened. They generally suffer from alexithymia, finding it hard or impossible to identify feelings except for the "feeling" (really a thought) that they are fat. Insight into the seriousness of the disorder is lacking. They are most commonly entangled in their illness, believing, "I *am* my eating disorder," rather than "I *have* an eating disorder," as one would say about having influenza, for example.

The publications of Serpell and Treasure et al. have been too much overlooked, for multiple reasons: they present results free of statistics, have no control group, prescribe no medications, involve no laboratory studies, and include no genetic studies or brain imaging. These publications are simply enormously useful. Writing "A Letter to My Illness" is a helpful and widely applicable approach to enhancing the engagement and motivation of patients in treatment.

Background of the Benefits of Writing

In *Fasting Girls*, Joan Brumberg of Cornell University examined the diaries of girls of the nineteenth century. She found that girls of more than a century ago experienced menarche much later than girls today; as a result, they were able to navigate the shoals of puberty later, within a more structured social context (Brumberg, 2000). The Nun Study of Aging and Alzheimer's Disease, which began in 1986, analyzed the autobiographies of women entering a religious order at an average age of 22. The investigators correlated the linguistic density of these early autobiographies with the onset of cognitive impairment and Alzheimer's disease: the more linguistically dense the autobiographies at this early age, the later the onset of cognitive impairment (Iacono et al., 2009).

The practical side of the process is to ask patients 2 to 4 days after admission to write on one sheet of paper "A Letter to My Illness: You Are My Friend Because . . . ," responding in paragraphs or bullet points or in any manner comfortable for them. Then ask them to write on a second sheet of paper "A Letter to My Illness: You Are My Enemy Because . . ." The patient then reads their letters aloud to the team (or to the therapist) the next day. It is often astounding how a seemingly tongue-tied, alexithymic patient responds fluently to this task.

Writing these letters accomplishes several goals.

1. The focus is changed from a spotlight on the patient, who may feel pushed into a corner, to a focus on the illness. The patient is okay; the illness is not.
2. The patient becomes a coinvestigator with the therapy team or therapist: "We are on the same side."
3. The writing process respects the intelligence of the patient.
4. It engages more of the patient's hidden brain-based skills than do question and answers.
5. It individualizes the patient's history and psychodynamic themes.
6. It utilizes a patient's most recent functioning activity: school (for the younger patient) and work (for the older patient) are usually continued despite the presence of severe illness. Starved, cachectic patients and patients who binge or binge and purge tend to go to school or to work no matter what.
7. It uses the language of the patient, not the language of the therapist—it is a stealth entry into the patient's experiences.

There are multiple benefits for the therapist or therapy team.

1. This process integrates well with cognitive behavioral therapy (CBT). Meaningful therapy almost always involves homework, both with these letters and with CBT.
2. These letters individualize the patient—each letter is unique; no longer is the patient simply another starved, cold, resistant patient in a long line of similar-appearing patients. Looking forward to what these patients write increases the pleasure for the therapist and decreases the burnout potential.
3. These letters bring the patient out from the wallpaper—patients stand out separate from their illness.
4. The therapist has the privilege of finding out what is going on "behind the curtain" of the illness.
5. It sets up a lifelong pattern of self-investigation and insight that can contribute to continued effective treatment.

6. Letters written close to discharge are an effective measure of treatment progress and, as the example below illustrates, of long-term recovery.

Letters Written during Illness and after Sustained Recovery

This is how a 49-year-old married woman viewed her illness 30 years ago:

YOU ARE MY FRIEND:

- Because it makes me feel in control
- Because I am thinner than everyone else and I like that
- Because it makes me feel confident and strong
- Because I can read a cookbook and it makes my hunger go away
- Because I like my clothes loose
- Because people compliment me on how skinny I am

YOU ARE MY ENEMY:

- Because I always think about food, and I am tired of it
- Because I sometimes feel like I am going crazy
- Because I can't spend time with my sisters to go shopping because I haven't run yet, and I have to do that first all the time
- Because I can't go out to eat with my family
- Because I don't want to socialize or talk to people anymore; I have no friends
- Because I am anxious and frightened all the time
- Because I am tired and don't feel good

This is what the patient writes now, in her sustained recovery:

YOU ARE MY FRIEND TODAY:

- Because when I am stressed, it makes me not have to think about anything
- Because I still like to be skinnier than others

YOU ARE MY ENEMY TODAY:

- Because it would not let me be a part of my friends' and family's life
- Because it would not let me try new things

- Because it keeps me from dealing with my real emotions
- Because it makes my family worry about me
- Because I know it is behavior I do when I am not dealing with my real issues
- Because I am proud that I recovered, and I don't want to fall back into old behaviors

Patients are told about *normative cultural distress*, a shrewd concept, described by Rodin, Silberstein, and Striegel-Moore (1985), reassuring patients that having occasional thoughts about dieting and giving lip service with peers to the desire to be thinner (while having dessert) is quite normal, provided the behaviors are healthy and disordered eating thoughts do not dominate their lives.

Some patients write what is almost a romance or love story about their eating disorder: "You are my friend because you are always there for me; you rescued me when things were chaotic—my boyfriend left me; my family moved to a new area; no one liked me in the new school; everyone was thinner than I was. But I could depend on you to help me. Never go away. I need you." Details of their lives are revealed as they face their illness, rather than facing someone who, in their eyes, is going to take away their way of life.

Here's a letter by a young man with anorexia nervosa:

> You are my friend because our relationship has lasted 18 years. We have been through many trials and challenges together, but we have always managed to get back together because our relationship has taken priority over everything else. School became a threat to us, but we stayed firm to preserve our relationship. I was tempted to make long-term commitments, but this would have forced me into adulthood before I was ready. You were exciting.
>
> You are my enemy because you have stolen so much from me. Every time I turn my back and let my guard down, you are there to pilfer more. I despise you. Where is my identity? It has been stolen. Where is the athlete? Gone! You have taken small pieces of my self-esteem consistently over time. You are crafty. I demand you return my life, my spirit, my core. You are slippery, illusory, and cunning.

This letter was written by a gay man with bulimia nervosa:

> I grew up with a very abusive, neglectful mother. My father was an alcoholic. I was picked on constantly by other kids. I became very looks and body conscious and would try anything to be attractive. When I started lifting weights, I liked the reaction I got from people being intimidated. Nobody picked on me anymore or bullied me. If you are not thin, muscular, and sexy, you are invisible. Being thin, muscular, and attractive is all I have to offer. Our culture wants everyone to be beautiful and young.

In summary, writing a letter to their eating disorder is an innovative method to increase patients' engagement and motivation in treatment. This method also benefits therapists and therapy teams by individualizing treatment and forming a therapeutic alliance.

Stories I Tell My Patients

Storytelling is an old and honored art. Great literature conveys its message to us in stories. In a way, the stories remain the same—they tell of life, love, death, birth, hope, loss, discovery, fear, quest, beauty, humor, truth, falsehood, despair, faith, and all the other issues of life. Some time ago I felt stuck in interacting with my patients in psychotherapy, and on rounds I realized we were trading words rather than making real progress. I began to tell short stories to find a way around the intellectualized defenses and to get to the issues. It seemed to get attention, sometimes sowing a seed, sometimes challenging a wall of resistance. No matter how old we get, we can relate to roses and bike trips, to shadows and ketchup. Sometimes the challenge of treatment may be helped by stories.

Where Are You When You Are Eating?

A group of four high school students were having lunch at the new Southwestern Grill on a Saturday afternoon. It was a fun restaurant—with real cactus plants, warm colors, and authentic food. After the food arrived, John immediately thought back to how his wrestling coach had

warned him about getting too fat in the off-season. With mechanical action he ate, but his mind was on that interaction and on his determination that he would never again be criticized for his weight. "Hey, get off my case, Coach," he wished he could say, but John couldn't get off his own case. Gun to the head, he couldn't say after the meal what he had eaten.

Sue got nervous as soon as she took one look at the tortilla chips and mango salsa the waiter put on the table before they even ordered, and once her Santa Fe chicken salad arrived, Sue feared that she would never fit into her prom dress in a few weeks. While her jaws worked, she saw herself trying to squeeze into her beautiful gown, its seams tearing on the dance floor. The image felt so real that she could sense her rising anger and disgust about her hips. Food? What food? The plate was clean, but so was any memory of the food.

Joan ordered a plank steak with hot peppers, but you would never know it if you asked her afterward. That food, that smell, that plate radiated anxiety and brought her back to when her boyfriend had patted her on the hips and said she was getting a little heavy. Her stomach churned, and she could barely swallow. She relived the rage that had boiled inside, but she stuffed down the anger so she wouldn't lose the boyfriend. She thought of all the things she could have said, but never did.

Brad decided on the house special, which sounded good when the waiter described it to him. While he was eating, he had no idea what the food tasted like. It was like a silent movie. He said some things to the others but couldn't remember what. It was a script. His mind was on those years in junior high school when he had been teased mercilessly as a "fat slob." The humiliation was still there, tingling in his bones, even three or four years later, and it was certainly more real than the flavors of the house special. His body was drenched in sweat, and even with these close friends, he was afraid that they were judging him.

When they got together on campus on Monday afternoon, it was funny, sad, and puzzling. None of them could remember what they had had for lunch on Saturday. In fact, none of them could remember their morning showers or even what they'd studied in their classes moments earlier. Their minds were everywhere else. One look at their abdomens

and hips for Sue and Joan sent them into old tapes of how much they disliked their bodies. One look at their chests and shoulders sent John and Brad into a panic about the past and the future. They were there, but they weren't there.

Jon Kabat-Zinn (1990), a pioneer of the powerful therapeutic method of mindfulness, which has shown utility across many diagnoses, asked a question of the audience during a lecture: "When you took a shower this morning, where were you?" Almost everyone in the audience smiled. Almost no one had been really in the shower, feeling the water, letting it play over the hair and run down the body in warm rivulets of pleasing sensation. Some were into their schedule for the day. Some worried about yesterday's sessions. Others rehearsed the anger they had swallowed when talking to their supervisors the day before. Almost no one was where they were, as far as their minds were concerned.

We can help our patients deal with meals by having them practice here-and-now mindfulness about the process of eating—being present with all their feelings, especially the pleasant sensations the body is set up to perceive when hunger is satisfied, and with a social interaction that is real, not mechanical. When clinicians and therapists first practice being where they are at that moment, with full awareness, without judgment, with openness to what thoughts and feelings are going on, they are on their way to helping patients be mindful. Let's help our patients, when they are eating meals, to be really present, to experience taste and scent and social interaction, letting the past stay in the past, and the future in the future. Being healthy means being aware of and enjoying the sensations of eating in a social context. Be mindful. Help our patients be mindful. Let's give that "monkey mind" some rest.

Follow Your Star

Sam: "Our Day Program for Eating Disorders is close to completion for this group. You've all worked very hard. I'd like to hear what your plans are after the group is over."

Brittany: "I'm excited by all the expectations I have. I expect to be free of all the preoccupation with weight I used to have. I expect to get a

perfect job, and to do it perfectly. My expectation for relationships is to have lots of friends, to never argue again with my family, and to make every day a sunny one for myself and everyone around me. That's the star I'm following."

Ted: "I'm excited too, but my expectations are way different. I expect my life will be all positive thinking, no more negatives. Those urges to binge and purge will all be gone. I expect to exercise with perfect form and moderation; no more of the out-of-control exercise. My star is sending me strong rays letting me know that I can't fail."

Brynn: "There are going to be a lot of stars in the sky for me, too. My boyfriend tells me that he expects me never to compare myself with others like I used to do. That was the pits—always seeing myself as fat and stupid. Never again. I expect I'll be a perfect French major with my improved concentration. In fact, I'm calling my star 'ma belle etoile,' French for 'my beautiful star.'"

