Chapter 7
Eating Disorders

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Classification and Diagnosis

Three diagnostic categories of eating disorders were described in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV TR): anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified [1]. A large majority of young people with clinically significant eating disorders, however, did not meet criteria for these disorders, and were assigned to the residual and heterogenous category, eating disorder not otherwise specified [2].

The focus of the Eating Disorder Work group of the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) in creating the DSM-5 section on Feeding and Eating Disorders was to create a clinically useful, evidence-based manual for the accurate and consistent diagnosis of eating disorders across the lifespan. As such, the DSM-IV-TR sections on Feeding and Eating Disorders of Infancy or Early Childhood (feeding disorder of infancy or early childhood [FDIC], pica, and rumination disorder) and the section on Eating Disorders were combined into one section called Feeding and Eating Disorders. This new section includes (1) anorexia nervosa; (2) bulimia nervosa; (3) binge-eating disorder; (4) avoidant/restrictive food intake disorder; (5) pica; (6) rumination disorder; (7) other specified feeding or eating disorder; and (8) unspecified feeding or eating disorder.

Feeding and Eating Disorders include several important changes. Overall, the section has taken a lifespan approach to eating disorders, lowered the thresholds of symptom severity, considers behavioral indicators of eating disorder symptoms even in the absence of direct self-report, and uses multiple factors to ascertain symptom profiles [3]. Adoption of these recommendations is believed to allow for

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earlier identification and intervention to prevent the exacerbation of eating disorder symptoms in young people [3].

The major changes that appear in the Feeding and Eating Disorders section of the DSM-5 [4] encompass the recognition of binge-eating disorder as a formal diagnosis, include modest revisions to the diagnostic criteria for anorexia nervosa and bulimia nervosa, eliminate the diagnostic category eating disorder not otherwise specified, and include avoidant/restrictive food intake disorder, previously described in the DSM-IV TR section "Feeding and Eating Disorders of Infancy or Early Childhood" [5]. These changes aim to clarify existing criteria, ensure more accurate diagnoses, and minimize use of the catch-all heterogeneous category, eating disorder not otherwise specified [5].

Anorexia nervosa, which primarily affects adolescent girls and young women, is characterized by distorted body image and excessive and persistent restriction of energy intake that leads to significantly low body weight, in the context of what is minimally expected for age, sex, developmental trajectory, and physical health. Individuals with anorexia nervosa have either an intense fear of gaining weight or persistent behavior that interferes with weight gain despite low weight. The requisite for numeric weight cutoff requirements and requirement for amenorrhea have been eliminated in DSM-5 [2].

Bulimia nervosa is characterized by repeated episodes of binge eating followed by compensatory behaviors (self-induced vomiting; use of laxatives, diuretics, or other medication, including complementary and alternative medications; fasting; or excessive exercise) that are used to counteract weight gain. DSM-5 criteria reduce the frequency of binge eating and compensatory behaviors that people with bulimia nervosa must exhibit, from twice to once weekly.

Binge-eating disorder was officially recognized in DSM-5 as its own diagnostic category of eating disorder. In DSM-IV TR, binge-eating disorder was not recognized as a disorder but rather described in Appendix B: Criteria Sets and Axes Provided for Further Study and was diagnosable using only the catch-all category of eating disorder not otherwise specified [6]. Binge-eating disorder is characterized by recurrent episodes of eating an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances, with episodes marked by feelings of lack of control [6]. Individuals with binge-eating disorder may eat rapidly and until uncomfortably full, even when not physically hungry. Feelings of guilt, embarrassment, and disgust are common along with binge eating alone to hide the behavior. This disorder is associated with marked distress and occurs, on average, at least once a week over three months.

Avoidant/restrictive food intake disorder, previously known as FDIC in the DSM-IV TR, is characterized by persistent failure to meet appropriate nutritional or energy needs and results in one or more of the following: (1) significant loss of weight (or failure to achieve expected weight gain in children); (2) nutritional deficiency; (3) dependence on enteral feeding or oral nutritional supplements; and/or (4) marked interference with psychosocial functioning [6]. This disorder is a broad category intended to capture individuals who substantially restrict their food intake and experience marked physiologic or psychosocial problems but do not

meet criteria for a feeding or eating disorder [2, 7]. The energy and/or nutritional needs in a patient diagnosed with ARFID are not the result of the lack of available food or a culturally sanctioned practice. Further, the diagnosis of ARFID cannot be made if the adolescent has abnormalities in the way in which they perceive their body weight or shape. Finally, it is not explained by another medical or mental disorder that, if treated, the eating problem will go away. For example, some young people may avoid eating solid food after a gastrointestinal illness and develop significant nutritional problems [2]. Extreme and persistent picky food preferences during childhood leading to clinically significant problems are also captured by the ARFID category [2].

