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Pediatrics 2014;134;582; originally published online August 25, 2014;
DOI: 10.1542/peds.2014-0194

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http://pediatrics.aappublications.org/content/134/3/582.full.html

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Eating Disorders in Children and Adolescents: State of the Art Review

This is the 3rd in our series on Adolescent Health.

abstract

Despite their high prevalence, associated morbidity and mortality, and available treatment options, eating disorders (EDs) continue to be underdiagnosed by pediatric professionals. Many adolescents go untreated, do not recover, or reach only partial recovery. Higher rates of EDs are seen now in younger children, boys, and minority groups; EDs are increasingly recognized in patients with previous histories of obesity. Medical complications are common in both full and subthreshold EDs and affect every organ system. No single cause of EDs has emerged, although neurobiological and genetic predispositions are emerging as important. Recent treatment paradigms acknowledge that they are not caused by families or chosen by patients. EDs present differently in pediatric populations, and providers should have a high index of suspicion using new *Diagnostic and Statistical Manual, 5th edition* diagnostic criteria because early intervention can affect prognosis. Outpatient family-based treatment focused on weight restoration, reducing blame, and empowering caregivers has emerged as particularly effective; cognitive behavioral therapy, individual therapy, and higher levels of care may also be appropriate. Pharmacotherapy is useful in specific contexts. Full weight restoration is critical, often involves high-calorie diets, and must allow for continued growth and development; weight maintenance is typically inappropriate in pediatric populations. Physical, nutritional, behavioral, and psychological health are all metrics of a full recovery, and pediatric EDs have a good prognosis with appropriate care. ED prevention efforts should work toward aligning with families and understanding the impact of antiobesity efforts. Primary care providers can be key players in treatment success. *Pediatrics* 2014;134:582–592

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KEY WORDS

eating disorders, anorexia nervosa, bulimia nervosa, family-based treatment

ABBREVIATIONS

AN—anorexia nervosa
BED—binge eating disorder
BN—bulimia nervosa
CBT—cognitive behavioral therapy
DSM-5—*Diagnostic and Statistical Manual, 5th edition*
ED—eating disorder
EDNOS—eating disorder not otherwise specified
FBT—Family-based treatment
PCP—primary care provider
RCTs—randomized controlled trials

Dr Campbell conducted the initial literature review and drafted and revised the initial manuscript; Dr Peebles further contributed to the literature review and reviewed and revised the manuscript; and both authors approved the final manuscript as submitted.

www.pediatrics.org/cgi/doi/10.1542/peds.2014-0194

doi:10.1542/peds.2014-0194

Accepted for publication Apr 7, 2014

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PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

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FINANCIAL DISCLOSURE: The authors have indicated they have no financial relationships relevant to this article to disclose.

FUNDING: No external funding.

POTENTIAL CONFLICT OF INTEREST: The authors have indicated they have no potential conflicts of interest to disclose.

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Despite their high prevalence, associated morbidity and mortality, and available treatment options, eating disorders (EDs) continue to be underdiagnosed by pediatric professionals. Many adolescents go untreated, do not recover, or reach only partial recovery. This article provides an update of the current ED literature in children and adolescents. Anorexia nervosa (AN), bulimia nervosa (BN), and other EDs are presented. The epidemiology, etiology and pathogenesis, clinical presentation, and diagnosis of EDs are reviewed. New diagnostic criteria from the *Diagnostic and Statistical Manual, 5th edition* (DSM-5), updates on complications of EDs, and advances in their evidence-based treatment are highlighted, as is the role that primary care providers (PCPs) have in positively affecting treatment outcomes and the prevention of EDs in children and adolescents.

EPIDEMIOLOGY

Pediatric EDs are more common than type 2 diabetes, and the epidemiology is changing, with higher rates of EDs in younger children, boys, and minority groups.[1–3] The lifetime prevalence of AN is between 0.5% to 2%,[4] with a peak age of onset of 13 to 18 years.[5] AN has a mortality rate of at least 5% to 6%,[6,7] the highest mortality rate of any psychiatric illness.[8] The lifetime prevalence of BN is higher at between 0.9% and 3%,[9,10] with an older age of onset of 16 to 17 years.[11] Although mortality rates in BN are estimated to be ∼2%,[12] the risk of lifetime suicidality and suicide attempts in BN are much higher.[6] On the basis of the criteria from the fourth edition of the DSM, most adolescents were diagnosed with EDs not otherwise specified (EDNOS), a group of heterogeneous disorders composed primarily of subthreshold AN or BN.[13] The estimated lifetime prevalence of EDNOS in adolescents is 4.8%.[14] Rates of medical complications in EDNOS are similar to full-threshold disorders.[15] Although female patients account for most ED diagnoses, males have accounted for 10% of ED cases over the past years,[4] with some studies reporting up to 25% of cases being male.[16] Furthermore, younger patients diagnosed with EDs are more likely to be boys, with a female to male ratio of 6 to 1, compared with a 10 to 1 ratio in adults.[3,17] Dieting behaviors are a risk factor for developing an ED and are highly prevalent; ∼50% of girls and 25% of boys report dieting during the past year.[18] Moreover, 30% of girls and 15% of boys had disordered eating behaviors severe enough to warrant medical evaluation, and 9% of girls and 4% of boys reported daily self-induced vomiting.[19]