Sam: "I can tell you are all enthusiastic about being free from your eating disorders. You've all worked hard to put anorexia and bulimia behind you. Let me try out an idea and see what you think. I'm going to suggest you follow a different star, one I put in capitals: STAR. To my mind, expectations are demands in disguise. They are recipes for disappointment, not that you mean to do that. There's also some passivity to expectations—a kind of demand that things should happen a certain way, or that a certain event should happen. What do you think of substituting the STAR system of *goals* in place of *expectations*? Use the STAR memory trick.

"The *S* stands for specific—not vague, like being a perfect student or being completely happy each day. How would you know what a perfect student is? Or what a perfect day is? In contrast, consider a specific goal like learning 30 phrases of conversational Spanish. That's clear and specific.

"The *T* stands for time limited—for example, having a goal of learning 30 phrases of Spanish in 30 days. That gets you out of the having to do it yesterday, or never getting around to it.

"The *A* stands for achievable—meaning your goal is very reasonable and can be accomplished by normal human beings. Almost

anyone can learn 30 phrases of Spanish, as opposed to being a perfect Spanish student. Perfection isn't achievable, and the alternative is to set yourself up for the self-label of being a failure when you are not fulfilling your expectation of being a perfect student.

"The *R* stands for ratable—the goal can be measured. Measured, not judged. For example, you can ask a Spanish-speaking friend to listen to your answers when she reads out the phrases in English and you answer in Spanish. Compare that with the unratable expectation of being a perfect language student, or always being cheerful every day—everybody's going to be grumpy some days. By the way, if you expect to be cheerful every day and get out of bed grumpy, you can have a goal of going back to bed and getting out on the opposite side.

"So, now you can have a goal of 30 phrases of Spanish in 30 days, a specific, time-limited, achievable, and ratable goal that will be satisfying to accomplish. What do you guys think?"

Brittany: "That makes sense. I feel a lot of pressure off my shoulders. I didn't realize I had been painting myself into a corner with my expectations."

Ted: "I know I can plan on doing 30 minutes of moderate exercise a day, four days a week, in place of the expectation of a perfect moderate workout."

Brynn: "It feels like I'm getting away with something when I don't have to be perfect in fulfilling my expectations, but I like it. You're right. I wouldn't know when I had become a perfect French student."

Sam: "Well, guys, let's try following the STAR plan for goals after group is over, instead of impossible expectations. These two words, *impossible* and *expectation*, are sort of redundant, anyway. The neat thing about goals is that they can be changed when it's appropriate. They're flexible. They can be modified to more or fewer, to speed them up or slow them down. There's only a learning experience, not demands in disguise. See you for the final session next week."

So many of our patients have perfectionist traits and suffer from unreasonable expectations and then think of themselves as failures.

Expectations are seldom achievable and usually reflect all-or-none reasoning. What a recipe for disappointment and failure! Saying "I expect never to argue with my parents again" is like expecting warm weather in January in Iowa. Sometimes it happens but don't expect it. You'll be very disappointed. How much better are the STAR goals? Patients usually feel relieved and freer with STAR goals instead of expectations. You can flex goals—they can be revised as experience shows that you can learn 30 Spanish phrases in 15 days, or maybe need 45 days. It works for our relationships with patients to have treatment goals, not expectations. We're vulnerable to our own impossible expectations and feeling worn out because these hidden expectations are not specific or achievable. Go for goals. Follow the STAR.

Halt, Bad Dog

It kept lunging at her. Tamara felt as if she was being attacked by a bad dog. The attack continued relentlessly. She placed her hands on her throat for protection. "How did I get myself into this position? I swore it would never happen again. I said I'd never let myself get this vulnerable." All she could think of was running away. But she knew from experience that it would follow her wherever she went. She couldn't outrun it. Running didn't help. She'd like to call a friend, but there wasn't time. The assault grew stronger. Her breath grew rapid and shallow. She knew she couldn't avoid it any longer. So she opened the freezer, took out the quart of macadamia nut ice cream, and swallowed it in chunks as fast as she could. Then came the last of the brownies.

Her mother called out to her: "What's wrong, Tamara?"

"Nothing, Mom. I had the crazy idea I was being attacked by a bad dog."

"That's silly," her mother said. "Our miniature dachshund never attacks anyone. She would give her hot dog to a burglar."

"Yes, Mom, I know."

In her weekly session, Tamara explained to Kay, her therapist, how it felt. "The attack got stronger and stronger, and I wanted to say 'Stop,' but it didn't stop." Kay gently told Tamara that she had indeed suffered

an attack, a binge attack, which Tamara had related in her own somewhat dramatic way. Kay decided to use the concept of a dog attack, and she shared an approach with Tamara.

"Tamara, let's think of your binge urges as attacks by a bad dog. And then let's stop them by saying, 'Halt, bad dog.'"

"That's nuts. Even I, with my dramatic tendencies, know there's no real dog."

"Yes, but that's a better analogy than you may realize. Here's what I mean. Most binge urges, like alcohol urges, occur when a person feels crummy. The alcohol rehabilitation counselors have taught many alcohol-abusing patients to say 'HALT' when they have an urge to drink. That's a shorthand way of saying, 'Are you hungry, angry, lonely, or tired?'—four of the biggest triggers for an alcohol binge. Asking yourself which specific crummy or dysphoric feeling you have as soon as the binge urge starts allows you to deal directly with the trigger, and that stops the attack. You've learned in therapy to practice specific, different approaches when you are hungry, or angry, or lonely, or tired. I'm going to suggest you not only say 'HALT' to stop the dog in its tracks but say 'HALT, BAD DOG!'

"You know the HALT feeling words. Continue the mnemonic: BAD stands for bored, anxious, or depressed. DOG stands for demoralized, overwhelmed, and given up. The moment you feel that attack coming—and I do want you to continue to visualize it as a bad dog attacking—say out loud, if no one is close to you, or say to yourself, 'HALT, BAD DOG.' Then deal directly with whichever feeling is asking for a pseudo-solution through a binge—the bad dog attack really is halted. Try it, Tamara."

The HALT mnemonic has been used for some time with patients who abuse alcohol. It applies just as well to binge urges. Frank, my cotherapist and an expert social worker, and I decided in our group to try out a more complete phrase: HALT, BAD DOG. The key is getting a patient to inhibit the binge response for just a little while so that, while sitting on the urge, there is enough time to give the dysphoria a name. Our patients then use the different approaches they have learned for different types of emotional distress. Once an accurate name is given, by thinking HALT, BAD DOG to remind themselves, they can use the appropriate action

plan. Some people carry reminders on 3 × 5 index cards or a smartphone in their pocket, one for each letter of HALT, BAD DOG. It gives power to the patient to know that these attacks can be slowed down, held off, and then avoided completely. Try it with your patients. Ruff. Ruff.

The 5:36 Express from Paddington Station

Paddington Station was as busy as usual, with end-of-day commuters rushing to their train compartments to head for home. London was grimy, soot filled the air . . . it would be oh so pleasant to return to the countryside for a long summer evening. As everyone knows from reading Agatha Christie and other British mystery writers, murders take place on the Paddington Express. Today, it was not to be murder, but something that seemed almost as awful—at least to Jane. You enter British trains, at least the older ones, directly from the train platform, stepping up into a carriage through the little swinging doors. Jane briskly entered her compartment and uttered a sigh of relief that she would be alone for the journey, alone with her thoughts, letting the work of the day slide from her mind with each passing mile. Just as the train was about to leave the platform, a burly man with a handlebar mustache jerked open the door and entered. Oh well, you can't always control those things. But the situation soon became worse than simply having to share a compartment. The burly man needed a shave and a bath. As soon as the train picked up speed, he pulled out an enormous salami that reeked of garlic. Then he opened a tin of sardines. Jane was not sure what the last straw was—taking off his shoes and socks to clip his toenails, or spreading out the dirty contents of his suitcase on the vacant seats.

Jane thought: "Now what are my options? I can kill him and throw him from the train, but I wouldn't want to take someone's life. I could jump from the train myself and hope for the best. That would not be very helpful either. The train must be going 55 miles an hour. I could pitch a fit, jump up and down, cry, scream, and glare at him, but I know the other carriages in the train are full, so there is nowhere else for him to go. I suppose I can settle back, read my book, let the unpleasant man be as

he is. Then I will breathe a sigh of relief when he gets off at his stop or when I get off at mine, whichever comes first."

And that is what Jane did on the 5:36 from Paddington Station. The unpleasant man, with his inconsiderate behavior, his salami, his sardines, his socks and nail clippings, his untidy suitcase into which he stuffed all the dirty clothes he had spread around him, that unpleasant person and all the unpleasantness that went with him left the train about halfway to Jane's destination. After Jane had aired out the compartment, she pulled out her knitting, settled into a pleasant routine, and enjoyed the remainder of her trip. No murder today on the 5:36 from Paddington. No jumping out the window of a speeding train. What she needed was patience and the knowledge that she would soon be without her unpleasant companion because either he would leave first, or she would arrive at her station.

Spells of painful moods, depression, anxiety, boredom, loneliness, all are temporary states, much like the experience Jane had with the unpleasant man in her train compartment. We can help our patients during episodes of dysphoria by asking them to imagine they are on a train ride. Someone very unpleasant enters and creates an uncomfortable situation—no joke, it's unpleasant to have this jerk on board. But you have to remind yourself that the unpleasant, almost unbearable, person will be getting off the train at his stop, or "Jane," our patient, will be getting off the train at her stop. It's a temporary situation. Knowing that this "person," a metaphor for a painful state of mind, is only temporary, and really believing that fact, will help a patient practice "mindfully being with" that state of mood until it changes or goes away. The problem comes when a person tries to "murder" that mood state, to commit harm to self, becomes desperate about getting rid of the state of mind. These visitors to the compartments of our speeding lives enter without our consent from time to time. They are not pleasant. We validate the fact that they are unpleasant. But we teach our patients that we can tolerate them without taking desperate measures. In fact, we can go beyond "being with" these states of mind; we can become curious about them. "Now I wonder what caused that person to enter my compartment? What

makes him so messy and unpleasant? Maybe he's got no place to go. And I did see that he was writing a poem. It could be he's not always totally unpleasant. Maybe he could teach me something if I stopped fuming and asked him a few questions, like, Who are you? What brings you here? How long do you plan to ride on my train?"

Amid our struggles to separate ourselves from unpleasant "visitors," we can do things that are much worse than putting up with these visitors. What is most important to remember is that these unpleasant visitors will be leaving the train.

Now, it's time to pull out one of the old Agatha Christie novels, or one of the films made from her novels, and enjoy a good murder mystery about the 5:36 from Paddington Station. Murders on the trains in mystery novels are okay. Having to murder temporary mood states in our lives is not okay.

Ask the Dry Cleaner

Meg wanted expert advice for anything important in her life. Her cosmetician always selected the perfect hair color for her. She wouldn't think of changing the arrangement of her living room without guidance from her trusted interior designer, Susie, who had designed the homes of some of the city's most prominent families. Her bike came from a French firm that specialized in the titanium bicycles used in the Tour de France. Her yoga instructor guided each client with a personal fitness program.

But Meg had one area in which she was unsure of how to proceed—what should she weigh? Her gynecologist was working with her to figure out why her periods had stopped. Her physician, Dr. Ryan, thought she might be too thin. Meg thought, without saying so, that there was no way she was too thin. After her last visit with the doctor, Meg had a great idea—when she went to pick up her dry cleaning, she would ask Mrs. Lang if she was too thin. Mrs. Lang could remove any spot from any article of clothing. No stain was too tough.

The next day, Meg went to Mrs. Lang for her weekly laundry. "I was wondering," she asked the lovely Mrs. Lang, "do you think I am too thin?"