A recent study has shown that the DSM-5 diagnostic criteria have good reliability for anorexia nervosa and bulimia nervosa, and acceptable reliability for binge-eating disorder. Further research on the reliability, validity, and clinical utility are needed for all diagnostic categories [8].

Epidemiology

Published epidemiologic data on eating disorders predate the DSM-5 changes. Therefore, this chapter focuses on the available epidemiologic data prior to these changes. The changes in the DSM-5 will result in new epidemiologic data, resulting in a higher incidence and prevalence of anorexia nervosa, bulimia nervosa, and binge-eating disorder. Eating disorders have peak onset during adolescence, with a reported incidence of anorexia nervosa of 109/100,000 in 15–19 year olds [9]. While the overall incidence rate of anorexia nervosa in adults has remained stable over the past decades, there has been an increase in the high-risk group of 15–19-year-old girls [9, 10]. It is uncertain whether this finding is due to earlier detection of anorexia nervosa or an earlier age at onset [10]. The incidence of eating disorders under the age of 13 has been estimated between 1.1 and 2.6/100,000 in three nationally representative pediatric surveillance studies in Canada, Australia, and United Kingdom [11–13]. Eating disorders have also been reported in association with several chronic illnesses and disabilities, in particular type 1 diabetes mellitus [14, 15].

The lifetime prevalence of eating disorders in adults is about 0.6 % for anorexia nervosa, 1 % for bulimia nervosa, and 3 % for binge-eating disorder [16, 17]. Women are more affected than are men [16]. Eating disorders have been reported in both developing and developed nations [18–20]. An increasing occurrence of eating disorders in non-Western societies has been associated with cultural transition and globalization [21–23]. Individuals with eating disorders have significantly elevated mortality rates, with the highest rates occurring in those with anorexia nervosa [24]. The mortality rate from anorexia nervosa is estimated at 5.9 %, the highest rate of mortality among all mental disorders [25]. Comorbid psychiatric conditions are also highly prevalent in individuals with eating disorders [16, 17]. Despite the magnitude, many individuals with eating disorders do not seek treatment [16].

Recent studies have reported that the prevalence of avoidant/restrictive food intake disorder in tertiary care pediatric eating disorders program range from 5 to 14 % [26, 27]. Additional studies from community-based samples are needed.

Pathogenesis and Etiology

Research into the pathogenesis of eating disorders has focused mainly on anorexia nervosa and bulimia nervosa. The causes of eating disorders are complex and include biological, hormonal, psychological, and environmental components. The heritability of eating disorders is similar to that of other psychiatric conditions [28]. Twin and family studies estimate heritability ranges between 50 and 83 % for anorexia nervosa, bulimia nervosa, and binge-eating disorders [29–31]. Hence, considerable genetic influences on the etiology can be assumed for eating disorders [32, 33]. Molecular genetic studies have identified genes and chromosomal regions that may contribute to the development of an eating disorder [34–37].

Many of the biological findings in eating disorders are a result of starvation and malnourishment. However, some are causally linked as risk or maintaining factors [38]. The brain is particularly vulnerable to the consequences of poor nutrition. Alterations in brain structure, metabolism, and neurochemistry have been identified in malnourished and emaciated individuals with anorexia nervosa [39–42]. Alterations in brain metabolism and atrophy have also been reported in bulimia nervosa [43, 44]. These changes are associated with many behavioral and psychosocial disturbances such as rigidity, emotional dysregulation, and social difficulties [38]. Although many symptoms resolve with weight gain, disturbances in brain circuits modulating appetite, mood, cognitive function, and impulse control may persist after recovery from an eating disorder [45–47].

