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Guidance for the Clinician in Rendering Pediatric Care

Clinical Report—Identification and Management of Eating Disorders in Children and Adolescents

abstract

FREE

The incidence and prevalence of eating disorders in children and adolescents has increased significantly in recent decades, making it essential for pediatricians to consider these disorders in appropriate clinical settings, to evaluate patients suspected of having these disorders, and to manage (or refer) patients in whom eating disorders are diagnosed. This clinical report includes a discussion of diagnostic criteria and outlines the initial evaluation of the patient with disordered eating. Medical complications of eating disorders may affect any organ system, and careful monitoring for these complications is required. The range of treatment options, including pharmacotherapy, is described in this report. Pediatricians are encouraged to advocate for legislation and policies that ensure appropriate services for patients with eating disorders, including medical care, nutritional intervention, mental health treatment, and care coordination. *Pediatrics* 2010;126: 1240–1253

INTRODUCTION

Increases in the incidence and prevalence of anorexia nervosa (AN), bulimia nervosa (BN), and other eating disorders in children and adolescents make it critically important that pediatricians be familiar with early detection and appropriate management of these disorders. Results of epidemiologic studies have indicated that the numbers of children and adolescents with eating disorders increased steadily from the 1950s onward.[1–4] During the past decade, the prevalence of obesity in children and adolescents has also increased dramatically,[5–9] accompanied by further emphasis on dieting and weight loss among children and adolescents.[10–15]

The epidemiology of eating disorders has gradually changed; there is an increasing prevalence of eating disorders in males[16–19] and minority populations in the United States[20–23] as well as in countries in which eating disorders had not been commonly seen.[3,4,24,25] Of particular concern is the increasing prevalence of eating disorders at progressively younger ages.[19,26,27] A recent analysis by the Agency for Healthcare Research and Quality revealed that from 1999 to 2006, hospitalizations for eating disorders increased most sharply—119%—for children younger than 12 years.[19]

It is estimated that approximately 0.5% of adolescent girls in the United States have AN, that approximately 1% to 2% meet diagnostic criteria for BN, and that up to 5% to 10% of all cases of eating disorders occur in males. A large number of people with eating disorders do not meet the strict criteria set forth in the American Psychiatric Association's

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KEY WORDS
anorexia nervosa, bulimia nervosa, eating disorders

ABBREVIATIONS
AN—anorexia nervosa
BN—bulimia nervosa
DSM-IV-TR—*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition Text Revision*
HPA—hypothalamic-pituitary-adrenal
SSRI—selective serotonin-reuptake inhibitor

The guidance in this report does not indicate an exclusive course of treatment or serve as a standard of medical care. Variations, taking into account individual circumstances, may be appropriate.

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Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR) for AN or BN and are labeled as having "partial syndromes" or "eating disorder not otherwise specified" (ED NOS).[28] There are many more patients with ED NOS than there are patients with AN or BN; the prevalence is estimated to be between 0.8% and 14%, depending on the definition used.[29] These patients often experience the same physical and psychological consequences as do those who reach the threshold for diagnosis of AN or BN.[28–34] Athletes and performers, particularly those who participate in sports and activities that reward a lean body habitus (eg, gymnastics, running, wrestling, dance, modeling) may be at particular risk of developing partial-syndrome eating disorders.[35,36]

The etiology of eating disorders is multifactorial, and there is increasing evidence from both family and twin studies for a strong genetic component that is shared between AN and BN.[37,38] The mechanism(s) by which genetic factors influence risk have not been elucidated, but various hypotheses have been proposed. Genetic predisposition to various trait disturbances such as behavioral rigidity, perfectionism, or harm avoidance may be more salient than genetic influences on eating, hunger, or satiety.[39–41] Genetic effects seem to be "activated" by puberty,[42–44] and there is strong evidence for genetic-environment interactions.[39,40]

Dieting has also been implicated as a potent proximal risk factor in the development of disordered eating and eating disorders.[45–47] In 1 community-based study, dieters at 5-year follow-up were at significantly higher risk of disordered eating behaviors (eg, vomiting or using diet pills or laxatives) than nondieters and were also at increased risk of obesity.[47] In another large community cohort, dieters were 5 times more likely to develop an eating disorder and severe dieters were 18 times more likely to develop an eating disorder than nondieters.[48]

Neuroendocrine abnormalities have been implicated in the etiology of eating disorders. Leptin is a circulating hormone produced in adipose tissue and seems to have a significant role in mediating the neuroendocrine effects of AN. Leptin concentrations are sensitive to the acute metabolic effects of decreased intake and energy deficits, and decreased circulating leptin concentrations reflect depleted stores of body fat.[49–51] Physical hyperactivity is a common feature of AN and sometimes manifests as restlessness, athleticism, or compulsive exercise. This hyperactivity also seems to be mediated by leptin.[51]

Physical hyperactivity associated with weight loss seems to occur in animals as well, apparently mediated by hyperactivity of the hypothalamic-pituitary-adrenal (HPA) axis. Syndromes that resemble AN, characterized by food refusal, physical overactivity, and extreme weight loss, occur in pigs, sheep, and goats bred for leanness.[52] Caloric restriction coupled with environmental stress produces animal models for binge-eating.[53] These animals overeat dramatically despite nutritional satiety and normal energy status, which strongly suggests that reward circuits are being activated rather than metabolic needs being satisfied.[43,53]

In community-based studies of adolescents, disturbances of body image and overconcern about body shape are common, although the prevalence of eating disorders remains low.[54] These results reinforce the likelihood of epigenetic effects in which the development of eating disorders reflects the intersection between genetic predisposition, environmental triggers, and personal experience.