Meg, as usual during cool weather, was wearing a mock turtleneck under an angora sweater, topped by her faux leather coat. "No, dear, I don't think you are too thin. You look beautiful to me," said the dry cleaner. "Thank you very much, Mrs. Lang. I appreciate your expert opinion."

Meg felt reassured. Dr. Ryan was an expert in hormones, of course, but she knew he couldn't be right about the weight. Not after hearing Mrs. Lang's opinion. It was so important to get advice from the right people.

Later that afternoon, Meg saw her dermatologist for evaluation of some sunspots on her cheek. If the specialist said that dermabrasion was necessary, Meg would not hesitate to make a follow-up appointment. After all, she valued expertise.

The comment from her gynecologist that Meg might be too thin still bothered her a bit. To confirm her own opinion about weight, Meg asked the UPS delivery woman about her weight. Ms. Lopez was a wonderful delivery person. Every new order from Amazon was placed neatly by Meg's front door. Ms. Lopez was 7 months pregnant but did not want to stop the job she loved until the ninth month. "No dear," said Ms. Lopez, "you don't look too thin to me. I wish I looked like you, but that's the way it goes with pregnancy. You'll know some day."

Dr. Ryan insisted that Meg see a specialist at the hospital's eating disorder clinic. Meg did so because she didn't want to displease the doctor, but she knew it was a waste of time. At the clinic, after a thorough evaluation, they said to Meg: "We believe you meet criteria for an early diagnosis of anorexia nervosa. You have lost too much weight. You are too thin for your age and height." Meg hesitated for a moment and then replied, "You know, that is not possible. I have gotten two expert opinions that say I am not too thin. Thank you for your time, but I'll be leaving now."

Meg knew what she was doing and what she wanted. She lined up appointments with her interior designer, her colorist, her dermatologist, and several other experts. As she was making these appointments, she thought, "That's so silly, that idea from the eating disorder clinic. I know I am fine in my weight. After all, Mrs. Lang and the nice UPS delivery person told me I am not too thin." Meg always went for expert advice when she needed help.

How many times have we seen patients who would never go to their stylist or dry cleaner for advice about an illness such as diabetes but think that anyone they know or meet who thinks their weight is fine is acceptable as an expert? Anorexic thinness in young women is almost always ego-syntonic. The cognitive distortion of filtering is alive and well: any opinion that does not agree with their perceptual distortion that they are just fine is filtered out, whether that opinion is from a parent, physician, or psychologist.

Sometimes, when I share the opinion, based on evaluation, that a young woman has early anorexia nervosa, and she says others think she is fine, I might reply, "Well, let's ask the plumber about your hair color, and the bakery owner about your interior design. How about it?" The reply is, "No, of course not. That's silly. I want experts to advise me." *Normative cultural distress* is so common that a woman believes she is "normal" in weight only when she is more than 15% below the ideal body weight, not just the population average. We have to use motivational techniques to help women accept help about weight.

It's Hard to Say Goodbye

Kent and Barb were staring gloomily into their Diet Cokes. "I sure miss Ed," said Barb. "He was always there for us. When no one else understood, he did. When he was around, I knew what to do with my stress."

"Yeah, it's a bummer," said Kent. "When I was a fat, pimply junior in high school, Ed came to my rescue, too."

After a few more minutes of reminiscing, Alissa and Twyla stopped by. "Hey, what are you guys so down about?"

Once Kent and Barb explained it to the two new arrivals, Alissa and Twyla nodded in agreement. "It's sure hard to find loyal friends these days," said Alissa. "And then when you lose one, the world is a lot scarier. Like, I'm going to a college across country this fall, and I don't know how I'll handle it." Twyla chimed in: "Totally agree with you. I'm returning to professional dance school for the summer program. My treatment is over, but what will I do with all those mirrors?"

Brent joined the group. "OK, don't tell me. I know what you are talking about. You have been reading my mind. It's Ed. He's gone, left town, scooted, vamoosed. I'm so tempted to run after him, search the internet. I know if I asked him to come back, he would. After all, he's not dead, just gone away. Every once in a while, I imagine him telling me to remember to follow his plan, especially when Mom and Dad get on my case, which is most of the time."

Laurissa had finished her group therapy notes and was walking past the group. "Today was a graduation, not a wake, and here you all are looking like your best friend has passed. I'd like you to get back on track and remember the real Ed. Sure, Ed was always there—always there in a bad way, so you didn't have to deal with the agony and messiness of adolescence. Each of you were trapped by Ed's confident advice that if you lost enough weight, or bulked up enough muscle, everything in life would go your way. No, you are not losing a friend. You are losing a noose, a strangler vine that wouldn't let you keep growing emotionally. Yes, you felt good for the moment when you followed him, when you either starved yourself, or did a ginormous workout, or had that 4:30 afternoon binge.

"Remember, today's unhealthy solutions become tomorrow's problems, whether it's drugs, eating disorders, or self-cutting. Your emotions went into the deep freeze. Now you are having to deal with them—hooray for feeling emotions. Emotions are feelings, not commands. Remember the exercises we did to cultivate positive emotions. We meant not only to kick Ed in the butt, not only to pick weeds, but to grow roses.

"After a couple of weeks of vacation for me, we'll resume the group therapy for anyone who is in the area. Hey, dudes, I'm not scolding you. It's totally natural to miss someone who gave the illusion of help—it was a form of help, but very temporary, and with an enormous price tag. How about holding a passing-away service for Ed? Then we'll have a celebration for your new life, led by yourself, by your true feelings, by the knowledge you have worked so hard to obtain. It's normal to mourn for Ed, but only for a bit. It's normal to be a bit scared without his dictatorship. But you have yourself back on a healthy pattern of dealing with life,

you have each other, you have your new buddies. It's time to say goodbye. Love you guys."

Eating disorders (Ed) are problem solvers but even bigger problem givers. You would have to be a little bit nuts to say, "No, don't straighten my broken leg," or "Don't take out my inflamed appendix." But eating disorders are different, and that is one reason that parents, and the public in general, don't understand that they are not simply a voluntary habit. Once developed, eating disorders have a life of their own. They do actually make some feelings better for a while; they allow a person to avoid making some difficult decisions; they allow people to deflect hurt feelings from unkind comments; they give the illusion that there is an effective sense of control; that others will like them more. But then the price tag comes in: the developmental arrest, the medical complications, the self-isolation, the treadmill of relentless exercise.

Many, not all, of our patients go through a time when they have improved in their disordered eating behavior and are recognizing the true need of dealing with life's issues in healthy ways, but they are not yet solid. This is the time when it is so tempting to call Ed back into the picture.

Thermometers and Thermostats

"Oh, I am so sleek and thin. Sometimes, I have a silver center surrounded by glass like a glamorous piece of jewelry dangling from the ears of a celebrity. And you know what silver costs these days! Otherwise, my center is red, and people can't stop looking at me. Red never goes out of style—like brightening up a boring black cocktail dress with a red handbag and shoes, or Marilyn Monroe's lipstick. I am so fashionable, people check me out every day—a thermometer. They want me. They need me."

"I can't argue with you. You certainly are glamorous—long and thin. I'm pretty clunky myself, usually rectangular or square. Nothing fancy about being beige or dull gray. Lots of times, people try to hide me. I'm a wallflower compared to you. I may only get looked at a couple of times a year. Then, someone pushes a few buttons on me, goes away, and I continue to gather dust for another six months."

"I feel sorry for you, my clunky thermostat friend. Wish I could do something for you. Oops, hold on. My silver-colored center just shot up. The sun is shining brightly on the terrace, where fabulous people are having cocktails, and they all keep staring at me. Believe me, I get plenty of attention."

"For a thermostat, I'm feeling a little happier for a change. My owner, a bearded guy who eats lots of granola and rides his bike everywhere, came up to me the other day and changed a few of the dials. He said, 'You know, little thermostat, you saved me hundreds of bucks last winter.' I may not be glamorous, but I do important work."

"Well, you are still a clunky piece of metal that hardly ever gets noticed. You are not thin and sleek. You don't have a silver or red center that goes up and down. Oops, excuse me for a moment. I suddenly shrunk to this little itty-bitty thing, and I'm feeling very cold. Ouch, I fell! My silver center is running out! I'm being swept up and put in the trash! Help me! Help me! Help—"

And then there was silence.

Meanwhile, the thermostat continued to do its job day after day, changing the temperature cycle, switching from air conditioning to heat, turning the furnace on and off. Not very glamorous but much valued.

In an interview in the *New York Times*, Professor Cornel West (2011) is quoted as saying, "You've got to be a thermostat rather than a thermometer. A thermostat shapes the climate of opinion; a thermometer just reflects it." What a wonderful summary of the difference between thermometers and thermostats! I encourage patients to become thermostats, not thermometers. Most patients with anorexia nervosa act like thermometers: when the fashion climate, or their friends, or the movies say thinner is in, they follow the climate and slim. Hemlines up, hemlines down. And it's sad. So often, they look glamorous only when dressed up, but medically they are starved and bony. Of course, guys follow trends and can be thermometers too: "Bulk up those pecs; get that body fat down." In contrast, a person who is a thermostat sets the climate, doesn't follow it, or more accurately, doesn't follow it when it is unhealthy. The thermostat controls the temperature, while the thermometer simply follows the temperature set by the thermostat or the external environment.

When the daughter of a friend of mine was eating a second hot dog during lunch with her dance team comrades, one said, "Ellie, you're going to get fat. That's your second hot dog. I wouldn't eat even one hot dog." Ellie calmly went on eating her second hot dog and gently said, "You know, Liz, these hot dogs are surprisingly good. We work out hard and need to eat enough calories to maintain our strength for the dance team. I wonder if you might have an eating disorder, or the beginning of one. There's a friend of my dad who coordinates an eating disorder clinic. I'd be glad to give you his name. And, oh, I think I'll get that chocolate ice cream for dessert." Ellie was setting the climate, not following it.

When patients learn to be a thermostat, not a thermometer, they become empowered to examine cultural trends and then decide whether to follow them or not. More importantly, they let other people know (in a nonjudgmental way) what they think and why they do what they do. Kudos to Professor West for his summary of the difference between thermometers and thermostats. Goofy story, maybe. Important concept, definitely.

The Girl in the Yellow Dress

Carla was an experienced Marine Corps sergeant who had seen service in Iraq and Afghanistan on three tours of duty. She was invited to the eating disorder evening group at the local community center to tell her story, because the group had helped her in the past. What a commanding presence she was: outfitted in desert camouflage gear, with broad shoulders but feminine in physique, with a squared-away appearance that engendered instant respect. On planes, passengers wanted to shake her hand and thank her. This is what she said at the meeting: "Guys, it's war out there, and war is hell, but someone has to protect our country, even if you don't agree with every policy. I want to tell you a story that keeps me up at night to this day, and when I do sleep, I have nightmares. The VA is doing its best to help, and it's coming along, but slowly.

"One day, we were in a small town in Afghanistan, being friendly with the locals to show them that we were on the side of peace. As we drove slowly through town, making stops to hand out diapers and vitamins to

groups of young mothers and their children, we were on the lookout for IEDs—that's improvised explosive devices for you civvies. Toward the end of the town, I saw one of those damn IEDs on the side of the road—disguised as a tin can behind a clump of weeds. I knew my duty. I stopped the jeep and started to walk cautiously toward the IED to inspect it and see if we could either disable or move it, so it wouldn't do any harm. Can't believe what happened. A young girl, about 8 or 9, had been following our slowly moving jeep. She darted out ahead of me and picked up the IED. You know what happened. The blast shredded her body and threw me back against the jeep and concussed one of my buddies and tore a hole in the face of another. When I came to my senses, I half-crawled over to what remained of the girl—only a few pieces of her yellow dress and a couple of shredded flowers she was trying to give me remained.