Environmental influences may also contribute to the risk of developing an eating disorder. For example, high-concern parenting in infancy may be associated with the later development of anorexia nervosa [48]. Perinatal complications and premature delivery may also increase the risk of development of an eating disorder by epigenetic mechanisms or damage to the brain from hypoxia [49]. The idealization of thinness in some developed societies encourages dieting and weight-control practices. Mass media propagate a slender ideal that elicits body dissatisfaction [50]. Girls who are obese, experience early puberty, criticism, teasing, and bullying are at increased risk of developing an eating disorder [51–53]. Personality traits such as perfectionism, concerns over self-control, sensitivity to rejection, and low self-esteem have also been implicated. Some sports, such as cheerleading, figure skating, gymnastics, dance, and long distance running, may promote weight loss or thinness, thereby encouraging an eating disorder to develop. Risk factors for eating disorders are summarized in Table 7.1 [52, 54–56].

Table 7.1 Potential risk factors for the development of an eating disorder

1.	Age and female gender
2.	Early childhood eating problems
3.	Childhood obesity or overweight
4.	Weight related teasing of the child/adolescent
5.	Dieting
6.	Perinatal adverse events (prematurity, small for gestational age, cephalohematoma)
7.	Personality traits such as perfectionism, anxiety, low self-esteem, obsessionality
8.	Early puberty
9.	Chronic illness
10.	Physical and sexual abuse
11.	Family history of psychiatric illness, eating disorder, or obesity
12.	Competitive athletics i.e., gymnastics, ice skating, ballet, wrestling
13.	Overanxious parenting

Based on data from [52, 54–56]

Assessment

Primary care clinicians play an important role in the initial detection, evaluation, and progression of eating disorders. Early detection and management of an eating disorder may prevent or lessen the medical complications and psychological consequences associated with starvation and progression of the illness [57, 58]. Primary and secondary prevention is achieved by screening for eating disorders as part of routine annual health care, providing ongoing monitoring and documentation of weight and height on growth charts, and paying careful attention to the signs and symptoms of an early eating disorder [58]. Screening questions regarding eating patterns and body image that can be used for all adolescents and young adults presenting for routine health care are shown in Table 7.2. In addition, the SCOFF screening questionnaire, although not validated in children and adolescents, can provide a framework for screening (Table 7.3). A recent meta-analysis has shown this to be a very useful screening tool [59].

Concern with weight and body shape is extremely common during adolescence [60, 61]. A significant number of pre-adolescents may also have a desire to be thinner [62, 63]. Canadian, American, and Australian cross-sectional data suggest that more than one in five teenagers are "on a diet" at any given time [60, 62, 64, 65]. Approximately 40–66 % of teenage girls and 20–30 % of teenage boys have attempted dieting in the past [66]. Any evidence elicited on history or physical examination of dieting, excessive concern with weight or shape, weight loss or failure to gain weight as expected for age and developmental stage requires further attention. Careful assessment for the possibility of an eating disorder and close monitoring at intervals as frequent as every 1–2 weeks may be needed until the situation becomes clear [58].

Table 7.2 Screening questions that may help to identify an eating disorder at a routine health care visit

What is the most/least you ever weighed? How tall were you then? When was that?
What is your ideal weight?
What do you do for exercise? Level of intensity? How stressed are you if you miss a workout?
Ask for specific dietary practices:
• 24-h diet and fluid history?
• Calorie counting, fat gram counting? Taboo foods? Restrictions?
• Early satiety, bloating, reflux?
• Any binge eating? Frequency, amount, triggers?
• Purging history?
• Use of diuretics, laxatives, diet pills, ipecac?
• Any vomiting? Frequency, how long after meals?
Menstrual history in females: age at menarche? Regularity of cycles? Last menstrual period?
Any history of depression, anxiety, suicidal ideation or attempts?
Use of cigarettes, drugs, alcohol? Sexual history? History of physical or sexual abuse?
Family history: obesity, eating disorders, depression, other mental illness, substance abuse by parents or other family members?
Review of symptoms:
• Dizziness, syncope, weakness, fatigue?
• Pallor, easy bruising or bleeding?
• Cold intolerance?
• Hair loss, lanugo, dry skin?
• Vomiting, diarrhea, constipation?
• Fullness, bloating, abdominal pain, epigastric burning?
• Muscle cramps, joint paints, palpitations, chest pain?
• Symptoms of hyperthyroidism, diabetes, malignancy, infection, inflammatory bowel disease?

Based on data from [58, 70]

Table 7.3 The SCOFF questions[a]

Do you make yourself **S**ick because you feel uncomfortably full?
Do you worry you have lost **C**ontrol over how much you eat?
Have you recently lost more than **O**ne stone in a 3-month period?
Do you believe yourself to be **F**at when others say you are too thin?
Would you say that **F**ood dominates your life?