ETIOLOGY

The exact etiology of EDs is unknown; there is thought to be an interface between genetic and biological predispositions, environmental and sociocultural influences, and psychological traits. Evidence continues to increase that EDs are heritable, with relatives of ED patients having 7 to 12 times greater risk of developing an ED.[20–22] Twin studies have estimated heritability of AN between 33% and 84% and BN between 28% and 83%.[4,22] Research is ongoing to identify specific chromosomes, genes, and proteins that may play a role in the development of AN and BN.[4] There are also neurobiological factors being studied in EDs, but it is uncertain whether they contribute to the development of EDs or result from the physiologic alterations caused by EDs.[23]

CLINICAL PRESENTATION

Adolescence is a critical period of development and a window of vulnerability during which EDs can develop. The explosive physical and cognitive development that occurs during this period lends itself to substantial differences in the presentation of EDs in children and adolescents; pediatricians are frequently the front-line providers diagnosing these disorders. An ED should be suspected in a patient of any weight who presents with weight loss, unexplained growth stunting or pubertal delay, restrictive or abnormal eating behaviors, recurrent vomiting, excessive exercise, trouble gaining weight, or body image concerns. Younger patients are likely to have atypical presentations; instead of rapid weight loss, they may present with failure to make expected gains in weight or height and may not endorse body image concerns or engage in binge eating or purging behaviors.[17,24] Boys and children and adolescents who are overweight or obese are at risk for delayed diagnoses and significant complications[3,25,26]; these populations require heightened vigilance by providers. Adolescents with chronic illnesses, especially insulin-dependent diabetes mellitus, are also at higher risk of developing ED behaviors and should be screened regularly.[27–31]

Screening tools, such as the brief SCOFF questionnaire,[32] although only validated in adults, are used in the primary care setting for ED screening in adolescents (Table 1). In addition, providers should evaluate all patients for high-risk behaviors, such as dieting or excessive exercise, and follow their growth trajectories and BMI to assess

TABLE 1 The SCOFF Questionnaire[a]

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 more than One stone (14 lb/6.3 kg) in a 3-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?

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

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for weight loss or failure to make appropriate gains. If an ED is suspected, it is important to obtain a comprehensive medical, family, and social history and a complete review of systems and to perform a thorough physical examination to evaluate for physical stigmata and medical complications of EDs (Table 2). Obtaining the history from both the patient and caregiver(s) is important; although time alone with the adolescent is recommended, history from caregiver(s) can be crucial in elucidating behaviors or cognitions that the adolescent may not report. In addition, the providers should always consider a complete differential diagnosis when evaluating a patient with a potential ED (Table 3).

DIAGNOSIS

New diagnostic criteria for EDs are published in the DSM-5, released in 2013.[33] Significant changes were made in an effort to improve the accuracy and precision of ED diagnoses, which will potentially allow for more targeted treatment. One major limitation of the fourth edition of the DSM was the diagnostic category of EDNOS, which accounted for the majority of ED diagnoses in most pediatric series.[34] EDNOS was a nonspecific diagnostic category that encompassed a wide spectrum of EDs, including subthreshold AN, subthreshold BN, and binge ED (BED).[14] This ambiguity led to misunderstandings of the clinical significance of the disorder and difficulty choosing the most effective therapy. To address these issues, the DSM-5 broadens the inclusion criteria for both AN and BN, BED is now a formal diagnosis, and other EDs have been further clarified.[35]

Adolescents with AN often present with dramatic weight loss or poor growth and may be preoccupied with food and weight. Restriction of entire food groups (ie, new-onset vegetarianism) or calories, and the development of food rituals are commonplace. They commonly refuse to eat foods they once enjoyed, avoid meals with family and friends, and overexercise in a rigid manner. Pubertal milestones such as linear growth or menstrual cycles are often affected.[5,16,24] DSM-5 criteria for AN consider expected weight and growth[36] in children and adolescents versus comparisons to population norms. They describe a restriction of energy intake relative to requirements, leading to a lower than expected body weight. In addition, behavioral criteria are considered equivalent to cognitive criteria, equating fear of weight gain to failure to gain weight in the face of low body weight or growth stunting.[5] Amenorrhea has been removed as a criterion because its use was never validated[36] and excluded males, premenarchal females, and adolescents who remain eumenorrheic despite low body weight.[37] Finally, body image distortion or an unusual focus on weight or shape are still included as