SCREENING FOR EATING DISORDERS IN PRACTICE

Primary care providers are in a unique position to detect the onset of eating disorders at the earliest stages and to stop their progression.[32,33] Pediatricians should screen for eating disorders as part of annual health supervision or during preparticipation sports examinations by monitoring weight and height longitudinally and paying careful attention to potential signs and symptoms of disordered eating.

Screening questions about eating patterns and body image should be asked of all preteens and adolescents. The *Bright Futures* guidelines provide examples for addressing this issue with adolescents of different ages.[55] The SCOFF questionnaire, although validated only in adults, can provide a framework for screening (Table 1).[56] Weight, height, and BMI should be determined regularly and plotted on appropriate growth charts. Deviations from normal are easier to identify visually, because nutritional insufficiency may be manifest by falloff in either height or weight percentiles rather than actual weight loss. Growth charts are available for plotting changes in weight, height, and BMI over time and for comparing individual measurements with age-appropriate population norms.

Any evidence of excessive weight concern, inappropriate dieting, or a pattern of weight loss requires further attention, as does primary or secondary

TABLE 1 The SCOFF Questionnaire[56]

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 **>1 stone** (6.3 kg or 14 lb) in a 3-mo 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?

One point should be given for every "yes" answer; a score of ≥2 indicates a likelihood of AN or BN.

amenorrhea or a failure to achieve appropriate increases in weight or height in growing children. In each of these situations, careful assessment for the possibility of an eating disorder and close monitoring at intervals as frequent as every 1 to 2 weeks may be needed until the situation is clarified. Adolescent girls who seek physician care for weight, shape, or eating concerns have been shown to be at significantly higher risk of a subsequent diagnosis of AN.[57]

A number of studies have shown that most adolescent girls express concerns about being overweight, and many may diet inappropriately.[10–12,14] Most of these children and adolescents do not have an eating disorder. On the other hand, it is known that patients with eating disorders often try to hide their illness, so simple denials by the adolescent do not exclude the possibility of an eating disorder. Obtaining collateral history from a parent may help identify abnormal eating attitudes or behaviors, although parents may, at times, be unaware or in denial as well. When an adolescent is referred to a pediatrician because parents, friends, or school personnel suspect the possibility of an eating disorder, it is likely that disordered eating is present. Pediatricians must, therefore, not be lulled into a false sense of security if the adolescent denies all symptoms. Table 2 outlines questions that are useful in eliciting a history of eating disorders, and Table 3 delineates possible physical findings in children and adolescents with eating disorders.

DSM-IV-TR criteria[28] for the diagnosis of AN and BN are outlined in Table 4. These criteria focus on the weight loss, attitudes and behaviors, and amenorrhea displayed by patients with eating disorders. Limitations of these criteria, especially as they relate to children and adolescents, have been discussed extensively in the literature,[54,58–61] and revisions to these criteria have been proposed for the fifth edition of the manual.[60,61] Alternative schema for the classification of eating disorders in children have been described to better reflect the range of eating issues seen.[58,62]

Younger patients (<13 years of age) with eating disorders are more likely to have premorbid psychopathology (depression, obsessive-compulsive disorder, or other anxiety disorders) and are less likely to have binge/purge behaviors associated with their illness. The predominance of females is far less; among the youngest patients with eating disorders, males and females may be equally affected. Weight loss often occurs at a faster rate than in older patients. Still, studies have shown that more than half

TABLE 2 History

Specific history
What is the most you ever weighed? How tall were you then? When was that?
What is the least you ever weighed in the past year? How tall were you then? When was that?
What do you think is your healthy weight?
What would you like to weigh?
Exercise: how much, how often, level of intensity? How stressed are you if you miss exercising?
Current eating habits: adequacy of intake, portion sizes, food restrictions, picky eating, fluid intake, ritualized eating habits? Recent vegetarianism? Excessive noncaloric fluid intake?
24-h diet history?
Calorie-counting? Fat gram–counting? Carbohydrate-counting?
Any binge-eating? Frequency? Triggers?
Purging history?
Use of diuretics, laxatives, diet pills, or ipecac? Ask about elimination pattern, constipation, diarrhea. Any vomiting? Frequency? Timing in relation to meals?
Any previous therapy? What kind and how long? What was and was not helpful?
Symptoms of hyperthyroidism, diabetes, malignancy, infection, inflammatory bowel disease?
Family history: obesity, eating disorders, depression, other mental illness (especially anxiety disorders and obsessive-compulsive disorder), substance abuse by parents or other family members?
Menstrual history: age at menarche? Regularity of cycles? Last menstrual period?
Use of cigarettes, drugs, alcohol?
Use of anabolic steroids (especially in boys)?
Use of stimulants?
Involvement with proanorexia ("pro-ana") or probulimia ("pro-mia") Web sites
History of physical or sexual abuse?
Review of symptoms
Dizziness, presyncope, syncope, fatigue?
Pallor, easy bruising or bleeding?
Cold intolerance? Cold extremities?
Palpitations, chest pain, shortness of breath? Exercise intolerance?
Hair loss, lanugo, dry skin?
Fullness, bloating, abdominal pain, epigastric burning?
Vomiting, symptoms of gastroesophageal reflux?
Change in bowel habits? Diarrhea, constipation, rectal bleeding?
Weakness, muscle cramps?
Menstrual irregularities?