[a]One point for every "yes"; a score of ≥2 indicates a likely case of anorexia nervosa or bulimia nervosa

Possible findings on physical examination are detailed in Table 7.4. In addition, when an adolescent is referred to their clinician because of concerns raised by parents, friends, or school that he or she is displaying evidence of an eating disorder, it is most likely that the adolescent does have an eating disorder [58]. These concerns should be taken very seriously even if the adolescent denies all symptoms.

Initial laboratory investigations in an eating disorder evaluation should include a complete blood count and differential, platelet count, electrolytes (including calcium, phosphate and magnesium), glucose, liver function tests, thyroid-stimulating

Table 7.4 Possible findings on physical examination in patients with eating disorders

Bradycardia
Hypotension
Hypothermia
Cardiac murmur (mitral valve prolapse)
Dull, thinning scalp hair
Sunken cheeks, sallow and dry skin
Lanugo hair
Atrophic breasts (postpubertal)
Atrophic vaginitis (postpubertal)
Pitting edema of extremities
Emaciated, may wear oversized clothes
Flat affect
Cold extremities, acrocyanosis
Parotitis
Russell's sign (callous on knuckles from self-induced emesis)
Mouth sores
Palatal scratches
Dental enamel erosions

Table 7.5 Differential diagnosis of eating disorders

• Gastrointestinal: inflammatory bowel disease, celiac disease, malabsorption
• Endocrine: hyperthyroidism, diabetes mellitus, Addison's disease, hypopituitarism
• Rheumatologic: systemic lupus erythematosus
• Neurologic: central nervous system lesions (hypothalamic or pituitary tumors)
• Infections: tuberculosis, HIV
• Malignancy: leukemia, lymphoma, brain tumor
• Other: collagen vascular disease, cystic fibrosis
• Psychiatric disorders including mood disorders, anxiety disorders, somatization and psychosis

hormone, erythrocyte sedimentation rate, and urinalysis. Markers of nutritional status (albumin, vitamin D, folate, vitamin B12, iron, and other minerals) may also be considered. Additional tests (urine pregnancy, luteinizing and follicle-stimulating hormone, prolactin, and estradiol) should be considered in patients who are amenorrheic or have delayed puberty. An electrocardiogram should be performed on all patients. Bone densitometry should be considered in those females who are amenorrheic for more than 6 months [67]. Other tests such as echocardiogram, upper gastrointestinal tract series, or brain imaging should be considered in select circumstances as guided by the history and physical examination. For example, magnetic resonance imaging and neuropsychological assessment may be needed for patients with atypical features, such as hallucinations, delusions, delirium, and persistent cognitive impairment, despite weight restoration [68]. Normal laboratory investigations in patients with eating disorders do not exclude serious illness or medical instability. A broad differential diagnosis for the adolescent with symptoms of an eating disorder should always be considered (Table 7.5).

Medical Complications

Eating disorders in adolescents and young adults can cause *serious* medical complications in every organ system (Table 7.6) [42, 58, 69]. The medical complications occurring in individuals with an eating disorder are largely related to the effects of starvation, malnutrition, and weight-control behaviors such as vomiting and laxative abuse. The consequences of nutritional deprivation and metabolic impairment on the growing and developing adolescent body also depend on the length, severity, and number of episodes of restriction and, the timing of those episodes in relationship to normal periods of growth and physical development [70, 71].

Although many of the medical complications improve with nutritional rehabilitation and recovery from the eating disorder, some are potentially irreversible [42]. If the eating disorder occurs before the closure of the epiphyses, growth retardation may become potentially irreversible [72–76], resulting in failure to achieve expected adult height. Other potentially irreversible medical complications in adolescents include loss of dental enamel with chronic vomiting [77]; structural brain changes [39, 78]; pubertal delay or arrest [79]; and impaired acquisition of peak bone mass with an increased fracture risk secondary to low bone mineral density [80–83].