"That's only one of the traumatic events that happened in Afghanistan, but the worst. I can't get it out of my mind. If only I had been faster in getting to it. If only I had seen her and warned her away. If only. But 'if onlys' don't count. This precious little girl was gone forever. I wake up at night in a sweat dreaming of the explosion and trying to warn the girl. Every time I hear a car backfire, I hit the concrete instinctively. I break down when I see little girls in yellow dresses. No, I'm not going back to my eating disorder, and this PTSD will eventually get better. But that's what war does to you. I am accepting another tour of duty, partly because I believe that women belong in the armed forces, in every role. Any thoughts?"

After a few moments of silence, Leslie stood up and spoke with tears in her eyes. "What a story. But you know, Carla, it's war over here, too. I was raped at a party by someone I thought was a friend. I can't get it out of my mind either. I get the sweats at night and go right down to the refrigerator for a binge to settle down. I know it's not healthy, but these nightmares terrorize me. Sometimes I don't even remember that I had a binge, except for the bulge in my stomach in the morning. And all those reminders during a regular day that trigger my memories—not only memories, but it's like being right back there saying, 'No, no . . . ,' but he doesn't stop. My heart is racing. It's hard to breathe. Even if a friendly guy in my class taps my shoulder to ask a question, it freaks me out. And then I want to binge."

"Yeah," said Kyle. "I saw my cousin shoot himself in the head playing with a pistol he found in his dad's drawer. He thought it was empty. I can't go to any movies now that have guns. And those sweats you are talking about. That's me. When the flashback comes, my heart feels like it's coming out of my chest, it's beating so strongly. I feel hot and cold at the same time. I think that's what got me started with dieting and body building, and now I can't quit."

And so it went. Marie was sexually assaulted in her own bedroom by a cousin while their parents visited in the living room. It happened every time the families visited. She just couldn't tell her parents. They would never believe it. She decided that if she became so thin and bony that no one would be attracted to her, she would be safe; but she never felt safe, except in this group.

Everyone gave Carla a big hug at the end of the group. They all realized they were in this thing together—bad things, really bad things, happen in Iraq and Afghanistan, but also in the good old USA, and in other places. The group members decided to be more open about what happened to each one of them in the past, and to text each other if they were having a flashback. War is hell wherever it happens.

Carla's story is a true story told to me by a patient. To this day, she can't forget the trauma and violence in Iraq and Afghanistan, but especially the tragedy of the little girl in the yellow dress. Whether trauma, sexual or other, is related to an increased chance of developing eating disorders is somewhat controversial. Now, after far too long, the armed services are getting honest about the high prevalence of PTSD in service personnel. At least one-third of patients with eating disorders whom I have treated have had traumatic experiences and then relive the experiences. These vary from bad memories to a daily reliving of the trauma, often triggered by innocuous stimuli. Most have night terrors as well. For some of these patients, binge eating, dieting, or extreme exercise has become their "solution." The methods of treating PTSD are still evolving and generally improving. But certainly, no history and examination of a patient with an eating disorder are complete without a detailed, compassionate, gentle inquiry into a history of trauma—and no, it should not be done at the first meeting, but only when trust has been

gained. Whether the trauma is directly related to the onset and maintenance of the eating disorder is not the point—identification and treatment of PTSD is a package deal in treating patients with eating disorders. There are no Lone Rangers when it comes to eating disorders—virtually all of them come as package deals, and these days, PTSD and trauma, whether overseas or stateside, are too often part of the picture.

Choosing and Prescribing Psychotherapy

Clinicians at times may be unsure about which method of psychotherapy is best for patients with eating disorders. Some clinicians may have training that emphasized only one method of therapy, such as evidence-based cognitive behavioral therapy or psychodynamic therapy. Teaching in medical schools and graduate schools even varies by geography and the philosophical commitments of teachers and leaders.

A sequence of psychotherapeutic methods may be more helpful than any single method. I have found that a sequence of methods, as described below, meets the needs of most patients with eating disorders when a clinician has a long-term relationship with the patient. Health plan limitations are a reality, but whenever possible, a progressive sequence of psychotherapeutic methods adapted to the severity of the eating disorder may best support the patient's return to psychological freedom from the eating disorder and ability to move on with the challenges of life.

Background

The history of psychiatry has been marked by tension between psychodynamic psychiatry and biological psychiatry. The former method has emphasized an understanding of the psychological meaning behind human behavior, whereas the latter has studied the biological substrate of normal and abnormal behavior and emphasized psychopharmacology. Psychodynamic theories and practice dominated psychiatric thinking in the United States until the 1960s, when more systematic methods of diagnosis, epidemiologic studies, and more effective methods of treatment began to cast doubt on some of the classic psychodynamic

assumptions. Categories of diagnosis and treatment trials, rather than intrapsychic process issues, came to the forefront. As a result, psychiatry tended to adopt an either/or way of thinking, which led to artificial polarities that asked such questions as, "Is the condition treatable through psychotherapy or should medications and other physical methods be used?"

Multiple studies have documented that several major changes have taken place in the field of psychiatry with respect to psychotherapy. Many psychiatric illnesses have been shown to respond best to a combination of psychological and psychopharmacologic methods, even though one modality may play the major role. More than one method of psychotherapy is now available, providing alternatives to the once-dominant technique of psychoanalytic psychotherapy. As a result, prescribing either a single primary evidence-based psychotherapeutic method or a sequence of psychotherapy methods to accommodate the patient's changing needs and capacity for psychotherapeutic work may prove most beneficial. Additionally, evidence supports the prescription of both a main method of psychotherapy and additional adjunctive psychotherapeutic modalities.

In place of the either/or polarized thinking about physical and psychological methods of treatment or a dogmatic insistence on one particular form of psychotherapy, the practicing psychiatrist now has the flexibility in prescribing psychotherapy much like the internist has in prescribing an antibiotic. Although it would be naive and premature to assume that psychotherapy prescription has the same empirical support as the rational prescription of an antibiotic, enough studies on psychotherapy research have been published to suggest the analogy is a useful one. Just as a physician must be able to answer certain questions before prescribing a medication, the psychiatrist should find answers to similar questions before beginning a course of psychotherapy. They must determine the goals to be attained with the method of treatment; review the evidence that the chosen method has a strong possibility of improving the specific condition of a particular patient; assess the risks involved; explore the available alternatives; and establish which aspect of the total condition psychotherapy can be expected to help. Finally, the psychiatrist must determine how psychotherapy can best be integrated

with psychopharmacology for conditions in which pharmacology or other physical methods have been demonstrated to be effective so that each modality can work most effectively in partnership.

Definition of Psychotherapy

Jerome D. Frank (1982), a widely respected student of comparative psychotherapy systems, defines psychotherapy as follows:

> Psychotherapy is a planned, emotionally charged, confiding interaction between a trained, socially sanctioned healer and a sufferer. During this interaction the healer seeks to relieve the sufferer's distress through symbolic communications, primarily words but also sometimes bodily activities. The healer may or may not involve the patient's relatives and others in the healing rituals. Psychotherapy often includes helping the patient to accept and endure suffering as an inevitable aspect of life that can be used as an opportunity for personal growth.

The two essential purposes of psychotherapy, in Frank's view, are to *restore morale* and *to help the patient solve specific life problems*. Beyond those basic purposes, several additional factors enter into the choice of a specific form of psychotherapy or a combination of psychotherapeutic methods, as noted later in this chapter.

FDA Tests: Safety and Efficacy

If psychotherapy is to be prescribed in a manner somewhat analogous to prescribing medication, it may be worthwhile to ask first whether psychotherapy meets the basic tests of the US Food and Drug Administration (FDA) for approving new medical treatments—safety and efficacy. Any treatment that has the potential for helping to heal has some potential for misuse. Freud himself said that the application of psychoanalysis to schizophrenia, for example, was a mistaken use of the art, but he went ahead and used psychoanalytic thinking to suggest an etiology for schizophrenia. Following the ancient medical dictum of *primum non nocere*—first, do no harm—psychotherapy, *applied properly*,

and following the ethical guidelines of the American Psychiatric Association, can be said to be a *safe*, although not entirely risk-free, procedure. The *efficacy* of psychotherapy has been a more controversial issue. The extreme views present in psychiatry suggest, on the one hand, that psychotherapy by a trained individual has no specific effectiveness beyond that which would be accomplished by any caring individual. The other extreme claims that psychotherapy is specifically curative for virtually any psychiatric disorder. The evidence suggests that psychotherapy has a definite but variable benefit according to the disorder, being much more effective in bulimia nervosa and nonpsychotic depression than in schizophrenia or dementia. Anorexia nervosa has been more difficult to study because patients are often hospitalized and suffer from severe medical complications, but short of strictly randomly assigned studies of psychotherapeutically treated versus not treated methods (probably unethical), the current preponderance of evidence supports the prescription of CBT, especially for anorexia nervosa.

The basic test of whether a hypothesis is a scientific statement or a philosophical statement is achieved by determining if the proposed method can be experimentally tested so that the hypothesis can be either refuted or confirmed (Kuhn, 1970). Some statements regarding psychotherapy can be tested empirically, but others remain more related to philosophical issues. The prescription of a particular form of psychotherapy should ideally develop out of testable forms of treatment. Recent experimental work in the *psychotherapies* (probably a better word than *psychotherapy* as a single generic term) have examined the therapeutic effectiveness of specific forms of psychotherapy for well-defined disorders, using as controls either patients who received no active treatment or those given other modes of treatment, such as medication. Using those procedures for testing, we can say that CBT, for example, appears to be as effective as tricyclic antidepressants or SSRIs in treating moderately depressed, nonpsychotic individuals (Simons et al., 1985). It is more effective than medication in treating bulimia nervosa. Monosymptomatic phobias, to use another example, respond routinely to systematic behavioral desensitization (Marks, 1969).

In place of the question "Does psychotherapy work?" (which would be about as ambiguous as the question "Do medicines work?"), practicing psychiatrists should be asked, "Does psychotherapeutic method A work for patients with disorder B, using operational definition C of practical application for improvement in their disorder?" This more scientific question not only fails to inhibit psychotherapy treatment but paves the way for progress in the field so that the psychiatrist can become increasingly more effective. A scientific mindset toward the traditional prescription of psychotherapy does not minimize the need for the art of psychotherapy or the need for sensitive relational skills.

Why has psychotherapy only recently been examined in more scientifically? For one thing, research in that area can be much more difficult than research conducted in the field of organic medical disease, where reductionist methods of biochemistry and neurophysiology have led to great advances. Some theoretical statements central to certain forms of psychotherapy are phrased in terms not testable by the scientific method. For example, two central tenets of psychoanalysis, the existence of the unconscious and the belief in psychic determinism (Brenner, 1955), are philosophical, not scientific, statements.

Second, therapists who sincerely and energetically treat individuals one by one in the outpatient setting, using traditional psychotherapy methods, are usually not trained in the experimental approach to hypothesis testing. In addition, they often feel that the improvement of an individual patient under their treatment validates their psychotherapeutic approach. Other therapists may feel that a scientific approach will inhibit the art of psychotherapy or take the place of empathetic interpersonal relationships. Finally, many training programs teach a particular form of psychotherapy in a dogmatic manner without examining its grounds for credibility or its ability relative to other forms of psychotherapeutic treatment to alleviate psychiatric disorders. This critique of unexamined teaching applies to both biologically based and psychotherapeutically rigid programs. The book *The Perspectives of Psychiatry* (McHugh and Slavney, 1983) describes how multiple methods of approaching mental life and human behavior, each with its own strengths and vulnerabilities,

can be integrated and used in the diagnosis and treatment of patients with psychiatric illness.