TABLE 3 Physical Examination Findings Sometimes Seen in Children and Adolescents With Eating Disorders

Sinus bradycardia; other cardiac arrhythmias
Orthostatic changes in pulse (>20 beats per min) or blood pressure (>10 mm Hg)
Hypothermia
Cachexia; facial wasting
Cardiac murmur (one-third with mitral valve prolapse)
Dull, thinning scalp hair
Sialoadenitis (parotitis most frequently reported)
Angular stomatitis; palatal scratches; oral ulcerations; dental enamel erosions
Dry, sallow skin; lanugo
Bruising/abrasions over the spine related to excessive exercise
Delayed or interrupted pubertal development
Atrophic breasts; atrophic vaginitis (postpubertal)
Russell sign (callous on knuckles from self-induced emesis)
Cold extremities; acrocyanosis; poor perfusion
Carotenemia (orange discoloration of the skin, particularly palms and soles)
Edema of the extremities
Flat or anxious affect

TABLE 4 Diagnosis of AN, BN, and Eating Disorders Not Otherwise Specified, From DSM-IV-TR[28]

AN

1. Refusal to maintain body weight at or above a minimally normal weight for age and height (ie, weight loss that leads to maintenance of body weight 85% of that expected or failure to make expected weight gain during period of growth and leads to a body weight of 85% of that expected).
2. Intense fear of gaining weight or becoming fat, even though underweight
3. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of current body weight
4. In postmenarcheal females, amenorrhea (ie, the absence of at least 3 consecutive menstrual cycles)

Types

- Restricting type: no regular bingeing or purging (self-induced vomiting or use of laxatives and diuretics)
- Binge-eating/purging type: regular bingeing or purging behavior

BN

1. Recurrent episodes of binge-eating characterized by (*a*) eating, in a discrete period of time, an amount of food that is definitely larger than most people would eat in a similar period of time and under similar circumstances and (*b*) a sense of lack of control over eating during the episode
2. Recurrent inappropriate compensatory behavior to prevent weight gain, such as self-induced vomiting, misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise
3. The binge-eating and inappropriate compensatory behaviors both occur, on average, at least twice per week for 3 mo
4. Self-evaluation unduly influenced by body shape or weight
5. The disturbance does not occur exclusively during episodes of AN

Types

- Purging type: the person has regularly engaged in self-induced vomiting or misuse of laxatives, diuretics, or enemas
- Nonpurging type: the person has used other inappropriate compensatory behaviors, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas

Eating disorder not otherwise specified

Disorders of eating that do not meet the criteria for either AN or BN; examples include

- All criteria for AN are met except the patient has regular menses
- All criteria for AN are met except that despite significant weight loss, weight remains in the normal range
- All criteria for BN are met except that binge-eating and inappropriate compensatory behaviors occur less frequently than twice per week or for a duration of <3 mo
- A patient with normal body weight who regularly engages in inappropriate compensatory behavior after eating small amounts of food (eg, self-induced vomiting after eating 2 cookies)

TABLE 5 Differential Diagnosis of Eating Disorders

Gastrointestinal disorders
Inflammatory bowel disease
Celiac disease
Infectious diseases
Chronic infections (human immunodeficiency virus infection, tuberculosis, others)
Endocrine disorders
Hyperthyroidism (hypothyroidism)
Diabetes mellitus
Other endocrine disorders (eg, hypopituitarism, Addison disease)
Other psychiatric disorders
Obsessive-compulsive disorder and anxiety disorders
Substance abuse
Other disorders
Central nervous system lesions (including malignancies)
Other cancers
Superior mesenteric artery syndrome (more commonly a consequence of severe weight loss)

of all children and adolescents with eating disorders may not fully meet all DSM-IV-TR criteria for AN or BN because they do not articulate body-image dissatisfaction or because their inadequate nutrition is manifest by growth failure rather than weight loss to less than 85% of expected weight.[63,64] These patients experience the same medical and psychological consequences of their disorders as do patients who meet criteria for AN or BN. Indeed, because the sequelae of weight loss (or failure to gain weight appropriately) may have even more worrisome implications for younger patients, relaxation of the diagnostic criteria for children and adolescents has been proposed in the development of the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* to facilitate earlier diagnosis and treatment.[61]

INITIAL EVALUATION OF THE PATIENT WITH DISORDERED EATING

When screening raises suspicion of an eating disorder, initial evaluation includes establishing the diagnosis, evaluating medical and nutritional status, determining severity, and performing an initial psychosocial evaluation. This comprehensive evaluation is often performed in the pediatric primary care setting, and primary care clinicians who feel competent and comfortable in performing this assessment are encouraged to do so. Others should refer to appropriate medical subspecialists and mental health personnel to ensure that a complete evaluation is performed. A differential diagnosis for the adolescent with symptoms of an eating disorder can be found in Table 5.

Because eating disorders can affect every organ system and the medical complications can be serious or even life-threatening, a comprehensive history should be taken and a comprehensive physical examination should be performed. The most frequently seen medical complications are listed in Table 6 and are detailed in the following section.