Table 7.6 Medical complications resulting from eating disorders

Medical complications from purging
1. Dehydration and electrolyte imbalance (hypokalemia; hypophosphatemia); hypochloremic alkalosis
2. Use of ipecac: irreversible myocardial damage
3. Chronic vomiting: esophagitis; dental erosions; Mallory-Weiss tears; rare esophageal or gastric rupture
4. Use of laxatives: metabolic acidosis; increased blood urea nitrogen concentration; hyperuricemia; hypocalcemia; hypomagnesemia; chronic dehydration
5. Amenorrhea (can be seen in normal or overweight individuals with bulimia nervosa); menstrual irregularities
Medical complications from caloric restriction
1. Cardiovascular: Electrocardiographic abnormalities: low voltage (sinus bradycardia, T wave inversion, ST segment depression, prolonged corrected QT interval); dysrhythmias include supraventricular beats and ventricular tachycardia; pericardial effusions; congestive heart failure; sudden death; mitral valve prolapse; orthostatic hypotension or tachycardia
2. Gastrointestinal: delayed gastric emptying; slowed gastrointestinal motility; constipation; bloating; hypercholesterolemia; abnormal liver function tests; fatty liver; superior mesenteric artery syndrome; gallstones
3. Renal: increased blood urea nitrogen concentration (from dehydration, decreased glomerular filtration rate) with increased risk of renal stones; total body sodium and potassium depletion caused by starvation; peripheral edema; urinary incontinence
4. Hematologic: leukopenia; anemia; iron deficiency; thrombocytopenia
5. Endocrine: euthyroid sick syndrome; amenorrhea; hypercortisolism; hypercholesterolemia; hypoglycemia; pubertal delay; impaired linear growth; low bone mineral density
6. Neurologic: cortical atrophy; seizures (secondary to metabolic derangements); cognitive deficits

Based on data from [42, 58, 69]

Eating disorders are life-threatening illnesses. At least one-third of all deaths in adults with anorexia nervosa are due to cardiac complications [84–86]. Cardiac abnormalities are often present in the early stages of the eating disorder and may be reversible with prompt identification and treatment [87–89]. Common cardiovascular complications include electrocardiographic abnormalities such as sinus bradycardia, decreased voltage and prolonged QTc, orthostatic hypotension, increased vagal tone, poor myocardial contractility, mitral valve prolapse, reduction in left ventricular wall thickness and mass, and silent pericardial effusion [42, 69, 87–92]. Sinus bradycardia is present in 35–95 % of adolescents with anorexia nervosa, and is believed to be due to the reported increased vagal tone and decreased metabolic rate [42, 69, 91, 92]. Electrocardiographic abnormalities may also be due to other secondary causes such as metabolic and electrolyte disturbances, illicit drugs, medications, or complementary and alternative therapies [42].

Cardiovascular complications occur not only in the initial stages of the disorder but also during refeeding. Refeeding syndrome is a term that refers to various metabolic abnormalities that occur in severely malnourished patients following carbohydrate administration [93, 94]. Clinically, refeeding syndrome consists of a constellation of cardiac, hematological, and neurological symptoms. It has been reported in 6 % of hospitalized patients and can include congestive heart failure and pedal edema, a prolonged QT interval with arrhythmia, tachycardia, and sudden cardiac death [68, 89]. Although multiple organ systems may be involved, cardiac and neurologic dysfunction has been noted in those most severely affected [94]. Hypophosphatemia, a potentially life-threatening complication, is recognized as the biochemical hallmark for refeeding syndrome [95]. Refeeding hypophosphatemia has been associated with the degree of malnutrition [96]. Other electrolyte derangements (i.e., hypokalemia, hypomagnesaemia, hypocalcemia) may also occur and generally result from transcellular shifts of fluid and electrolytes as well as total body depletion [95]. Electrolyte disturbances require immediate attention.

The major endocrine abnormalities associated with eating disorders include hypogonadotropic hypogonadism, hypercortisolemia, hypoglycemia, growth hormone resistance, impaired linear growth, and sick euthyroid syndrome [97, 98]. The clinical manifestation of dysfunctional hypothalamic–pituitary–ovarian axis is amenorrhea and pubertal delay [69]. Development of a low bone density is a serious complication in adolescents with eating disorders, as adolescence is a critical period for the attainment of peak skeletal mass [42]. The pathogenesis of bone loss is associated with impaired bone formation and increased bone resorption, hypoestrogenemia, decreased levels of IGF-1, low dehydroepiandrosterone (DHEA) concentrations, increased cortisol levels, physical activity, poor nutrition, reduced leptin levels, low calcium and vitamin D intake, and low body mass [42, 99]. Weight restoration and the resumption of menses is the safest and most effective way to increase bone mineralization in adolescents with anorexia nervosa [80, 100]. Oral estrogen–progesterone combination pills have not been proven to be effective in increasing bone mineral density. Recent data suggests that physiologic estrogen in the form of the transdermal patch in older girls (bone age >15 years) increases spine and hip bone mineral density. However, complete catch-up in bone mineralization did not occur [101].