Psychotherapeutic models can and should be tested by appropriate evidence-seeking methods for multiple reasons. In this day of increasing scrutiny of treatment modalities by third-party payers and governmental sources, data are needed to show that the methods of treatment administered are indeed effective. Psychotherapy can be a long, expensive process and may appear to critical eyes to be an outmoded, high-cost treatment that can be replaced by more easily prescribed medications. Several evidence-based forms of psychotherapy have been demonstrated to be effective in a time-limited approach, typically 8–16 sessions. Resistance by psychiatrists to a more scientific approach toward psychotherapy may eventually lead to reduced levels of payment by medical insurance companies when effectiveness cannot be confirmed. The major reasons for a scientific approach to psychotherapy, however, are more fundamental: we want to know if what we are doing to help people is more effective than the mere passage of time or a general level of caring that could be given by a nontrained individual. We wish to continually improve in our practice, and that can be done only by using valid and reliable methods of scrutinizing our treatment approaches for evidence of effectiveness. We need to know that the methods we are using are indeed the best ones for a particular patient.

By using a more evidence-based approach to prescribing psychotherapy, there is no doubt that many psychiatric conditions that cause great psychological suffering and behavioral disorders can and have been shown to improve substantially with psychotherapy, even if the process is lengthy and costly. Few third-party payers would object to long-term treatment of diabetes or recurring cancer. In short, the needs of both psychiatrists and their patients will be better met by the adoption of a scientific approach to psychotherapy without sacrificing the utterly essential qualifications of human respect and empathy in the process of patient care. The distinguished psychiatrist and philosopher Karl Jaspers (1963) spoke of the need for psychiatrists to have a dual-orientation approach to the patient. On the one hand, the psychiatrist must rigorously take a history and do a full mental status examination so that a

clear, scientific definition of the psychopathology involved can be assessed. Only then can they enter into the second component of treatment: forming an empathic relationship with the patient, appreciating the nature of the existential suffering, and offering understanding, hope, and a specific means of improvement. A scientific effort at evaluation of the various forms of psychotherapy in no way reduces the field of psychiatric treatment to a simplistic method or robs it of the richness and complexity of understanding and relieving human suffering.

Prescribing a Method of Psychotherapy

The methods of psychotherapy can be summarized by describing for each method several essential facts: the key players, or those who have founded and researched the method; the basic idea underlying the method; the basic mode of application; the disorders for which each method has been shown to be suited; and some general comments regarding the method's employment (table 20.1). The use of a single method of psychotherapy based on unexamined teaching that may be decades old is generally no longer appropriate for many general psychiatrists. In its place, several evidence-based methods of psychotherapy, each having some shared as well as some unique features, need to be learned so the appropriate method can be chosen, or complementary and adjunctive therapies can be chosen according to the needs of the patient. If each proponent of each different kind of psychotherapy were mentioned, the list of individual names would quickly grow into hundreds. In practice, however, only a few major forms of psychotherapy are commonly chosen, and they can be categorized according to one of the following goals: to understand the nature of illness; to improve distressed moods; to change abnormal behaviors; to change painful, ineffective, or abnormal ways of thinking; to deal with existential issues of meaning and suffering; to improve dysfunctional family patterns; to make meaningful connections on the basis of insight and interpretation of transference; to deal with living in the here and now; to build a toolbox of lasting patterns of action and thinking that continue after therapy is concluded; and to emphasize supportive empathetic care.

TABLE 20.1. The methods of psychotherapy

Method	Key players	Central concept	Mode of application	Disorders/situations best treated	Comments
Behavioral therapy (BT)	Marks; Wolpe	Progressive desensitization along a hierarchy of increasing fears leads to less restricted, more normal behavior.	Teach method of relaxation; pair relaxation with a hierarchy of increasing fears until feared object, situation, or condition is tolerated.	Monosymptomatic phobias; panic attacks; inhibited sexual response; binge eating	Real-life (in vivo) or ideational (in vitro) methods both effective. Other examples include sensate focus therapy (sexual disorders) and response inhibition (bulimia nervosa).
Cognitive behavioral therapy (CBT)	Beck; Rush; Garner; Garfinkel; Fairburn	Painful moods, self-defeating behaviors, and abnormal eating behaviors result from overvalued or irrational beliefs (*grids*) and automatic thoughts.	Identify irrational mental *grids*; challenge and refute irrational beliefs by evidence gathering; practice new, appropriate and supportive ways of thinking and acting.	Mild to moderate nonpsychotic depression; binge eating disorder; bulimia nervosa; obsessive-compulsive disorders; probably anorexia nervosa	Often paired with other behavioral methods.
Dialectical behavioral therapy (DBT)	Linehan	Patterns of abnormal behaviors and thinking can be changed by increasing emotional and cognitive regulation.	Combines CBT with distress tolerance, acceptance, and mindfulness meditation.	Borderline personality disorder; suicidal ideation; substance abuse; mood disorders; some eating disorders	Reduces rate of rehospitalization and reduces suicidal gestures in year after treatment.
Existential psycho-therapy	Frankl; Yalom	Issues of meaning, values, identity, suffering, and finiteness are central to authentic living.	Clarify values and identity; deal with fears of nothingness and the meaning of painful experiences.	A concluding method in a sequence of psychotherapies; for existential issues rather than for severe major psychiatric disorders	Has roots in philosophy and religion—may have agnostic or theistic forms. Overlaps fields of philosophy and religion.
Family-based therapy	Minuchin; Halley	An individual patient's symptoms are often an expression of underlying family dysfunction.	Identify maladaptive systems of family functioning, inappropriate norms, undercommunication, and lack of generational boundaries. Establish healthy, age-appropriate functioning.	The younger the patient, the more likely family therapy is necessary and useful for anorexia nervosa; as adjunct in schizophrenia and mood disorders, behavioral disorders of children, and distressed family states	Multiple innovative techniques include reassignment of roles, "care lists," dramatization of family patterns, clarification of communications.

Interpersonal psychotherapy (IPT)	Klerman; Paykel; Sullivan; Weissman	Improvement in distressed relationships leads indirectly to improvement in the specific psychiatric disorder.	Time limited (12–16 sessions); uses practical diagrams of relationships; directive; generally does not focus on the disorder itself.	Ambulatory patients with bulimia nervosa; postpartum depression; distressed family functioning	Rated as moderately effective, slightly slower than CBT, for bulimia nervosa; interrupts one leg of triangle of mood-thinking-behavioral interaction.
Mindfulness	Kabat-Zinn	Mindfulness is a feature of human consciousness, which a person can increasingly become aware of in the here and now.	Teach nonjudgmental acceptance and awareness of ongoing emotions and physical and mental sensations.	Anxiety disorders; stress reduction; grief; pain; depression	Derived from the nonreligious component of Buddhism; has preventive value.
Psychodynamic methods	Adler; Freud; Jung; Kernberg; Klein; Kohut	Confusing or painful feelings, behaviors, and relationships are linked in a causal way with crucial developmental experiences of both an intrapsychic and interpersonal nature.	Free associations, dream work, and interpretations of the transference of relationship lead to insightful understanding of personal development, working through of painful feelings or dysfunctional ways of thinking and behaving, and developing new patterns of living.	Insightful, motivated, distressed individuals with internal conflicts, situational stresses, unsatisfying relationships; some personality disorders; complex eating disorders	Newer forms include transactional analysis, object relations, and self-psychology schools. Short-term dynamic therapy described by Sifneos.
Psychoeducation	Donley; Tomlinson; Anderson; Garner; Garfinkel	Education about the nature and course of illness empowers the patient to deal with disorders in a healthy way.	Provide nonjudgmental information about the illness to the patient and family; clarify misinformation. Aims to create alliance with the treatment team.	Essential for all disorders in the diagnostic and treatment phases; the first step, and intrinsic to, continuing treatment where additional forms of psychotherapy are appropriate.	Basic but often underused; creates trust when given in warm, supportive manner.
Reality therapy	Glasser	Holding individuals accountable for responsible behavior leads to more improvement than insight-oriented methods for these individuals.	Confront patients with irresponsible behaviors by limit setting and positive reinforcement of healthy behavior. Emphasis is on here and now, not insight.	Antisocial behavior; histrionic behavior	Best documented form of psychotherapy for antisocial behavior.
Supportive psychotherapy	Bloch	Acute traumatic events or chronic stresses lead to emotional distress and loss of or regression of preexisting defense mechanisms.	Give reassurance and explanation; offer specific suggestions; restore morale; change the environment where necessary.	Overwhelming life circumstances; severe personality disorders; adjunctive treatment for major mental illness	Reintegration of preexisting defenses is the goal, not working through and "sealing over," not "uncovering" or developing more mature defenses.

Note: See the Suggested Readings for publications supporting studies of these psychotherapeutic methods.

Many different psychotherapy methods share important features, such as developing a trusting interpersonal relationship, showing empathy, and providing timely responses or interpretations. Enough empirical work has been done, however, to suggest that—unless one's practice is limited to a specific type of patient—a sequential or concurrent combination of several psychotherapy techniques is probably best for most patients. It is crucial for the therapist, if concomitant methods are used, such as CBT and psychodynamic psychotherapy, to be aware of which method is at the forefront for each treatment session. The essential decisions regarding psychotherapy that need to be made include the following:

- Will psychotherapeutic methods be central or adjunctive for this disorder?
- What skills or experiences does the patient bring to the therapy situation that may make one method more appropriate than another? For example, a noninsightful patient with severe alexithymia may not benefit from an insight-oriented form of psychotherapy.
- Are there contraindications to a particular form of therapy? Any treatment that can help can also be used in a harmful manner. Intensive psychodynamic psychotherapy may be disorganizing rather than helpful for a patient with severe borderline personality disorder, for example.
- Does the practitioner have the skills necessary to implement the chosen form of psychotherapy? If not, how can the skills be learned?

Practicing mental health professionals may be prisoners of their own methods of training. There is a tendency to practice unreflectively the kind of training that one has worked hard to learn in the past, often accentuated by a sense of personal benefit and psychological understanding obtained by the therapist during the training period. Being a prisoner of one's method of learning, however, does not serve the mental health professional or the patient well. Openness to new evidence-based methods that leads to taking courses and workshops in additional evidence-based forms of psychotherapy is essential.

Psychotherapeutic Options

Some examples may be useful in illustrating the choice of a psychotherapy method for a specific disorder in a patient. Incidentally, the term *patient*, which derives from the word for "suffering," is used here rather than *client*. Although the term *client* has been advocated recently to designate the sufferer and represents a worthy effort to see the suffering individual as the initiator of the therapeutic services, we feel the term is less suitable to the historical role of the physician, psychologist, or other mental health professional as a caregiver.

Case Study 1

W.A. has suffered an acute myocardial infarction at age 49. He is doing well medically in the intensive care unit but is complaining of depression. He has a compulsive personality and was raised in a family characterized by angry striving rather than supportive interaction and reasonable goals. The consultation-liaison psychiatrist identifies the need for some control on the patient's part during treatment and asks the medical staff to allow W.A. to independently take a single small dose of an antianxiety agent from a bedside single-dose supply when he feels very anxious. He is given control of time-limited small IV doses of morphine when he has cardiac pain. He is also given precise and comforting psychoeducation concerning the mild nature of his infarction, the absence of need for myocardial revascularization, and the probability of a return to work and marital relations in 6 weeks. W.A. is given nonblaming information regarding his type A driven personality and a specific referral to a mindfulness group following discharge. His therapy has been designed to be supportive, informative, and directive, attempting to integrate his existing defenses and suggesting nonblaming ways to diminish his compulsive lifestyle, which may predispose him to increased risk for future cardiac symptoms. He is taught a method of relaxation that he finds lowers his blood pressure.