Most laboratory results will be normal in patients with eating disorders; however, normal laboratory results do not exclude serious illness or medical instability in these patients. Still, an initial laboratory assessment should include a complete blood cell count; measurement of serum electrolytes, calcium, magnesium, and glucose; liver function tests; urinalysis; and measurement of thyrotropin level. Additional studies (eg, urine pregnancy test, serum luteinizing and follicle-stimulating hormones, serum prolactin, and se-

TABLE 6 Medical Complications That Result From Eating Disorders

General
Dehydration
Hypokalemia
Hypomagnesemia
Hyponatremia
Irreversible cardiomyopathy and myositis (ipecac toxicity)
Amenorrhea and menstrual irregularities
Low bone mineral density; osteoporosis
Cognitive deficits
Mood symptoms
Obsessive/compulsive symptoms
Suicide
Caloric restriction and weight loss
Inability to maintain body temperature
Prolonged corrected QT interval or increased QT dispersion (uncommon but may predispose patient to sudden death)
Dysrhythmias (including supraventricular beats and ventricular tachycardia, with or without exercise)
Other electrocardiographic abnormalities
Mitral valve prolapse
Pericardial effusions
Delayed gastric emptying and impaired gastrointestinal tract motility
Constipation
Bloating; postprandial fullness
Hypoglycemia
Hypercholesterolemia
Abnormal liver function test results
Sterile pyuria
Anemia, leukopenia; thrombocytopenia
Sick-euthyroid syndrome
Growth retardation
Cortical atrophy
Vomiting-related
Hypochloremic metabolic alkalosis (vomiting)
Esophagitis
Gastroesophageal reflux
Dental erosions
Mallory-Weiss tears
Esophageal or gastric rupture (rare)
Aspiration pneumonia (rare)
Laxative-related
Hyperchloremic metabolic acidosis (laxative abuse)
Hyperuricemia
Hypocalcemia
Fluid retention (may gain up to 10 lb in 24 h) with laxative withdrawal
Refeeding
Diaphoresis and night sweats
Polyuria and nocturia
Peripheral edema
Refeeding syndrome

rum estradiol) may be indicated for patients with amenorrhea. Bone densitometry, using age-appropriate software, should also be considered for those with amenorrhea for more than 6 to 12 months. Other studies including erythrocyte-sedimentation rate, screening for celiac disease, or radiographic imaging, such as computed tomography or MRI of the brain or studies of the upper or lower gastrointestinal system, should be considered if there are uncertainties about the diagnosis. An electrocardiogram should be performed for any patient with cardiovascular signs or symptoms, for any patient with electrolyte abnormalities, or for any patient with significant purging or weight loss.

The initial mental health assessment should include an evaluation of the patient's obsession with food and weight, his or her understanding of the diagnosis, and his or her willingness to receive help. The patient's social functioning at home, in school, and with friends should be assessed. Psychiatric comorbidity is common with eating disorders and is often previously undiagnosed.[34,65] The pediatrician should identify other potential psychiatric diagnoses (such as depression, anxiety, or obsessive-compulsive disorder), which may be a cause or consequence of disordered eating. Use of tobacco, alcohol, or illicit drugs or misuse of prescription or over-the-counter medications may also complicate the management of eating disorders. Suicidal ideation and history of physical or sexual abuse or violence should also be assessed. Suicide attempts and completed suicide are relatively common, particularly for patients who have binge/purge or purging behavior and are a major contributor to eating disorder–associated mortality. Death from suicide is 50 times more likely in patients with AN,[66] and 25% to 35% of patients with BN report a history of attempted suicide.[34]

The parents' reaction to the illness should also be assessed. Parental indifference or denial of the problem or inconsistent views about treatment may affect the course of the illness and recovery.

Determining where and by whom the patient will be treated is an important and practical component of the initial evaluation. Patients with limited nutritional, medical, and psychological dysfunction can be managed in the pediatrician's office in conjunction with outpatient nutrition and mental health support. Patients who are more ill often require more intensive services, ideally delivered by a specialized multidisciplinary team, and sometimes in day-treatment, hospital, or residential settings.

MEDICAL COMPLICATIONS IN PATIENTS WITH EATING DISORDERS

Medical complications associated with eating disorders are listed in Table 5, and details of these complications have been described in many reviews.[32,33,67–74] Significant complications are seen in both outpatients and inpatients.[75] Most of the medical complications of eating disorders resolve with refeeding and/or resolution of purging.[70] However, there is increasing concern that some complications—particularly growth retardation, structural brain changes, and low bone mineral density—may, with time, become irreversible.[72] Malnutrition underlies many of the somatic symptoms seen initially, and these changes are often adaptive to the associated energy deficits. Over time, adaptation fails and signs and symptoms reflect the inability to compensate for inadequate nutrition. Metabolic rate decreases, body temperature can no longer be maintained, and nearly every organ system is compromised.[70,75,76]

Common cardiovascular signs and symptoms include orthostasis with blood pressure and/or pulse changes, bradycardia, and poor peripheral perfusion characterized by cold extremities, delayed capillary refill, and sometimes

acrocyanosis. Conduction abnormalities may occur as a result of myocardial atrophy and are thought to be the most common proximal cause of death with AN. Repolarization abnormalities, characterized by QTc prolongation and/or increased QT dispersion, are reported with widely variable prevalence and seem to be more frequent in older patients and with increasing duration of illness.[77] Repolarization abnormalities are potentially life-threatening and should be managed aggressively. Pericardial effusion, a functional mitral valve prolapse, myocardial dysfunction, and emetine (ipecac-related) cardiomyopathy are all seen less frequently. Congestive heart failure can occur during refeeding, particularly in the setting of electrolyte abnormalities.[72,73,78]