In addition, a prospective, randomized controlled study using oral micronized DHEA and estrogen–progesterone combination pills prevented bone loss in young women with anorexia nervosa compared to the decrease in areal BMD in women receiving a placebo [102]. Although most of the endocrine changes that occur in anorexia nervosa represent physiologic adaptation to starvation, some may persist after recovery [97, 98].

Alteration in renal function manifesting as abnormal blood urea nitrogen, decreased glomerular filtration rate, hematuria, and proteinuria have been described in patients with eating disorders [69]. Urea and creatinine are generally low and normal concentrations may mask dehydration or renal dysfunction [103]. Further, 17 % of adolescents with anorexia nervosa have been shown to have nocturnal enuresis, which is thought to be related to decreased functional bladder capacity and detrusor instability [104].

Serum pH and electrolyte abnormalities are common and result from starvation, laxative abuse, diuretic use, dehydration, or the practice of water loading to artificially increase weight [69]. Metabolic alkalosis occurs in patients who vomit or abuse diuretics and acidosis in those misusing laxatives [103]. Hypokalemia frequently results from purging by vomiting or laxative abuse. Hyponatremia is often due to excessive water intake, but may also occur in chronic energy deprivation or diuretic misuse [103]. Symptoms of electrolyte abnormalities are rarely present or are denied by patients [69].

Hematologic abnormalities may include anemia, leukopenia, and thrombocytopenia [105, 106]. These changes are generally attributed to starvation-mediated gelatinous bone marrow transformation, which resolves with proper nutritional rehabilitation [105, 106]. Gastrointestinal abnormalities include slowed gastric emptying, constipation, abdominal bloating and pain, and elevated aminotransferases. Abnormalities of liver enzymes may occur before or during refeeding [103]. Hypercholesterolemia is another common finding but its significance for cardiovascular risk is uncertain [103]. Other abnormalities include micronutrient deficiencies, hyperamylasemia, hypercarotenemia, elevated creatine kinase, xerosis, lanugo-like body hair, acrocyanosis, slower wound healing, and reduced fever response [103, 107–109].

Treatment

Eating disorders are associated with extremely complex medical and psychosocial issues that are best addressed by an interdisciplinary team of medical, nutritional, mental health, and nursing professionals who are skilled and knowledgeable in working with adolescents with eating disorders and their families [57, 110]. Initial evaluation of the adolescent with a suspected eating disorder includes establishment of the diagnosis; determination of severity, including evaluation of medical and

Table 7.7 Indications for hospitalization of an eating disorder

1.	Severe malnutrition (weight <75 % average body weight for age, sex, and height)
2.	Dehydration
3.	Electrolyte imbalance (hypokalemia, hyponatremia, hypophosphatemia)
4.	Cardiac dysrhythmia
5.	Physiological instability
	a. Severe bradycardia (heart rate <50 beats/minute, daytime; <45 beats/minute at night)
	b. Hypotension
	c. Hypothermia
	d. Orthostatic changes in pulse (20 beats per minute) or blood pressure (10 mmHg)
6.	Arrested growth or 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)
11.	Acute psychiatric emergencies (e.g., suicidal ideation, acute psychosis)
12.	Comorbid diagnosis that interferes with the treatment of the eating disorder (e.g., severe depression, obsessive compulsive disorder, severe family dysfunction)

Based on data from [57, 110]

nutritional status; and performance of an initial psychosocial evaluation. Depending on the patient and family circumstances, various levels of treatment options are available for adolescents with eating disorders (inpatient, outpatient, day hospital, or residential treatment). The time to full recovery from an eating disorder may take several years [111].

Indications for hospitalization are listed in Table 7.7 [57, 58, 110]. The main goals of inpatient treatment are medical stabilization and weight restoration through nutritional rehabilitation (about 2–3 lbs per week). Recent studies have shown that safe weight gain can occur starting with approximately 1,400—2,000 kcal/day with regular nutritional advancements [96, 112–114]. Close monitoring of weight, vital signs, fluid shifts, and serum electrolytes during the first week of hospitalization is recommended [96, 112–114]. Attempts should be made to achieve weight gain through the oral route; however, short-term nasogastric feeding may be necessary in some patients. Supplementation with calcium (1,300 mg/day), in accordance with the Institute of Medicine recommendations for adolescents [115] and vitamin D (600–1,000 IU) is often necessary. The recommended length of hospitalization has not been established, although risk of relapse is lower in patients who are discharged closer to ideal body weight compared to patients discharged at very low body weight [116].