TABLE 2 History Questions and Physical Examination Findings in EDs

History	Physical Examination Findings
Past medical history	Anorexia nervosa and other restrictive disorders
Time course of weight loss, including minimum and maximum weight during adolescence.	Sinus bradycardia
Perceived goal weight/healthy weight	Cardiac arrhythmias including QT prolongation
Body image concerns	Orthostatic changes in pulse >20 or blood pressure >10
Dietary habits including 24-hr recall, history of restricting, binge eating, and/or purging	Hypotension
Exercise history	Hypothermia
Previous therapy	Dry, pale skin
Secretive behaviors	Orange discoloration of skin
Symptoms of systemic illnesses, such as inflammatory bowel disease, diabetes mellitus, celiac, lupus	Lanugo
Relevant review of systems: presyncope, syncope, headaches, fatigue, exercise intolerance, sleep disturbance, dry skin, increased shedding of hair/hair loss, cold intolerance, easy bruising, delayed wound healing, mood changes	Bruising/abrasions over spine
Family history	Acrocyanosis
Eating disorders, obesity, depression, anxiety, alcoholism or drug abuse, bipolar affective disorder, schizophrenia	Thinning scalp hair
Pubertal/menstrual history	Facial wasting
Menarche, last menstrual period, changes in regularity, duration	Cachexia
Timing of thelarche and pubarche if premenarchal	Atrophic breasts
Growth rate deceleration	Scaphoid abdomen
For males: history of decreased erections or nocturnal emissions	Dependent edema
Social history	Flat or anxious affect
Recent stressors, family, school, friends	BN and other purging disorders
Tobacco, alcohol, illicit drug use	Sinus bradycardia or cardiac arrhythmias including QT prolongation
History of physical or sexual abuse	Orthostatic changes in pulse >20 or blood pressure >10
Use of pro-eating disorder Web sites	Callouses or abrasions over knuckles due to self-induced vomiting
Mood changes	Parotid enlargement
Time with friends	Dental enamel erosions, caries, oral ulcerations
Engagement in fun activities	Mood lability
Recent stressors, family, school, friends	Scleral hemorrhage
	Palatal petechiae
	Loss of gag reflex

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TABLE 3 Differential Diagnosis of EDs

Endocrine disorders
Hyperthyroidism or hypothyroidism
Diabetes mellitus
Hypercortisolism
Adrenal insufficiency
Gastrointestinal disorders
Inflammatory bowel disease
Celiac disease
Infectious diarrhea
Immunodeficiency or chronic infections (ie, HIV, tuberculosis)
Psychiatric disorders
Depression
Obsessive compulsive disorder/anxiety
Substance abuse
Other disorders
Superior mesenteric artery syndrome
Malignancies
Central nervous system tumors (ie, prolactinoma)
Pregnancy
Excessive exercise/energy imbalance
Rheumatologic disease
Wilson's disease
Porphyria

criteria but are not required if the patient persistently fails to recognize the seriousness of his or her low body weight.

The hallmark of BN is recurrent episodes of binge eating accompanied by inappropriate compensatory behaviors. An objective binge episode involves eating more food in a discrete period of time than most people would eat, coupled with feeling a loss of control. DSM-5 criteria for BN require objective binge episodes and subsequent compensatory behaviors at least once per week for 3 months. Patients with BN may be of any weight and often have frequent weight fluctuations from fluid shifts. Caregivers or peers may notice the development of mood swings, surreptitious behaviors (ie, increased time in the bathroom after meals, hiding food), or periods of fasting or excessive exercise.[9,33]

The distinguishing feature between BED and BN is that episodes of binge eating are not associated with inappropriate compensatory behaviors. Patients with BED and BN display marked distress regarding binge eating and will often binge in secret. The frequency of recurrent episodes of binge eating was decreased in the DSM-5 similar to BN.[33,38]

Additional new categories in the DSM-5 with likely impact are avoidant restrictive food intake disorder, other specified feeding and EDs, and unspecified feeding and ED. "Avoidant restrictive food intake disorder is not uncommon in children[35,39] and comprises a variety of restrictive eating behaviors (ie, swallowing phobias, textural aversions) that do not involve a fear of weight gain or distorted cognitions but lead to significant physical and emotional impairment. Other specified feeding and EDs refers to atypical AN (normal-weight AN), subthreshold BN, purging disorder, and night eating syndrome. Unspecified feeding and ED comprises any other clinically significant EDs that do not fit the aforementioned categories.[33,39]

Complications

EDs can affect every organ system, and complications can occur at any weight.[16,24,40,41] It is important for providers to act quickly and decisively when they suspect an ED in all patients to avoid complications and the potential for chronicity.

CARDIOVASCULAR SYSTEM

Cardiac complications are common[15,40,42]; patients with EDs often present with bradycardia, hypotension, arrhythmias, and changes in heart rate variability.[15,40,43–46] Hypotension and postural changes in heart rate and blood pressure can result from decreased cardiac mass leading to systolic dysfunction, in addition to volume depletion and autonomic dysfunction.[16,24,42–44,47,48] Associated physical symptoms may include headache, presyncope, syncope, and exercise intolerance, although patients are frequently asymptomatic even in the face of profound vital sign instability.[16,24,48–50] Patients with chronic purging are at risk for cardiomyopathy, and up to one-third of hospitalized patients with AN have mitral valve prolapse and pericardial effusion.[41,43,46,51] Several small studies have demonstrated almost complete reversibility of both structural and functional derangement,[43,51] although ipecac abuse can lead to an irreversible cardiomyopathy.[52–55] Serious cardiac complications are not unique to AN but are also seen in other normal-weight EDs, particularly atypical AN and BN.[15,56–62]