Gastrointestinal complaints are common and sometimes precede diagnosis of the eating disorder. Delayed gastric emptying and increased intestinal transit time often contribute to subjective descriptions of bloating and postprandial fullness, which can further compromise nutritional restoration. In patients who vomit, symptoms of gastroesophageal reflux are common, and upper gastrointestinal bleeding sometimes occurs. Severe bleeding secondary to Mallory-Weiss tears of the esophagus is rare. Constipation is common and often difficult to manage. Nutritional strategies, stool softeners, or polyethylene glycol 3350 (Miralax) are the treatments of choice; stimulant laxatives should be avoided. Rectal prolapse sometimes occurs in the setting of constipation and/or laxative abuse. Hepatic transaminase levels are often elevated as a consequence of malnutrition and are not usually indicative of viral hepatitis. Hypertrophy of the salivary glands often occurs and may be a clue to binge-eating and/or vomiting. Esophageal or gastric rupture are catastrophic but rare complications that usually occur during refeeding.[73]

Fluid and electrolyte abnormalities may occur as a result of purging or with increasing cachexia. Dehydration can be seen in any patient with an eating disorder and can sometimes lead to orthostatic symptoms, presyncope, or syncope. Chronic dehydration and the body's effort to conserve water may induce a pseudohyperaldosteronism, which also leads to hypokalemia. However, significant deficits in total body potassium and the associated risk of arrhythmia may exist even with a normal serum potassium level. Patients with vomiting may have a hypochloremic metabolic alkalosis because of chronic loss of hydrochloric acid. Patients who abuse laxatives may have a hyperchloremic metabolic acidosis related to bicarbonate wasting. Dilutional hyponatremia can be seen in patients who "water load" instead of eating or to misrepresent their weight at outpatient visits. Hypomagnesemia that results from inadequate intake is associated with sudden cardiac death, may interfere with potassium repletion in patients who are hypokalemic, and sometimes contributes to refeeding syndrome.[70] Edema, sometimes significant, may be seen as a result of hypoproteinemia, during refeeding, or in association with laxative abuse.[70]

Endocrine dysfunction is common and includes hypothyroidism, hypercortisolism, and disturbances of the HPA axis, which result in hypogonadotropic hypogonadism, luteal phase abnormalities, and anovulation. Euthyroid-sick syndrome (low free thyroxine, normal thyrotropin) is the most common thyroid abnormality and is reversible with refeeding. Supplemental thyroid hormone is not indicated. Activation of the HPA axis has been clearly demonstrated. In addition to its deleterious effects on growth, thyroid function, and the reproductive system, HPA hyperactivity also contributes to the appetite suppression and physical overactivity that characterize eating disorders.[79] Hypothalamic suppression causing amenorrhea is attributable not only to weight loss but also to physical overactivity, emotional stress, and the metabolic changes associated with acute energy deficits[70,75]; it sometimes precedes weight loss.[70] Hypothalamic secretion of gonadotropins reverts to a prepubertal pattern that reverses with refeeding.[70] Amenorrhea is an important marker for increased risk of low bone mineral density and osteoporosis (discussed in a later paragraph),[80–83] and an intriguing recent report suggested that amenorrhea is also associated with the cognitive impairments seen with AN.[84]

Common skin changes include lanugo, dry scaly skin, and yellow discoloration related to carotenemia. Acrocyanosis can be seen when perfusion is poor. Hair and nail changes are often seen as well, and angular stomatitis may be related to either vomiting or vitamin deficiencies.[70]

Growth retardation, short stature, and pubertal delay may all be seen in prepubertal and peripubertal children and adolescents with eating disorders.[75,85] Many endocrine abnormalities contribute to this growth failure; abnormal thyroid function, abnormal adrenal function, low levels of sex steroids, and uncoupling of growth hormone from insulin-like growth factor 1 (IGF-1) have all been implicated.[72] Catch-up growth has been inconsistently reported in the literature; younger patients may have greater and more permanent effects on growth.[72,86]

Low bone mineral density is a frequent complication of eating disorders in both male and female patients. It is worrisome not only because of the increased risk of pathologic fractures

but also because of its potential to be irreversible and compromise skeletal health across the entire life span. The pathophysiology of abnormal bone mineralization in the eating disorders is likely to be multifactorial; proposed mechanisms include deficiencies of gonadal steroids (estrogen and/or testosterone), deficiencies of calcium and vitamin D, reduction in lean muscle mass and its mechanical effects on bone, and excesses of endogenous glucocorticoids related to hyperactivity of the HPA axis. The reversibility of skeletal changes is unclear and probably varies on the basis of disease severity, the timing of illness and recovery, and perhaps genetic factors. Because adolescence is a critical period for bone mineralization, younger patients with AN are at higher risk of skeletal changes than are older patients. Treatment strategies, such as supplemental estrogen, bisphosphonates, calcium, and vitamin D replacement, have not been shown to be consistently effective, are not a substitute for nutritional recovery, and are not recommended for routine use.[72,87,88]

Volume deficits in both gray and white matter of the brain and associated increases in the cerebrospinal fluid space occur with weight loss in AN and are proportional to weight loss. Brain changes may be associated with elevated cortisol concentrations related to HPA-axis dysfunction, analogous to changes now being reported in other psychiatric disorders such as posttraumatic stress disorder.[89] Cognitive impairment has been demonstrated across the wide range of neuropsychological domains but does not seem to be directly proportional to structural brain changes.[84] Functional imaging studies of the brain show decreases in both global and localized brain activity, but it is unknown whether these decreases precede or are a consequence of weight loss or whether they are reversible.[90] Normalization of white matter occurs with refeeding; however, gray matter changes seem to persist despite weight recovery.[84,89]