All adolescents with eating disorders should undergo a mental health evaluation, and be evaluated for potential treatment, which may include the use of anxiolytic or

antidepressant medications. Although there remains relatively little research on interventions that address the complex mental and physical needs of adolescents with eating disorders, evidence-based research supports that family-based treatment, also known as the Maudsley approach, is an effective first-line outpatient treatment for adolescents with eating disorders and protective against relapse, particularly in anorexia nervosa [117–120]. Family-based treatment is an intensive outpatient treatment that utilizes parents/caregivers as a primary resource to renourish their affected child or adolescent [121]. Typically, one therapist is involved, along with a physician to provide medical care [122]. Although family-based treatment is effective for adolescents with bulimia nervosa, cognitive-behavioral therapy that focuses on changing the specific eating attitudes and behaviors that maintain the eating disorder may be more effective in older adolescents and young adults [123]. The evidence for binge-eating disorders in adolescents is insufficient to draw any conclusions; however, cognitive-behavioral therapy, interpersonal therapy, and dialectical behavior therapy may be helpful [123]. It is important to note that correction of malnutrition is required for the mental health aspects of care to be effective.

The literature regarding treatment efficacy and outcomes for eating disorders in adults is of highly variable quality [120, 124]. Current evidence does not suggest any one particular psychotherapeutic modality for adults with anorexia nervosa [125]. In bulimia nervosa, cognitive-behavioral therapy is frequently used and may reduce the risk of relapse after weight restoration [110, 124, 126, 127]. Psychological interventions that have shown effective in the treatment of bulimia nervosa also show promise in binge-eating disorder, particularly modified cognitive-behavioral therapy, interpersonal therapy, and dialectical behavior therapy (dialectical behavior therapy, also known as DBT, combines cognitive-behavioral approaches for emotion regulation and reality-testing) [128, 129].

The literature on pharmacologic treatment in either in the acute or maintenance phases of anorexia nervosa remains sparse and inconclusive [38, 130]. There is currently no strong evidence of beneficial effects using antidepressants and antipsychotics in adolescents and adults with anorexia nervosa [130, 131]. Medications, specifically selective serotonin reuptake inhibitors (such as fluoxetine, sertraline, paroxetine, fluvoxamine, and citalopram) may be used to treat comorbid psychiatric disorders such as anxiety, depression, and obsessive compulsive disorder or behavior [132]. There is conflicting evidence as to whether antidepressants reduce the risk of relapse in older adolescents with anorexia nervosa who have attained 85 % of expected body weight [133]. The use of atypical antipsychotics in adolescents with anorexia nervosa is encouraging; however, it is limited to case series and case reports [134–136]. These medications have been shown to be effective in reducing anxiety and obsessional thinking in adolescents with anorexia nervosa.

Antidepressants have been shown to have a positive effect in patients with bulimia nervosa. Fluoxetine is the only medication approved by the FDA for the treatment of bulimia nervosa resulting in decrease in binge-eating and purging episodes in 55–65 % of patients [127]. Antidepressant medication in combination with cognitive-behavioral therapy appears to be superior to either modality alone in the treatment of older adolescents or adults with bulimia nervosa [137].

Prognosis

Eating disorders are marked by a serious course and outcome in many afflicted individuals. In anorexia nervosa, there is an almost 18-fold increase in mortality, including a high suicide rate, chronic course in approximately 20 %, and more than half of patients showing significant psychiatric comorbidity [138]. The prognosis for adolescents with anorexia nervosa is better than for adults, mainly due to the shorter duration of illness and younger age at diagnosis [138–140]. Other factors associated with good prognosis include early identification and treatment, less weight loss, and strong support network [38, 141]. Worse prognosis is associated with a history of extreme or precipitous weight loss, vomiting, and somatic and psychiatric comorbidity [141, 142]. Recovery tends to follow the rule of thirds: one-third of patients fully recover, one-third resort to disordered eating strategies and behaviors as their default coping strategies, and one-third have a chronic and relapsing course [138, 139, 141, 143]. Little research has been done into the prognostic factors and outcome of bulimia nervosa and binge-eating disorder.

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