Case Study 2

K.B. suffers from panic disorder, which has made her fearful of continuing her volunteer work in the community. Fearing new

attacks, she has begun to experience anticipatory anxiety, and she has also developed secondary depressive symptoms. The outpatient psychiatrist she has chosen prescribes behavioral desensitization integrated with cognitive behavioral methods of psychotherapy to alleviate the panic attacks and to diminish the resulting anticipatory anxiety and avoidance behavior. Using educational material by Claire Weeks (1969), the psychiatrist explains to the patient a plan to engage a psychotherapy assistant in the beginning of treatment to help K.B. walk through situations like those that triggered panic attacks in the past, such as a movie theater, driving over a long bridge, and shopping in a crowded department store. K.B. learns to become an observer of the panic attack, watching it crescendo and then diminish, experiencing lower and lower peak anxiety with each successful "walk through" of a panic attack. The patient's beliefs that she is dying or going crazy are identified and cognitively refuted using evidence for and against her cognitive distortion. A support group is recommended.

Case Study 3

H.C. has grown up in a family marked by three generations of alcoholism and has been the object of both physical and verbal abuse. She has developed a self-defeating pattern of trying to please everyone around her. Through 2 years of twice-weekly dynamic psychotherapy, she has effectively reconstructed her developmental pattern whereby she grew up switching generational roles to become a parent to her parent, anticipating her father's violent rages, having learned to say and do whatever was necessary to inhibit his rages. Her anger toward her ineffective mother, who has enabled her father's alcoholism, becomes apparent to her for the first time as she learns not to be frightened of angry feelings. She learns not to take responsibility for her parents' behaviors or to blame herself for their behaviors; she becomes appropriately assertive. Through dynamic therapy, she frees herself of her sense of fatalism about following the path of her mother by being a woman who is destined to choose an alcoholic husband.

Case Study 4

L.D. is a 16-year-old antisocial young man who had repeatedly been brought before the courts for several minor offenses, including stealing. More recently, his offenses have become more serious and include armed robbery and cocaine abuse. Through his family's efforts, he has been placed in a closed unit for disordered adolescents. In that setting, he attempts to blame his parents and his environment for his behavior. His psychotherapist, prescribing reality therapy (Glasser, 1965), asks him to focus on his own irresponsible and illegal behavior—not accepting excuses for it. He is confronted with his lack of remorse, with his law breaking, and with a probable future involving long jail sentences. Only after limits on his behavior are consistently enforced for several months on a closed unit does he develop some depression and—for the first time—a sense of personal suffering and a bond with his therapist. He is granted short supervised passes with specific tasks to accomplish only when his behavior on the unit becomes responsible. He is told to "cut the B.S." when he tries to blame his parents for his behavior. He is eventually promoted to the position of chair of the daily unit group meetings and begins to take remedial school courses to obtain his GED.

Case Study 5

N.E. is a 24-year-old woman with paranoid schizophrenia who has suffered many relapses despite adequate doses of neuroleptic medication. Her family lives in an intense emotional atmosphere characterized by hostility and unclear communications. Through family therapy as an adjunct to her medication, the family's emotional tone becomes less intense, more calm, and more appropriate, and individual family members learn to express their feelings through words. Because of the interest shown toward them and their struggle with the chronic illness of their daughter, the parents begin to feel less overwhelmed and are able to quietly discuss issues, lowering the tense atmosphere in the family. In the year following family therapy, the patient greatly reduces the number of repeat hospital admissions

she needs, and her haloperidol (Haldol) dose is lowered to half the previous dose.

One Method, Adjunctive Methods, Sequencing Methods

Not only can one particular form of psychotherapy be more appropriate for a particular disorder in a given individual, but a primary, as well as a complementary/adjunctive, method may be indicated, for example, adjunctive dialectical behavioral therapy for an adolescent whose entire family is taking part in family therapy. Additionally, a sequence of psychotherapy methods may be the most useful prescription for a patient's needs. The following are some examples.

Case Study 6

G.A, a 19-year-old woman with a 7-year history of anorexia nervosa, binge-purge subtype, enters a hospital for treatment of her eating disorder. Because her weight is very low, her mental concentration is poor, and she feels cold, the initial prescription is for supportive and psychoeducational psychotherapy aimed at restoring her morale and instilling a sense of hope for the future along with knowledge about her illness. Psychotherapy is given in short daily sessions for the first 2 weeks. Once medical stabilization has taken place and nutritional rehabilitation is under way, an 8-week program in CBT is begun. Emphasis in psychotherapy shifts to cognitive behavioral work, identifying the patient's many irrational beliefs about the overvaluation of thinness and her fear that normal eating will make her fat. She learns to challenge and refute the abnormal patterns of thinking, trying for the first time to see the environment as it really is, and showing a willingness to accept praise when given rather than listening only to criticism. When she is transitioned to partial psychiatric hospital (day program) after attaining 85% of healthy body weight, and showing some skill in beginning CBT methods, the emphasis on CBT continues more intensively. For the first 6 months of outpatient psychotherapy, CBT is continued, with the emphasis gradually shifting from CBT alone to a beginning focus on psychody-

namic psychotherapy, helping the patient recognize the connections between her own life experiences and the function her eating disorder serves. She recognizes that becoming extremely thin when she was 13 was a way to keep her parents from separating. It also allowed her to gain attention that had been disproportionately given to her extroverted older sister. Later, when her parents' marital relationship improved and that purpose was no longer necessary to keeping her eating disorder, she began to recognize that her thinness additionally allowed her to avoid her fears of sexual development and possible advances from young men. She comes to understand that her eating disorder is also based on a phobic avoidance of developing a personal identity. She identifies her early childhood lack of internalization of a stable, nurturing object due to the impoverished relationship with her mother, who was frequently hospitalized for mental illness.

During the last 6 months of an 18-month outpatient psychotherapy experience, the emphasis gradually shifts to existential psychotherapy, helping G.A. make sense of her suffering, but with a return to either CBT reinforcement or a dynamic interpretation of the transference relationship when either is appropriate. The patient is then able to express her rage at years of wasted time and to reexamine her inherited family values, no longer afraid to change what she believes in. She develops a commitment to help others to avoid her experience of an eating disorder and to live each day to the fullest without regret for the past or fear of the future. She finds the story of Viktor Frankl (1962) helpful in allowing her to get beyond her self-imprisonment of anorexia nervosa.

Case Study 7

I.C., a 35-year-old physician on the faculty of a university, presents with bipolar II mood disorder and muscle dysmorphic disorder, sometimes called *reverse anorexia nervosa*. During the early phase of mood stabilization with lithium and an antidepressant, the psychotherapeutic emphasis is on supportive and psychoeducational psychotherapy to give him hope for eventual improvement beyond each mood shift and to understand both of his disorders, while respecting

his intellect and desire for knowledge of research studies. Through cognitive and dynamic therapy, he stops feeling guilty and self-blaming during depressive episodes, which have been accompanied by thoughts of suicide; he begins to be able to say, "It, the illness, is here again," not "I am my illness." He can say with conviction, "I feel sad, but I know that I am a good person." He begins to identify and change triggers of his mood shifts, including working late at night in the laboratory, continuing verbal fights on holidays with his parents, and feelings of inadequacy in dating only women who are needy. During the CBT phase of his therapy, he is able to change his overvalued belief in the necessity of large muscularity and very low body fat. He agrees to cover the mirrors in his house except for a small section to let him see to brush his hair and teeth, as a way to stop his mirror gazing and self-criticism based on thinking he is puny. Through psychodynamic psychotherapy, he recognizes his anger at his mother's psychological unavailability during his youth because of her mood disorder, which made her frequently ill and in need of him to act as a supportive parent. He understands why he has feared women who are like his aunt, who smothered him with unending hugs. Through his psychodynamic and existential phases of psychotherapy, he attempts to moderate his overvalued ambition of becoming board certified in three specialties to prove his worth to himself; he becomes less concerned about international fame for his research and starts placing greater value on developing caring relationships with women who are not needy or predatory. He finds that daily prescribed exercise helps regulate his mood. He gradually comes to view himself as fit and attractive, not tiny and unattractive.

Choosing a method of psychotherapy is an important, and can be a rational, decision in patient care. Sufficient research is now available to choose a particular form of psychotherapy, a duality of methods, or a sequence of methods that are appropriate for a given patient. The psychiatrist's psychotherapeutic techniques need to be examined and updated when the methods used are overly restrictive or inadequate for the patient's needs. By keeping in mind the general guidelines of Dr. Frank

(1982)—psychotherapy always has two main purposes: restoring morale and helping to solve problems—the clinician's choice of psychotherapy technique or techniques may determine the effectiveness of the intervention. Transcending all the methods of psychotherapy is respect for the patient as a unique individual, empathy for their suffering, and an attempt to match the patient's particular disorder with the most effective therapy available. At times, psychotherapy will provide the major, or sole, emphasis in treatment, but at other times, it serves a vital adjunctive role with medical interventions.

For eating disorders, evidence-based psychotherapy, especially CBT, but also IPT and DBT, are essential. Further, especially for eating disorders, psychotherapy is for many patients best prescribed in sequentially. There is no psychiatric disorder for which psychotherapy is not an essential part of comprehensive treatment, whether the major component, an equal component with medications, or a vital smaller component. Any psychiatric disorder, whether resulting from stressful events, from a vulnerable temperament, or from distressed relationships, or when it springs from well-documented genetic or biochemical predisposition, always creates human suffering (even an elated manic or hypomanic episode takes its toll), dysfunctional patterns of thinking and behaving, and eventually an altered sense of personal identity and worth. Choosing effective methods of psychotherapy or a sequence of psychotherapeutic methods based on the most scientific studies available and following the guiding principles of ethical treatment will allow psychiatry to continue to deal with the broad range of mental disorders that affect human beings.

New Additions to Psychotherapy Methods

In the half decade of 2016–2021, several helpful and well-documented additions to the field of psychotherapy for patients with eating disorders have been published. First, some definitions are in order. *Third-wave behavioral therapies* (Linardon, Fairburn, et al., 2017; Linardon, Gleeson, et al., 2019) refer to a cluster of new methods that recently have shown evidence of effectiveness in patients with eating disorders, including mindfulness-based interventions (MBI), compassion-focused

therapy (CFT), acceptance and commitment therapy (ACT), and schema therapy (ST).

As more understanding about some of the underlying psychopathological mechanisms supporting the eating disorder have been elucidated, an increased focus on the specificity of the psychotherapeutic methods has resulted. For example, awareness of the common emotional dysregulation features across the eating pathology in all categories of eating disorders (Prefit, Cândea, and Szentagotai-Tătar, 2019) has led to the inclusion of transdiagnostic methods for the resolution of alexithymia, improved distress tolerance, skills training in emotional regulation, and growth of positive emotions.

Adjunctive therapy methods have been increasingly adopted as a complement to, and an augmentation of, other longer-established methods of psychotherapy, based on a more tailored and individualized approach to specific patients. A demonstrated adjunctive therapy for family-based therapy (FBT) is dialectical behavioral therapy (DBT) skills for the individual patient within the family (Peterson, Van Diest, et al., 2020). Mindfulness as an adjunctive therapy combined with other primary methods of psychotherapy has been demonstrated to be a valuable component of healing (Sala et al., 2020; Juarascio et al., 2021). Mindfulness is recognized to be a lifelong practice of centering, calming, and clarification, and in addition, it has the benefit of prevention of eating disorders in students with predisposing eating behaviors (Soriano-Ayala et al., 2020). Mindfulness and mindful eating have the potential to address problematic eating behaviors by an increased awareness of internal, rather than external, cues to eat (Warren, Smith, and Ashwell, 2017).