TREATMENT CONTINUUM FOR CHILDREN AND ADOLESCENTS WITH EATING DISORDERS

Most adolescent patients with eating disorders will be treated in outpatient settings. Pediatricians play an important role in the management of these patients, assessing treatment progress, screening for and managing medical complications, and coordinating care with nutrition and mental health colleagues. Some pediatricians in primary care practice will feel comfortable in coordinating care; others will choose to refer some or all patients with eating disorders to those with special expertise. Depending on the availability of local resources, these providers may be a specialty eating disorders program, an adolescent medicine specialist, a psychiatrist, or another mental health provider.[32,91]

Collaborative Outpatient Care

Most children and adolescents with eating disorders will be managed in an outpatient setting by a multidisciplinary team coordinated by a pediatrician or medical subspecialist with expertise in the care of children and adolescents with eating disorders. Pediatricians generally work with nursing, nutrition, and mental health colleagues in provision of the medical, nutrition, and mental health care required by these patients.

It is generally accepted that medical stabilization and nutritional rehabilitation are the most important determinants of short-term outcomes and are essential for correcting cognitive deficits to allow for effective mental health interventions. Components of nutritional rehabilitation required in the management of patients with eating disorders have been presented in several reviews.[32,33,92–95] In the United States, oral refeeding is clearly the preferred modality for nutritional rehabilitation. However, for patients who are unwilling or unable to eat, supplements or nasogastric feeding may be life-saving.

Meals and snacks generally are reintroduced or improved in a stepwise manner for those with AN, which leads, in most cases, to an eventual intake of 2000 to 3000 kcal (or more) per day and a weight gain of 0.25 to 1 kg per week. Smaller, more frequent meals; increasing the caloric density of foods; and substituting nutrient fluids (eg, fruit juice) for water can sometimes help patients overcome the postprandial fullness and psychological barriers associated with the substantial increase in caloric intake that is required. Patients with abdominal complaints from acquired nutritionally mediated lactase deficiency may benefit from supplemental lactase. Meals are changed to ensure ingestion of 2 to 3 servings of protein per day. Daily fat intake should be slowly shifted toward a goal of 30 to 50 g per day. The stereotypical and obsessional eating habits favored by many patients with eating disorders and the observation that similar levels of weight loss and malnutrition can lead to dramatically different medical consequences suggest that deficiencies of specific micronutrients may share responsibility with protein-calorie malnutrition for the medical consequences in eating disorders.[70] Food variety should be encouraged, and a multivitamin should be recommended. Behavioral interventions are often required to encourage reluctant (and often resistant) patients to meet necessary caloric intake and weight-gain goals.[96–99]

Ranges for treatment goal weight should be individualized and based on age, height, pubertal stage, premorbid

weight, and previous growth trajectory. Furthermore, for growing children or adolescents, the goal weight range should be reevaluated at regular intervals (eg, every 3 to 6 months) on the basis of changing age and height. In postmenarcheal girls, resumption of menses provides an objective measure of biological health[100]; in 1 recent study, resumption of menses occurred at a mean BMI percentile of 27; 75% of the girls resumed menstruating once they had achieved and sustained approximately the 40th percentile for BMI.[101] Resumption of menses can also be used to refine the treatment goal weight.

Family-Based ("Maudsley") Therapy

Over the past decade, specialized eating disorder–focused family-based interventions, based on work originally performed at the Maudsley Hospital in London, have gained attention in the treatment of adolescent AN because of promising short-term and long-term outcomes. Although the etiologic underpinnings of this treatment approach have lost much of their support over time (ie, it is no longer believed that eating disorders are caused mainly by family dysfunction), family-based interventions, nevertheless, remain an effective and evidence-based treatment strategy for adolescent AN in both open trials and randomized controlled studies.[102–105] Family-based interventions are typically described as having 3 phases. In the first phase, parents, supported by the therapist, take responsibility to make certain that their adolescent is eating adequately and limiting other pathologic weight-control behaviors. In the second phase, substantial weight recovery has already occurred, and the adolescent is helped to gradually resume responsibility for his or her own eating. In the final phase of treatment, weight has been restored, and the therapy shifts to address the more general issues of adolescent development and how they may have been derailed by the eating disorder.[102] A manual for providers[106] and a family-support manual[107] are now available. Unfortunately, family-based treatment by experienced providers is not available in all communities. Nevertheless, the essential principles of family-based treatment can still be encouraged by community providers in their work with patients and families.[105] Family-based treatment may not be suitable for all patients; caution has been advised for families in which there is parental psychopathology or hostility toward the affected child, for older patients, or for patients who are the most medically compromised.[102,104] Additional randomized controlled studies of family-based treatment, including studies of long-term outcomes, are still needed. Family-based approaches are now being evaluated for the treatment of BN as well.[108]

Treatment of BN in adolescents has been poorly studied, and there is little evidence to guide treatment recommendations. For adults, BN-focused cognitive behavioral therapy is the treatment of choice. Pharmacotherapy (see "Pharmacotherapy") has been helpful as well.