Gastrointestinal

Gastrointestinal complications may occur secondary to malnutrition, vomiting, or binge eating. Complications secondary to malnutrition include delayed gastric emptying,[63] constipation, mild transaminitis, dyslipidemias, and superior mesenteric artery syndrome.[48,64–66] Patients who vomit risk esophagitis, and in severe cases, esophageal rupture and pneumomediastinum. They may present with reflux, hematemesis or parotid swelling.[67,68] Patients with AN typically report abdominal bloating, nausea, and postprandial fullness. Patients with binge eating behaviors are at risk for gastric dilation or rarely gastric rupture and pancreatitis.[69,70]

Electrolytes

Electrolyte disturbances occur in patients who engage in vomiting, laxative abuse, or diuretic use, with hypokalemia and hypophosphatemia being the most common.[71,72] Hypochloremic metabolic alkalosis may develop in patients who vomit, and hyperchloremic metabolic acidosis may develop in those who abuse laxatives.[73] Patients with malnutrition are at risk for refeeding syndrome during treatment, which includes hypophosphatemia,[74] hypokalemia,[73,75,76] and hypomagnesemia.[77]

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Endocrine

Patients with AN typically have hypothalamic suppression with low normal to low gonadotropin and sex hormone levels. Girls and boys may present with decelerated linear growth, pubertal delay, or pubertal regression, and menstrual dysfunction is common in females.[78–81] Low insulin-like growth factor-I and low to low-normal thyroxine and triiodothyronine levels are seen.[40,48] Sick euthyroid syndrome can be seen in severely malnourished patients and resolves with reversal of the malnourished state.[40,79,80] ED adolescents also risk reduced bone mineral density primarily due to poor nutritional intake, low BMI, and reduced fat mass.[45] Leptin plays a key role in energy homeostasis, and levels are low in malnourished states. A recent study demonstrated that if leptin levels are normalized, menstrual function and thyroid and bone markers improve in hypothalamic amenorrhea.[82]

Renal

ED Patients can develop dehydration and renal insufficiency due to severe fluid restriction or vomiting. Other renal abnormalities include pyuria and, less commonly, proteinuria and hematuria, which both clear with hydration and reversal of malnutrition.[40] Patients with AN may lose renal concentrating ability, which can result in high urine output and inaccurate specific gravity measurements on urinalyses.[83–86]

Hematologic

Bone marrow hypoplasia is seen in low-weight EDs, primarily leukopenia and anemia, with rare cases of thrombocytopenia. Leukopenia is not thought to increase infection risk, and all dyscrasias resolve with the reversal of malnutrition. It is important to evaluate for iron and vitamin B_{12} deficiency in anemic patients because these are easily reversed with supplementation. Finally, when evaluating for systemic illness, it is important to note that malnourished patients typically have a low sedimentation rate, usually below 5 mm/hr.[16,24,45]

Neurologic

Malnutrition significantly affects the brain in children and adolescents because of the dynamic changes that are occurring in cognitive and structural brain development during this period.[87–101] Severely ill patients with AN have been shown to have reduced brain tissue volume and impaired neuropsychological functioning.[87] One study demonstrated persistent gray-white matter deficits and cerebrospinal fluid levels elevations on magnetic resonance imaging of weight restored patients with AN.[102] However, several studies have demonstrated mixed results in the permanence of neurologic deficits.[87,103] Abnormalities in brain structure have been associated with low body weight and cortisol levels, whereas cognitive deficits are associated with menstrual function.[87]

Psychiatric

Psychiatric comorbidities are common in EDs but may be premorbid, comorbid, or present after recovery. Common disorders are depression, anxiety, obsessive-compulsive disorder, posttraumatic stress disorder, personality disorders, substance abuse disorders, and self-injurious behaviors.[104–110] In AN, the lifetime prevalence of depression and anxiety disorders is 50% to 68% and 30% to 65% respectively.[107] In BN the lifetime prevalence of mood disorders is 50% to 70%, anxiety disorders is 13% to 65%, substance use disorders is 25%, and personality disorders is 20% to 80%.[107]

TREATMENT MODALITIES

In 1995, the Society for Adolescent Medicine issued a statement that the treatment threshold for ED adolescents should be low because of potentially irreversible effects of EDs on growth and development, their mortality risk, and evidence that early treatment improves outcomes.[111] Children and adolescents are triaged to outpatient treatment, partial hospitalization, residential programs, and inpatient hospitalization based on severity of illness, duration of disease, safety considerations, and familial preferences. Treating patients in a home setting is preferred, but other models of care may be necessary and appropriate. In 2005, the American Academy of Pediatrics released criteria for inpatient hospitalization in patients with AN and BN (Table 4), which were reaffirmed in 2010.[16] This review focuses on emerging outpatient treatment modalities.