The validity of psychotherapy for patients with eating disorders has been increasingly demonstrated to be effective in real-world settings, an essential confirmation that the benefits initially demonstrated in structured research settings can be more widely applied with confidence (Brown et al., 2020; Mountford et al., 2021).

Psychotherapy for more recently recognized eating disorders, such as ARFID, has now been demonstrated. Thomas et al. (2020) treated 20 patients with CBT for adolescents over 20–30 sessions in a family-based or individual format, resulting in 85% of patients being much im-

proved or very much improved. Symptoms such as underweight, pica, and rumination all improved.

Internet-based methods of administering psychotherapy have been increasingly validated, not only as less expensive methods and for those taking prudent precautions in the COVID-19 pandemic, but also for those out of geographic reach, for individuals with work or family commitments incompatible with in-person treatment, and for extended follow-up (Munsch et al., 2019; Levinson et al., 2020). Additionally, studies are under way to demonstrate that internet-based methods are effective for preventing eating disorders in adolescents, the core group of the age range most vulnerable to developing such disorders (Zeller et al., 2020). Either by guidance in person, or by internet, self-help methods, for example, DBT, are growing in availability (Kenny, Carter, and Safer, 2020).

The period of 2016–2021 has empirically validated the benefits and advantages of family-based therapy for most adolescents with bulimia nervosa and anorexia nervosa with a BMI not requiring hospitalization (Gorell, Loeb, and Le Grange, 2019). FBT has been demonstrated to be an effective treatment strategy for anorexia nervosa in adolescents in the Asian context, widening its applicability (Wong, Goh, and Ramachandran, 2019).

An appealing and a previously often neglected perspective in the treatment of eating disorders has been to solicit patient input. Peterson, Becker, et al. (2016) have emphasized the benefits for developing the most effective treatment of patients with eating disorders by integrating research and clinical studies with patient perspective. Nilsen et al. (2020) sagely note that the young patient's subjective perspective of the recovery process can potentially strengthen therapeutic relationships and facilitate change.

Finally, even established and empirically validated methods, such as cognitive behavioral therapy, continue to evolve and increase in their effectiveness. An expanded form of CBT, CBT-E, represents a transdiagnostic treatment for all categories of eating disorders. It has been validated as a primary treatment for eating disorders by the National Institute for Health Care and Excellence (NICE) in the United Kingdom. Atwood and Friedman (2020) state that there is evidence to support

CBT-E as an efficacious and effective treatment for adults and older adolescents with a range of eating disorder diagnoses.

Summary

Some of the factors entering into a choice and prescription of psychotherapy methods include eating disorder subtype by *DSM-5*, medical severity, age (childhood, adolescence, young adult, older adult), sex and gender, sexual orientation, minority status, family involvement, comorbid psychiatric diagnoses, work demands, distance from in-person treatment, financial limitations, patient preference for in-person versus internet-based interventions, patient psychological mindedness, and provider training.

The treatment of eating disorders most often involves a combination of medical and psychotherapeutic methods. For the very ill patient, such as an individual with anorexia nervosa with a BMI of 10–12, the initial treatment is primarily medical for several weeks. Instilling a sense of hope and forming a relationship may be all the patient is capable of during this early phase. For a patient struggling with bulimia nervosa who is at normal weight and has normal electrolytes and normal findings on medical evaluation, the treatment may be primarily psychotherapeutic from the beginning of treatment and throughout. Many patients will need a combination of medical and psychotherapeutic interventions, with a changing ratio of these two components as treatment progresses. Psychoeducation from the beginning of treatment, even for highly medically compromised patients, prepares the way for more specific, individualized psychotherapy methods that can address the cognitive distortions, abnormal eating behaviors, emotional dysregulation, interpersonal dysfunction, and comorbid psychopathology. Once a patient is medically stable (if initially treated in the intensive care unit) and can tolerate more specific psychotherapy, the practice of writing a letter to the illness as friend and foe begins to establish a psychotherapeutic interaction based on understanding the individualizing features of these letters as they are reviewed together. The clinician can then plan a single method, a combination of methods, or a sequence of individualized psychotherapeutic methods, based on both the eating disorder di-

agnosis and the comorbid diagnoses. For example, for a patient diagnosed with bulimia nervosa who also meets the criteria for borderline personality disorder with past self-harm attempts, in the second phase of the sequence—evidence-based manualized psychotherapy—the clinician may choose a component of dialectical behavioral therapy in addition to cognitive behavioral therapy, although these two methods are increasingly being blended. For younger patients especially, and in groups, stories often communicate concepts that more technical terms do not. Beyond words, however, stories offer visualization and employ different parts of the brain, utilizing the strengths of even very ill patients.

The following guidelines apply to the treatment of a patient with an eating disorder by any single method of psychotherapy, or a combination or methods, adjunctive or sequential:

1. All patients require a quantitative assessment of psychological symptoms at the beginning of treatment, periodically during treatment, and at the conclusion of treatment, for their eating disorder symptomatology, as well as for any significant comorbidities that are integral to the treatment plan, for example, depressive symptoms, significant anxiety disorders, borderline personality disorder, substance abuse, PTSD, and sexual and other traumas. The Eating Disorders Examination Questionnaire (EDE-Q) is a full examination, appropriate to the beginning and conclusion of therapy. I have used the Eating Attitudes Test (EAT) and the Eating Disorders Inventory (EDI) for many years for briefer assessment. The PHQ-9 and GAD-7 for depressive and anxiety symptoms are widely available for the frequent, almost invariable depressive and anxiety comorbidities. Each comorbid diagnosis will have an easily administered and scored method available in the literature. A quantitative rating scale that is not onerous (less than 5 minutes to take and score) will benefit the therapist and the patient as well as demonstrating symptom severity and progress over the course of treatment.
2. A formulation of a few paragraphs after the initial meeting with a patient will include the rationale for the choice of psychotherapy

methods as well as summarizing features of the patient's history and mental status examination.

3. Any patient with medical symptoms will generally require the involvement of a physician for initial medical evaluation and intermittent contact.
4. The patient is a partner in decision making whenever possible and reasonable, even if decision making appears to be complex. Self-efficacy for the patient begins with joint decision making at the beginning of therapy from an understanding of the methods and the goals of the psychotherapy.

SUGGESTED CLASSIC READINGS

The following additional illustrative publications describe studies of the psychotherapeutic methods listed in table 20.1, with an emphasis on practical application (including workbooks). The emphasis in this list is on seminal original publications.

GENERAL OVERVIEW OF PSYCHOTHERAPIES

Gabbard, Glen O., Judith S. Beck, and Jeremy Holmes. *Oxford Textbook of Psychotherapy*. Oxford: Oxford University Press, 2007.

BEHAVIORAL THERAPY

O'Leary, K. Daniel, and G. Terence Wilson. *Behavior Therapy: Application and Outcome*. Englewood Cliffs, NJ: Prentice-Hall, 1975.

COGNITIVE BEHAVIORAL THERAPY

Fairburn, Christopher G, Zafra Cooper, and Roz Shafran. Cognitive behaviour therapy for eating disorders: A "transdiagnostic" theory and treatment. *Behavior Research and Therapy* 41, no. 5 (2003): 509–28.

DEPRESSION

Greenberger, Dennis, and Christine Padesky. *Mind Over Mood*. New York: Guilford Press, 1995.

DIALECTICAL BEHAVIORAL THERAPY

Linehan, Marsha M. *Cognitive Behavioral Treatment of Borderline Personality Disorder*. New York: Guilford Press, 1993.
Linehan, Marsha M. *DBT Skills Training Manual*, 2nd ed. New York: Guilford Press, 2014.

EATING DISORDERS

Grilo, Carlos M., and James E. Mitchell, eds. *The Treatment of Eating Disorders: A Clinical Handbook*. New York: Guilford Press. 2010.

EXISTENTIAL PSYCHOTHERAPY

Yalom, Irvin D. *Existential Psychotherapy*. New York: Basic Books, 1980.

FAMILY THERAPY

Madanes, Cloé, Alan S. Gurman, and David P. Kniskern, eds. *Handbook of Family Therapy*, vol. 2. Philadelphia: Brunner/Mazel, 1991.

INTERPERSONAL PSYCHOTHERAPY

Weissman, Myrna, John Markowitz, and Gerald Klerman. *Comprehensive Guide to Interpersonal Psychotherapy*. New York: Basic Books, 2000.

MINDFULNESS

Kabat-Zinn, Jon. *Full Catastrophe Living*. New York: Bantam Doubleday/Dell, 1990.

PSYCHODYNAMIC PSYCHOTHERAPY

Gabbard, Glen O. *Long-Term Psychodynamic Psychotherapy*. Arlington, VA: American Psychiatric Association, 2010.

PSYCHOEDUCATION

Garner, David. Psychoeducational principles in treatment. In: David Garner and Paul Garfinkel, eds. *Handbook of Treatment for Eating Disorders*. New York: Guilford Press, 1997, pp. 145–73.

REALITY THERAPY

Glasser, William. *Stations of the Mind: New Directions for Reality Therapy*. New York: Harper and Row, 1981.

SUPPORTIVE PSYCHOTHERAPY

Winston, Arnold, Richard N. Rosenthal, and Henry Pinsker. *Introduction to Supportive Psychotherapy*. Washington, DC: American Psychiatric Publishing, 2004.

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21

Family-Based Treatment for Eating Disorders

Sasha Gorrell, PhD, and Daniel Le Grange, PhD, FAED

COMMON QUESTIONS

How is family-based treatment (FBT) for eating disorders defined?
How are caregivers involved in eating disorder treatment?
How is FBT distinct as a model of eating disorder treatment?
What principles guide FBT in treating youth and adolescents with eating disorders?
Is there anyone for whom FBT might be less effective?
How might I orient my patients and their families to FBT?

Case Study

M.G. is a 16-year-old male with a diagnosis of anorexia nervosa. After participating in family-based treatment for three weeks, he has not met his weekly weight gain targets and continues to exhibit eating disorder symptoms, particularly food refusal. In the third FBT session, the therapist expresses concern about his struggles and his resulting lack of weight gain. M.G.'s parents acknowledge that treatment has thus far been very challenging and admit that they feel they lack the skills necessary to increase his nutrition. The therapist asks the parents to identify their primary barriers to refeeding. They report that mealtimes with M.G. are tense, anxiety provoking (for the whole family), and often result in more than one family member

yelling, with M.G. throwing food and dishes. The therapist helps the parents to identify ways that they can respond in those moments to remain calm and stay on task in the face of M.G.'s distress.

The therapist also invites M.G.'s parents to think through how they might set limits for him around not completing meals. With in-session problem solving, the parents identify several tangible strategies to address these behaviors, including providing the therapist with a detailed account of exactly what food (i.e., types and quantities) they prepare and serve to M.G. With a detailed report, it is evident to the therapist that M.G.'s daily caloric value is insufficient to promote weight gain in his underweight condition. Accordingly, the therapist carefully reviews each meal record from the prior few days with the parents and encourages them to provide suggestions as to how they might increase the density of each meal and snack. In so doing, the therapist helps the parents to focus on an approach that is specific to their own family culture and in keeping with what they used to prepare for M.G. (and his siblings) before the onset of the eating disorder. The therapist underscores that this initial strategy (i.e., parents deciding on and preparing all meals) should be followed daily, without exception.

Given the obvious struggle that M.G. experiences in meal completion, the therapist also helps the parents problem solve about how to encourage their son to complete what he is presented with. She specifically emphasizes that all decisions are to be entrusted to M.G.'s parents for now, and that it would not be helpful if they were to engage in any debate with him over details about refeeding, as any argument with "the eating disorder" is typically lost. The therapist also encourages them to work out a schedule to provide supervision, determining who would be sitting with M.G. at each meal. His parents are encouraged to communicate deliberately and clearly to him that (1) no discussions were to be had around types or amounts of food, (2) he is expected to finish what is on his plate, and (3) one or both parents will sit with him and provide support until he is able to finish all his food. In addition, his parents should clearly communicate to him that he will not be participating in afterschool sports or

activity other than essential daily tasks, as his team of providers had not cleared him for athletics, and any extra movement would simply make his much-needed weight gain harder to achieve.