Day-Treatment Programs

Day-treatment programs (day hospitalization, partial hospitalization) have been developed to provide an intermediate level of care for patients with eating disorders who require more than outpatient care but less than 24-hour hospitalization.[109–112] These programs have been used in an attempt to prevent the need for hospitalization; in some cases, they are used as a "step-down" from inpatient to outpatient care. Day-treatment programs are less costly and more accessible than traditional hospitalization. In addition, they allow for more family and social support and for recovery to occur in a more naturalistic environment that may be more generalizable.[109] Day treatment typically involves 8 to 10 hours of care (including meals, therapy, groups, and other activities) by a multidisciplinary staff 5 days/week. Evaluation of day-treatment programs has been characterized by small samples and the difficulty in undertaking randomized controlled trials.[113] Still, short-term outcomes have generally been reported to be good.[110,113,114] A recent study that used a range of outcome measures, including BMI and measurement of binge-purge behavior, demonstrated day treatment to be highly effective in the treatment of both restrictive and binge-purge AN and BN. Furthermore, these results were sustained or improved over 18 months of follow-up.[113]

Hospital-Based Treatment

Hospital-based treatment for eating disorders is less common when intensive outpatient or day-treatment programs are available. Hospitalization is much more frequently required for adolescent patients with AN than for patients with BN. Criteria for hospitalization of children and adolescents with eating disorders have been enumerated by the Society for Adolescent Medicine and are listed in Table 7.[32] Similar criteria are endorsed in the American Psychiatric Association's practice guideline for the treatment of patients with eating disorders[33] and by other organizations.[115] These criteria acknowledge that hospitalization may be required because of medical or psychiatric needs or when there is failure of outpatient treatment to achieve medical, nutritional, or psychiatric goals. Unfortunately, many third-party payers in the United States do not adhere to these criteria and make it difficult for some children and adolescents with eating disorders to receive the

TABLE 7 Criteria for Hospital Admission for Children, Adolescents, and Young Adults With Eating Disorders[32]

AN
<75% ideal body weight or ongoing weight loss despite intensive management
Refusal to eat
Body fat < 10%
Heart rate < 50 beats per min daytime; <45 beats per min nighttime
Systolic pressure < 90 mm Hg
Orthostatic changes in pulse (>20 beats per min) or blood pressure (>10 mm Hg)
Temperature < 96°F
Arrhythmia
BN
Syncope
Serum potassium concentration < 3.2 mmol/L
Serum chloride concentration < 88 mmol/L
Esophageal tears
Cardiac arrhythmias including prolonged QTc
Hypothermia
Suicide risk
Intractable vomiting
Hematemesis
Failure to respond to outpatient treatment

recommended level of care.[116,117] Children and adolescents have the best prognosis if their disease is treated rapidly and aggressively (an approach that may not be as effective for adults with a more long-term, protracted course).[91] Hospitalization, when indicated, allows for medical stabilization, adequate weight gain, and establishment of safe and healthy eating habits and improves the prognosis for children and adolescents. Discharge of hospitalized patients too soon often results in medical complications, a worse clinical course, and readmission. In 1 study, patients with AN who were discharged while still underweight had a 50% readmission rate compared with a rate of less than 10% for patients who had reached at least 90% of their recommended average body weight before discharge.[118]

The pediatrician involved in the treatment of hospitalized patients must be prepared to provide nutrition via a nasogastric tube or even intravenously when necessary. In hospitalized male adolescents, supplemental nighttime nasogastric feedings have been shown to significantly increase both weight gain and improvement in BMI compared with oral refeeding alone.[119]

Refeeding syndrome may occur in severely malnourished patients, particularly in the setting of aggressive nutritional rehabilitation. Refeeding syndrome refers to a constellation of metabolic, cardiovascular, neurologic, and hematologic complications primarily related to shifts of phosphate from extracellular to intracellular spaces in the setting of total body phosphorus depletion. The syndrome is most common in hospitalized patients during the first week of hospitalization and patients who are receiving supplemental enteral or parenteral nutrition. Cautious refeeding, careful monitoring of serum electrolyte, magnesium, phosphorus, and glucose levels, and a low threshold for phosphorus supplementation prevent the development of refeeding syndrome.[71,72,120–123] Refeeding syndrome is unusual after the first 2 weeks of nutritional rehabilitation or in patients being treated in the outpatient setting.

Pharmacotherapy

No medications have been approved by the US Food and Drug Administration for the treatment of AN.[124] Pharmacotherapy is sometimes prescribed but is typically targeted at comorbid symptoms of depression and anxiety. Selective serotonin-reuptake inhibitors (SSRIs) are most often used but may not be effective in severely malnourished patients. There is also limited evidence for the use of SSRIs for relapse prevention in AN.[125] In recent case reports and open-label trials, atypical neuroleptic agents, predominantly olanzapine (Zyprexa), have been noted to improve both weight gain and dysfunctional thinking in patients with AN.[126] A recently completed randomized, double-blind, placebo-controlled trial in adults showed a significant increase in weight gain in those who were taking olanzapine and a concomitant decrease in obsessive symptoms, although the effect size was modest.[127] Further evaluation of the effectiveness of these agents is underway, and caution is warranted because of the risk of developing insulin resistance and metabolic syndrome.

In contrast to AN, several pharmacologic agents have been demonstrated to be effective for the treatment of BN. Although only fluoxetine has been approved by the Food and Drug Administration, other SSRIs, serotonin/norepinephrine-reuptake inhibitors (eg, venlafaxine), and tricyclic antidepressants have also been shown to decrease binge-eating and purging in BN.[124,128] Topiramate has been shown to significantly decrease binge-eating and may be an option for patients who do not respond to or are not able to tolerate SSRIs.[129] Other drugs, including naltrexone and ondansetron (Zofran), are being used with some success in BN, although data are lacking to recommend their use more broadly.[130]

Hormonal supplementation, although capable of restoring menstruation, has not been shown to reliably improve bone mineral density and is not a substitute for nutritional rehabilitation and restoration of positive energy balance.