A paradigm shift in EDs is evident in newer treatment modalities. In older paradigms, patients with EDs were

TABLE 4 American Academy of Pediatrics Criteria for Inpatient Hospitalization in Eating Disorders

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

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thought to develop maladaptive eating behaviors in part because of overly controlling caregivers. This approach focuses on developing insight into the etiology of the disorder in psychodynamically informed individual treatment and/or cognitive behavioral therapy (CBT). These therapies focus on the patient's distorted body image and undue influence of weight and shape with a drive for thinness. A newer paradigm takes into account the biological and genetic contributions to EDs and views caregivers as critical allies in treatment.[112] In this approach, nutritional rehabilitation is considered an important factor in improving cognitions and is the primary initial focus of treatment rather than causation, with age-appropriate insight developing over time. This corresponds with the tenets of family-based treatment (FBT).[113,114]

Evidence for effective treatments in EDs in children and adolescents is growing but remains limited. Primary treatment modalities in pediatric AN are individual therapy, CBT, and FBT. FBT has the largest evidence base of any treatment of efficacy in adolescent and young adult AN populations with multiple clinical trials; there is also growing evidence that it is useful for children under age 12 with restrictive EDs.[113–116] FBT is superior to other currently available treatment methods, but its use is limited by a paucity of qualified practitioners. Although some differences between FBT and individual treatment diminished at long-term follow-up, FBT was more protective against relapse.[115] CBT has been studied in adolescents with BN and shows promise, but there is growing evidence that FBT is also effective[9,117–120]; additional research is needed comparing the 2 methods. CBT has also demonstrated efficacy in BED.[121] In subthreshold disorders, it is recommended that the patient be treated based on the full syndrome to which their disorder is most similar.[122] Children with textural aversions or swallowing phobias may also benefit from targeted occupational therapy.[123,124] Finally, translational research is underway targeting known deficits in neurocognitive processes, neurotransmitters affected in EDs, and neuroanatomic changes found on imaging studies to tailor treatment and improve treatment response in patients with EDs.[88–101]

Family-Based Treatment

In FBT an agnostic view is taken and the focus is not on the etiology of the disorder. Caregivers are not blamed but instead empowered to refeed their child back to health. The therapist and any other providers are considered consultants to caregivers in this work. Siblings are also supported in this treatment because they frequently have numerous concerns about their sick brother or sister.[125] Additionally, the disorder is externalized from the child to release blame toward the child for their disorder.[114] FBT progresses through 3 phases that target the goals of treatment in children and adolescents with EDs: physical, behavioral and psychological recovery. Phase I of FBT focuses on coaching the caregivers to refeed their child to recovery through specific therapeutic interventions. Food exposures are commonly used to target anxieties and aversions to certain foods or food groups; caregivers are encouraged to incorporate foods their children used to enjoy before the ED rather than to practice avoidance. Once the child is weight-restored, FBT progresses to Phase II, which focuses on gradually transferring developmentally appropriate control of eating back to the child or adolescent. Phase III works on relapse prevention and any other remaining developmental considerations, and then treatment termination. FBT typically is conducted over a 6- to 12-month time period.[114] Whereas in traditional treatments, fewer than half of AN patients fully recover within 2 to 5 years, a third partially recover, and 20% develop chronic illness,[126] 50% to 60% of patients in FBT achieve full remission within 1 year, another 25% to 35% partially recover (showing improvement but not full remission), and only 15% are nonresponsive to treatment. Thus, FBT is emerging as a first-line treatment in pediatric EDs.[113]

The role of the PCP during FBT is important to the success of treatment.[127] The PCP can serve as a consultant to both the caregiver(s) and the FBT provider, in addition to providing ongoing comprehensive medical assessments and monitoring. It is essential for the PCP to support the FBT provider in explaining the seriousness of the medical complications and prognosis of the ED and the importance of early and aggressive treatment, in addition to removing blame for the ED from either the caregiver(s) or the child. Finally, it is imperative that the PCP support complete weight restoration in the adolescent and full remission of the ED. Skills learned by the PCP in supporting FBT are useful regardless of the modality chosen for ED treatment because they help PCPs align with and remain respectful of caregivers.[113]

Pharmacotherapy

Pharmacologic agents are often used in patients with EDs, despite few studies demonstrating efficacy. There have been no published randomized controlled trials (RCTs) for antidepressant treatment in AN conducted in children and adolescents, and selective serotonin reuptake inhibitors and tricyclic antidepressants have not been shown to be better than placebo in weight gain or improvement in ED symptoms in adult AN. There are also no large RCTs on the use of atypical antipsychotics in the

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treatment of AN, although they may be useful in reducing anxiety and rigidity and improving early weight gain. There is no evidence to suggest that pharmacotherapy in AN should be first line, but it may play a role in individual patients resistant to treatment or with premorbid psychiatric conditions.[107,128–130] In BN, several RCTs in adults have found that antidepressants are effective in decreasing binge eating and purging symptoms. Specifically, fluoxetine has a strong evidence base and is approved by the Food and Drug Administration for use in adults with BN; thus far there is some evidence that the effects are similar in adolescents.[107] Other medications such as topiramate and ondansetron are currently being studied for use in adults with BN but are not routinely used.[107] In adults with BED, selective serotonin reuptake inhibitors seem to be effective in short-term reduction of binge eating but do not seem to be superior to CBT alone.[107]

RECOVERY GOALS

Although different metrics for recovery exist in the literature, most agree that behavioral recovery includes normalizing eating patterns and the return of flexibility in eating. Psychological recovery includes improved self-esteem and age-appropriate interpersonal, psychosocial, and occupational functioning. Weight and body shape should no longer have an undue influence on self-evaluation, and normal growth and pubertal patterns are restored.[131–135] Physical recovery includes full weight restoration, return of menses and/or pubertal progression, linear growth if expected, and reversal of most or all organ damage.[136,137] Nutritional restoration involves reaching a goal weight and the ability to eat a varied and balanced diet, but it is important to remember that a "maintenance weight" is often inappropriate in pediatric populations. Children and adolescents continue to grow and develop throughout puberty and into young adulthood.[113,138] Body composition and activity changes will mandate changes in weight even if a final adult linear height has been achieved. This is an important concept to highlight for parents and patients when working toward recovery.