M.G.'s parents are also encouraged to identify specific strategies that will help them to support one another in this endeavor and sustain a unified fight against the eating disorder, such as trading off shopping, preparing food, and supervising meals. The therapist also reminds the parents that, while M.G. is struggling with anorexia nervosa, he is still the son they have been able to feed since he was born; they have the skills and the knowledge necessary to help him but may temporarily need to put more structure in place to support the weight restoration process.

Background

Restrictive eating disorders (EDs) such as anorexia nervosa (AN) and bulimia nervosa (BN) commonly begin in adolescence and young adulthood, often resulting in a chronic course of illness. Further, there are no psychosocial or pharmacological treatments for adult AN, and more people die from this disorder than almost any other psychiatric condition. Given consistent evidence that early intervention results in the most promising treatment outcomes, ED treatment efforts during youth and adolescence are critical. While still much less researched than in adult populations, historical record of adolescent ED treatment over the last half century principally supports family therapy. Current published clinical guidelines recommend ED-specific family therapy as the first-line treatment for adolescents with AN and as a recommended treatment for adolescents with BN (National Institute for Health and Care Excellence, 2017). This chapter provides an overview of family-based treatment for AN and BN, including its structure and guiding principles. The text will also highlight patient and family characteristics that may affect clinical outcomes when using an FBT approach, as well as guidance and rationale in optimally orienting patients and their families to the FBT intervention model.

Caregiver Involvement in Eating Disorder Treatment

Historically, parents were mistakenly conceptualized as a causal factor in the pathogenesis of an ED, and as such, they were deliberately denied participation in their child's treatment. Over time, a theoretical and evidence-based shift away from an emphasis on parental responsibility in ED etiology has occurred, allowing families to now more often be actively involved in treatment, as indicated. In fact, the family (particularly the parents) can be viewed as the most influential resource in aiding a young person in the process of recovery. An understanding of the relational context within which the ED has developed also allows for an examination of how the disorder has negatively affected all members of the family, as well as the ways in which the family structure may exacerbate and maintain the ED.

A historically significant step forward in support of a family-based approach was the publication of Lock and Le Grange's treatment manual of FBT for adolescent AN in 2001 (Lock et al., 2001), now in its second edition, and for adolescent BN (Le Grange and Lock, 2009). Over the past two decades, FBT has been systematically studied in eight randomized clinical trials, six of which have focused on AN, yielding a relatively strong evidence base supporting the use of manualized FBT for adolescents with this illness. While less research has focused on BN, existing trials have provided provisional yet robust support for the use of FBT in the treatment of adolescent BN. In sum, involvement of families in interventions for both AN and BN with an FBT approach has demonstrated consistent and positive clinical outcomes, including significant reductions in psychological and medical morbidity as well as in treatment attrition rates (Lock and Le Grange, 2019). While preliminary, evidence from trials in samples of youth with pediatric obesity and avoidant/restrictive food intake disorder (ARFID) also demonstrates the potential benefit from the use of an FBT approach.

Structure of FBT

Along with the overarching principle that parents are a primary resource in their offspring's recovery, FBT is characterized by agnosticism,

whereby the family is encouraged to take a stance that refuses to take on the question of etiology or cause of the ED (table 21.1). This premise reduces blame for the current crisis on both the child as well as their parents and helps galvanize all family members around their shared treatment goals. FBT is most commonly an outpatient model of psychotherapeutic intervention and is most effectively implemented with the collaborative support of pediatric or adolescent medicine providers, as well as a registered dietician. Within this team scaffold, FBT is structured in three phases, with corresponding shifts in focus and parental and patient responsibility as treatment progresses.

In the initial stages of treatment (Phase 1), FBT mobilizes parental resources specifically in weight restoration, as well as in disrupting the cycle of any ED behaviors. The importance of an immediate focus on early weight progress is supported by consistent evidence that achieving approximately 2.5 kilograms (5.5 pounds) of weight gain by the end

TABLE 21.1. Selected principles of FBT

Principle	Description
Agnosticism	The patient and family are encouraged to join in a stance that refuses to take on the question of the cause of the ED. Working together with an agnostic stance can interrupt inaction that derives from guilt and instead facilitate parental self-efficacy.
Parental empowerment	The therapist takes an active role in guiding the family through the recovery process, but parents retain expert knowledge of their child. The therapist does not tell parents how to help their child recover and instead joins the parents in helping them to build self-efficacy and to problem solve the best ways to proceed through treatment.
Early focus on weight restoration and symptom reduction	In an effort to reduce long-term damage secondary to malnutrition, the treatment team must encourage parents to prioritize weight gain and to interrupt patterns of restriction and other ED symptoms.
Externalization	This principle effectively separates the illness from the adolescent, enabling parents to distinguish normal developmental autonomy (e.g., asserting independence) from ED cognitions and behaviors. Parents may then more effectively join with their child in working together to fight the ED.

of the first month of manualized FBT is a robust predictor of positive treatment outcomes (Lock and Le Grange, 2019).

In addition to restoring adequate nutrition and weight, treatment is initially focused on buttressing parents to mobilize their strengths and resources in subverting the cycle of their child's ED symptoms. FBT therapists serve as expert consultants, supporting parents to wholly assume responsibility for guiding their child in the process of recovery. In so doing, parents are encouraged to exert parental authority in ensuring that all contributing behavioral factors (e.g., dietary restriction, compensatory exercise) are minimized. The therapist both directs parental instincts and skills toward the ED target and frees them from the concern that their efforts will erode relationships within the family or exacerbate the illness.

As weight restoration is facilitated and behavioral symptoms remit, less parental authority is typically required, and parents may gradually restore autonomy over eating to the adolescent. In this stage of treatment (Phase 2), the therapist guides the parents in deciding when and in what manner they will allow the child to take increasing responsibility in choosing and preparing foods, and in eating meals and engaging in physical activity with little or no supervision. In time, as the child returns to the full range of age-appropriate activities (Phase 3), the therapeutic focus can shift to issues related to typical adolescent development (e.g., challenges that are academic or social) that were interrupted by the onset and course of the ED.

Factors that May Affect FBT Outcomes

An FBT approach may not be indicated for some persons, such as families who prefer individual treatment or an adolescent who is unwilling to involve caretakers. For most children and adolescents who struggle with an ED, however, involvement of the family in treatment is recommended and associated with better prognosis (Le Grange and Locke, 2009). For those who engage in FBT, early weight gain, parental criticism, and obsessiveness have all been identified as moderators that may significantly affect outcomes (Lock and Le Grange, 2019).

Early Weight Gain

At the start of FBT for AN, parents are tasked with assuming responsibility for refeeding and restoring nutrition for their child. Early in treatment, medical acuity also tends to exacerbate parental anxiety, strengthening the belief of many parents that they do not have the skill necessary to address this medical crisis. Therefore, successful early weight gain may assist parents in increasing their sense of self-efficacy. Parental self-efficacy has been proposed as a candidate mediator of FBT treatment outcomes. With this premise, earlier weight gain may lead parents in becoming more empowered within the context of treatment, thereby improving the likelihood of overall weight gain, symptom remission, and treatment success.

Parental Criticism

Expressed emotion is implicated across psychiatric disorders in negatively influencing clinical outcomes. Specific to EDs, parental criticism toward the unwell adolescent (as measured by expressed emotion) has been shown to negatively affect treatment outcomes when FBT is delivered in a conjoint format rather than a separated format (e.g., parent-focused therapy [PFT]). The guiding principles of externalization and agnosticism (see table 21.1) help minimize blame on the adolescent for the ED and reduce parental criticism related to its negative consequences. While a separated format, PFT, may be beneficial, helping families who are quite critical of their offspring when they have an ED remains a significant challenge.

Obsessiveness

Self-starvation is often joined by ED-related (and non-ED-related) obsessions and compulsions, including feeling a "need" to engage in rule-bound behaviors (e.g., cutting food into a certain number of pieces, or not exceeding an exact calorie count in a given meal). This temperament feature can also contribute to non-food-related ED behaviors (e.g., en-

gaging in a set amount or intensity of exercise to offset potential weight gain or perceived negative consequences). These behaviors are treatment resistant, and adolescents who experience greater severity in perseverative ED thinking or other obsessive-compulsive features may require a longer course of FBT to achieve positive treatment outcomes.

Key Considerations in Orienting Families to FBT

There are several ideas to consider when initiating FBT with families that may contribute to more efficient and successful engagement in treatment. Here, we highlight a few examples of how families might be optimally oriented toward an FBT approach.

1. Parents must be encouraged to embrace agnosticism, and to reduce their focus on why or how the ED came about. This perspective may be particularly challenging to adopt not only for caregivers, but also for providers, for whom a professional focus is naturally on questions of causation. The presence of comorbid psychopathology (e.g., anxiety or depression) may also pull attention from prioritizing the ED. Immediate weight restoration and ED symptom reduction is the priority in Phase 1 of FBT, a focus that is urgently needed to reverse the deadly consequences of these disorders. Therefore, it may be helpful to redirect parents to spend less time dwelling on *why* the ED began or on the management of associated affective symptoms and more time on quickly arresting the ED in its process.
2. Parents must take control of all eating-related behavior and be meticulous in this approach. Depending on the family culture, this may feel very different and even uncomfortable, particularly when the adolescent is older or has never required active parenting in the past. Any deviance from this consistent approach, however, fuels and maintains the ED and will only prolong the recovery process. Parents may benefit from being reminded that this is a time-limited effort, and that is critical to ensuring that an FBT approach will be effective.

3. In families with more than one parent, unity between caretakers in the treatment approach is critical. For example, the adolescent might learn that one parent is permissive of a certain behavior (e.g., not finishing a meal) whereas the other parent is stricter; taken together, the child may interpret this inconsistency to mean that meal completion is less important, ultimately strengthening entrenched ED-related beliefs. Consistency in expectations and response to their child's behavior is something the parents likely have experienced in non-ED-related parenting. In the context of an ED, consistency (i.e., caregiver unity) reduces the tendency toward "splitting" the parents in their collaborative efforts against the ED and undermines the grip that the ED holds over the child.
4. Last, parents may be relieved to begin FBT and feel a sense of optimism that they have gotten the help they need. While honoring this optimism, it is also sometimes helpful to remind parents that *they* are the experts in knowing how to help their child recover. Further, EDs are insidious and entrenched disorders that typically require a great deal of time and resources to resolve. An FBT approach requires that the entire family engage in what many parents describe as a "marathon, not a sprint." Therefore, parents may need to actively discuss ways in which they can support one another in maintaining their stamina and adherence to overseeing food preparation and meal supervision. Whenever possible, families may also be encouraged to draw on extended family and friends for emotional and practical support and resources.

Summary

Evidence from several decades of research principally supports a family-based approach in treating children and adolescents who struggle with ED symptomology. A manualized FBT approach is currently the front-line method for intervening in adolescent AN and a recommended approach for adolescent BN. With this FBT model, parents are initially

tasked with refeeding their offspring and interrupting ED symptoms. Taking control of all eating-related matters helps to quickly reverse the dangerous effects of malnutrition and weight loss. Over time, adolescents are gradually granted more autonomy over eating and supported in their return to developmentally normative activity. Families may need to be directed to orient toward assuming full responsibility for feeding their child, reducing blame on the adolescent for the ED crisis, and in prioritizing immediate weight restoration and symptom reversal.

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