PROGNOSIS

The prognosis of eating disorders in adolescents has varied widely in the literature, and outcomes have depended on methodology, definitions of recovery, and duration of follow-up in the studies reported.[131] Adolescent outcomes are significantly better than the outcomes reported in adults. Longitudinal reports reflect a more optimistic and less hopeless outcome; followed over time, the majority of patients fully recover, and an even

larger proportion have a behavioral cure (normal eating, normal weight, and resumption of menses). However, these results accrue only after more than 10 years of follow-up; therefore, patients, their families, and clinicians must be prepared to remain engaged in what may sometimes be a protracted treatment process.[132–134]

Strober et al[132] conducted an important study in which 95 people who had been hospitalized for AN as adolescents were followed for 10 to 15 years. By the end of follow-up, 86.3% had achieved partial or complete recovery, and there were no deaths. However, the median time to partial recovery was 57.4 months, and the median time to full recovery (met by >75% of the study population) was 79.1 months. A study from Germany produced similar findings; at 10-year follow-up, 69% of the patients (including 7 boys) had achieved full recovery, and there were no deaths. Again, however, the course was protracted and the authors pointed out a high rate of residual psychiatric disorders even after full recovery from AN.[135]

Patients with an earlier age of onset seem to have a better prognosis.[85,134] Other characteristics associated with a better prognosis include shorter duration of symptoms and a better parent-child relationship. Purging behavior, physical hyperactivity, more significant weight loss, and disease chronicity are all associated with a less favorable prognosis.[134] Even after recovery, there are high rates of residual psychiatric illness—predominantly depression and anxiety—that persist.[133,136,137] A meta-analysis of 119 AN outcome studies showed little improvement in the success of treatment over the 5 decades reviewed.[133]

Mortality rates for adolescents with both AN and BN are lower than those that have historically been reported.[133,134] In a recent meta-analysis, the mortality rate among adolescents with AN was reported to be 1.8% compared with a mortality rate of 5.9% when adults and adolescents were considered together.[134] Mortality, when it does occur, is most often attributable to the complications of starvation or to suicide.[66]

PEDIATRICIANS' ROLE IN PREVENTION AND ADVOCACY

Efforts to prevent eating disorders can take place both in practice and community settings, such as schools. Primary care pediatricians can help families and children learn to apply the principles of proper nutrition and physical activity and to avoid an unhealthy emphasis on weight and dieting.[138] In addition, pediatricians can screen to detect the early onset of disordered eating and be careful to avoid seemingly innocuous statements (such as "you could stand to lose a little weight") that are sometimes reported by patients to have triggered the onset of their eating disorder. At the community level, there is general agreement that changes in the cultural approaches to weight, dieting, and body image will be required to decrease the growing numbers of children and adolescents at risk of developing eating disorders. This cultural shift is made more challenging by the increasing prevalence of obesity and the competing responsibility to address its health risks as well.[15]

A variety of successful programs for preventing eating pathology have been developed for various settings.[139] The largest effect sizes were seen in programs targeted at high-risk populations, in programs that were interactive rather than didactic, and in programs aimed at older adolescents. Content varied even in the most successful programs, which suggests that a variety of approaches may be effective. Multisession programs were more effective than single-session programs,[140] and there has even been some concern that single-session programs may be counterproductive.[141–146] An important question currently being asked is whether we can work simultaneously toward the prevention of eating disorders and obesity.[15]

Reimbursement issues continue to limit the access of many patients with eating disorders to appropriate services. Availability of mental health services, lack of mental health parity, and service "carve-outs" all have been barriers to patients and families who seek clinically necessary treatment and seem to be disproportionately problematic for patients with eating disorders. Despite evidence of its effectiveness, family-based treatment is not available in many communities. Through advocacy, pediatricians can help support health care reform efforts that will ensure that children and adolescents with eating disorders are able to receive necessary care.

GUIDANCE FOR PEDIATRICIANS

1. Pediatricians need to be knowledgeable about the risk factors and early signs and symptoms of disordered eating and eating disorders.
2. When counseling families on preventing obesity, pediatricians should focus on healthy eating and building self-esteem while still addressing weight concerns. Care needs to be taken not to inadvertently enable excessive dieting, compulsive exercise, or other potentially unhealthy weight-management strategies.
3. Pediatricians should be encouraged to calculate and plot weight, height, and BMI by using age- and gender-appropriate charts and assess menstrual status in girls at annual health supervision visits.
4. Pediatricians should screen patients for disordered eating

and related behaviors and be prepared to intervene when necessary.

5. Pediatricians should monitor or refer patients with eating disorders for medical and nutritional complications.
6. Pediatricians need to be familiar with treatment resources in their communities so that they can coordinate or facilitate multidisciplinary care.
7. Pediatricians can play a role in primary prevention during office visits and through school-based and community interventions with a focus on education, early screening, and advocacy.
8. Pediatricians are encouraged to advocate for legislation and policy changes that ensure appropriate services for patients with eating disorders, including medical care, nutritional intervention, mental health treatment, and care coordination, in settings that are appropriate for the severity of the illness.

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