The determination of goal weight in this population is complex: the provider typically works with a registered dietician experienced in treating EDs and must consider previous weight and linear growth trajectories if previously normal, genetic potential with the use of midparental height, and the median body weight using standardized Centers for Disease Control and Prevention BMI growth curves for height, age, and gender.[138,139] It is important to note that children and adolescents are not "little adults," and because of their hypermetabolic state, once nutrition is introduced, their caloric needs are high, typically between 3000 and 6000 kcal daily. They may remain hypermetabolic for up to 2 years,[113,140] so it is important not to reduce caloric intake prematurely once they reach their weight goal; instead, the treatment team can work on activity increases as development of muscle mass requires continuation of caloric goals. Recent studies have affirmed the safety of more aggressive nutrition approaches in ED treatment.[141–146] RDs can help reinforce these concepts; in newer approaches registered dieticians meet with caregivers to answer questions rather than with patients individually. Caregivers should avoid deferring all nutrition decisions to the registered dietician but rather use this consultation to better empower their efforts at refeeding.

PREVENTION

Developing effective primary and secondary prevention efforts is critical in EDs because of their high rate of future medical complications, psychiatric comorbidities, and risk of suicidality and relapse.[147] Several features of successful ED prevention programs from a recent meta-analysis of ED prevention programs are described in Table 5[147]; secondary prevention efforts may benefit from targeting caregivers as well.[148] Table 6 delineates 5 public health recommendations focused on shifting the focus from weight to the promotion of a healthy lifestyle for adolescents and their families to facilitate the creation of a positive body image in adolescents.[149] There are growing concerns that an antiobesity focus in pediatric public health may result in an increase in EDs, and future obesity prevention and treatment efforts should track ED cognitions as well as extreme weight control behaviors.[25,26]

TABLE 5 Features of Successful ED Prevention Programs

1. Target high risk adolescents over 15 y of age
2. Deliver intervention by trained individuals
3. Intervention content should include body acceptance and dissonance induction[a]

[a] Involves taking an active stance against the culturally mediated thin ideal, which leads to cognitive dissonance and a shift in belief systems toward an antithin ideal.

TABLE 6 Recommendations for Preventing ED and Obesity for Health Care Providers from Eating Among Teens

1. Inform adolescents that dieting, and particularly unhealthy weight-control behaviors, may be counterproductive. Instead, encourage positive eating and physical behaviors that can be maintained on a regular basis.
2. Do not use body dissatisfaction as a motivator for change. Instead, help teens care for their bodies so that they will want to nurture them through healthy eating, activity, and positive self-talk.
3. Encourage families to have regular, and enjoyable, family meals.
4. Encourage families to avoid weight talk. Talk less about weight and do more to help teens achieve a weight that is healthy for them.
5. Assume overweight teens have experienced weight mistreatment and address with teens and their families.

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CONCLUSIONS

EDs in children and adolescents are prevalent and have serious medical and psychological consequences. Children and adolescents have increased potential for long-term complications, thus it is imperative that providers recognize the risk factors and screen for EDs in their patients. Early recognition and aggressive treatment is needed to prevent complications and chronicity. Treatment efforts that focus on weight restoration, reducing blame, and actively incorporating caregivers and families have emerged as particularly effective. The evidence base for ED treatment modalities continues to grow, but to be successful, the treatment team, the family and the PCP must work in collaboration to promote remission and to prevent relapse in this population. Future research is needed to refine treatment in pediatric ED patients and to clarify the role of pharmacotherapy in the treatment of these disorders. Primary and secondary prevention of EDs are also important in improving the health of children, adolescents, and their families.

REFERENCES

1. Smink FR, van Hoeken D, Hoek HW. Epidemiology of eating disorders: incidence, prevalence and mortality rates. *Curr Psychiatry Rep.* 2012;14(4):406–414
2. Domine F, Berchtold A, Akre C, Michaud PA, Suris JC. Disordered eating behaviors: what about boys? *J Adolesc Health.* 2009; 44(2):111–117
3. Pinhas L, Morris A, Crosby RD, Katzman DK. Incidence and age-specific presentation of restrictive eating disorders in children: a Canadian Paediatric Surveillance Program study. *Arch Pediatr Adolesc Med.* 2011;165(10):895–899
4. Sigel E. Eating disorders. *Adolesc Med State Art Rev.* 2008;19(3):547–572, xi
5. Weaver L, Liebman R. Assessment of anorexia nervosa in children and adolescents. *Curr Psychiatry Rep.* 2011;13(2):93–98
6. Herpertz-Dahlmann B. Adolescent eating disorders: definitions, symptomatology, epidemiology and comorbidity. *Child Adolesc Psychiatr Clin N Am.* 2009;18(1): 31–47
7. Franko DL, Keshaviah A, Eddy KT, et al. A longitudinal investigation of mortality in anorexia nervosa and bulimia nervosa. *Am J Psychiatry.* 2013;170(8):917–925
8. Sullivan PF. Mortality in anorexia nervosa. *Am J Psychiatry.* 1995;152(7):1073–1074
9. Hoste RR, Labuschagne Z, Le Grange D. Adolescent bulimia nervosa. *Curr Psychiatry Rep.* 2012;14(4):391–397
10. Swanson SA, Crow SJ, Le Grange D, Swendsen J, Merikangas KR. Prevalence and correlates of eating disorders in adolescents. Results from the national comorbidity survey replication adolescent supplement. *Arch Gen Psychiatry.* 2011;68 (7):714–723
11. Sim LA, McAlpine DE, Grothe KB, Himes SM, Cockerill RG, Clark MM. Identification and treatment of eating disorders in the primary care setting. *Mayo Clin Proc.* 2010;85(8):746–751
12. Fichter MM, Quadflieg N. Twelve-year course and outcome of bulimia nervosa. *Psychol Med.* 2004;34(8):1395–1406
13. Eddy KT, Le Grange D, Crosby RD, et al. Diagnostic classification of eating disorders in children and adolescents: how does DSM-IV-TR compare to empirically-derived categories? *J Am Acad Child Adolesc Psychiatry.* 2010;49(3):277–287, quiz 293
14. Le Grange D, Swanson SA, Crow SJ, Merikangas KR. Eating disorder not otherwise specified presentation in the US population. *Int J Eat Disord.* 2012;45(5):711–718
15. Peebles R, Hardy KK, Wilson JL, Lock JD. Are diagnostic criteria for eating disorders markers of medical severity? *Pediatrics.* 2010;125(5). Available at: www.pediatrics.org/cgi/content/full/125/5/e1193
16. Rosen DS; American Academy of Pediatrics Committee on Adolescence. Identification and management of eating disorders in children and adolescents. *Pediatrics.* 2010; 126(6):1240–1253
17. Peebles R, Wilson JL, Lock JD. How do children with eating disorders differ from adolescents with eating disorders at initial evaluation? *J Adolesc Health.* 2006;39 (6):800–805
18. Neumark-Sztainer D, Wall M, Larson NI, Eisenberg ME, Loth K. Dieting and disordered eating behaviors from adolescence to young adulthood: findings from a 10-year longitudinal study. *J Am Diet Assoc.* 2011;111(7):1004–1011
19. Austin SB, Ziyadeh NJ, Forman S, Prokop LA, Keliher A, Jacobs D. Screening high school students for eating disorders: results of a national initiative. *Prev Chronic Dis.* 2008;5(4):A114
20. Lilenfeld LR, Kaye WH, Greeno CG, et al. A controlled family study of anorexia nervosa and bulimia nervosa: psychiatric disorders in first-degree relatives and effects of proband comorbidity. *Arch Gen Psychiatry.* 1998;55(7):603–610
21. Trace SE, Baker JH, Peñas-Lledó E, Bulik CM. The genetics of eating disorders. *Annu Rev Clin Psychol.* 2013;9:589–620
22. Strober M, Freeman R, Lampert C, Diamond J, Kaye W. Controlled family study of anorexia nervosa and bulimia nervosa: evidence of shared liability and transmission of partial syndromes. *Am J Psychiatry.* 2000;157(3):393–401
23. Kaye WH, Wierenga CE, Bailer UF, Simmons AN, Bischoff-Grethe A. Nothing tastes as good as skinny feels: the neurobiology of anorexia nervosa. *Trends Neurosci.* 2013; 36(2):110–120
24. Rosen DS. Eating disorders in children and young adolescents: etiology, classification, clinical features, and treatment. *Adolesc Med.* 2003;14(1):49–59
25. Pinhas L, McVey G, Walker KS, Norris M, Katzman D, Collier S. Trading health for a healthy weight: the uncharted side of healthy weights initiatives. *Eat Disord.* 2013;21(2):109–116
26. Sim LA, Lebow J, Billings M. Eating disorders in adolescents with a history of obesity. *Pediatrics.* 2013;132(4). Available at: www.pediatrics.org/cgi/content/full/132/4/e1026
27. Arigo D, Anskis AM, Smyth JM. Psychiatric comorbidities in women with celiac disease. *Chronic Illn.* 2012;8(1):45–55
28. Pinhas-Hamiel O, Levy-Shraga Y. Eating disorders in adolescents with type 2 and type 1 diabetes. *Curr Diab Rep.* 2013;13(2): 289–297
29. Quick VM, Byrd-Bredbenner C, Neumark-Sztainer D. Chronic illness and disordered eating: a discussion of the literature. *Adv Nutr.*2013;4(3):277–286

Downloaded from pediatrics.aappublications.org at McMaster University on September 2, 2014

30. Quick VM, McWilliams R, Byrd-Bredbenner C. Case-control study of disturbed eating behaviors and related psychographic characteristics in young adults with and without diet-related chronic health conditions. *Eat Behav.* 2012;13(3):207–213
31. Schmitt TL. Disordered eating in adolescent females with T1DM. *Nurse Pract.* 2012;37(9):38–42
32. Hill LS, Reid F, Morgan JF, Lacey JH. SCOFF, the development of an eating disorder screening questionnaire. *Int J Eat Disord.* 2010;43(4):344–351
33. Association AP. *DSM-5: Diagnostic and Statistical Manual of Mental Disorders.* 5th ed. Arlington, VA: American Psychiatric Association; 2013
34. Thomas JJ, Vartanian LR, Brownell KD. The relationship between eating disorder not otherwise specified (EDNOS) and officially recognized eating disorders: meta-analysis and implications for DSM. *Psychol Bull.* 2009;135(3):407–433
35. Ornstein RM, Rosen DS, Mammel KA, et al. Distribution of eating disorders in children and adolescents using the proposed DSM-5 criteria for feeding and eating disorders. *J Adolesc Health.* 2013;53(2):303–305
36. Bravender T, Bryant-Waugh R, Herzog D, et al. Classification of eating disturbance in children and adolescents: proposed changes for the DSM-V. *Eur Eat Disord. Rev Mar.* 2010;18(2):79–89
37. Miller KK, Grinspoon S, Gleysteen S, et al. Preservation of neuroendocrine control of reproductive function despite severe undernutrition. *J Clin Endocrinol Metab.* 2004;89(9):4434–4438
38. Bulik CM, Marcus MD, Zerwas S, Levine MD, La Via M. The changing "weightscape" of bulimia nervosa. *Am J Psychiatry.* 2012;169(10):1031–1036
39. Norris ML, Robinson A, Obeid N, Harrison M, Spettigue W, Henderson K. Exploring avoidant/restrictive food intake disorder in eating disordered patients: A descriptive study. *Int J Eat Disord.* 2014;47(5):495–499
40. Palla B, Litt IF. Medical complications of eating disorders in adolescents. *Pediatrics.* 1988;81(5):613–623
41. Katzman DK. Medical complications in adolescents with anorexia nervosa: a review of the literature. *Int J Eat Disord.* 2005;37(suppl):S52–59; discussion S87–59
42. Olivares JL, Vázquez M, Fleta J, Moreno LA, Pérez-González JM, Bueno M. Cardiac findings in adolescents with anorexia nervosa at diagnosis and after weight restoration. *Eur J Pediatr.* 2005;164(6):383–386
43. Mont L, Castro J, Herreros B, et al. Reversibility of cardiac abnormalities in adolescents with anorexia nervosa after weight recovery. *J Am Acad Child Adolesc Psychiatry.* 2003;42(7):808–813
44. Galetta F, Franzoni F, Prattichizzo F, Rolla M, Santoro G, Pentimone F. Heart rate variability and left ventricular diastolic function in anorexia nervosa. *J Adolesc Health.* 2003;32(6):416–421
45. Misra M, Aggarwal A, Miller KK, et al. Effects of anorexia nervosa on clinical, hematologic, biochemical, and bone density parameters in community-dwelling adolescent girls. *Pediatrics.* 2004;114(6):1574–1583
46. Kastner S, Salbach-Andrae H, Renneberg B, Pfeiffer E, Lehmkuhl U, Schmitz L. Echocardiographic findings in adolescents with anorexia nervosa at beginning of treatment and after weight recovery. *Eur Child Adolesc Psychiatry.* 2012;21(1):15–21
47. Hudson LD, Court AJ. What paediatricians should know about eating disorders in children and young people. *J Paediatr Child Health.* 2012;48(10):869–875
48. Meczekalski B, Podfigurna-Stopa A, Katulski K. Long-term consequences of anorexia nervosa. *Maturitas.* 2013;75(3):215–220
49. Docx MK, Gewillig M, Simons A, et al. Pericardial effusions in adolescent girls with anorexia nervosa: clinical course and risk factors. *Eat Disord.* 2010;18(3):218–225
50. Iraghi G, Perucca A, Parravicini U, et al. Severe bradycardia in an asymptomatic young subject: is there an indication to permanent cardiac pacing [in Italian]?. *G Ital Cardiol (Rome).* 2006;7(4):299–302
51. Oflaz S, Yucel B, Oz F, et al. Assessment of myocardial damage by cardiac MRI in patients with anorexia nervosa. *Int J Eat Disord.* 2013;46(8):862–866
52. Effects of ipecac on the heart. *N Engl J Med.* 1986;314(19):1253–1255
53. Brotman MC, Forbath N, Garfinkel PE, Humphrey JG. Myopathy due to ipecac syrup poisoning in a patient with anorexia nervosa. *Can Med Assoc J.* 1981;125(5):453–454
54. Friedman EJ. Death from ipecac intoxication in a patient with anorexia nervosa. *Am J Psychiatry.* 1984;141(5):702–703
55. Ho PC, Dweik R, Cohen MC. Rapidly reversible cardiomyopathy associated with chronic ipecac ingestion. *Clin Cardiol.* 1998;21(10):780–783
56. Brown CA, Mehler PS. Medical complications of self-induced vomiting. *Eat Disord.* 2013;21(4):287–294
57. Buchanan R, Ngwira J, Amsha K. *Prolonged QT interval in bulimia nervosa. BMJ Case Rep.* 2011;March 25
58. Kennedy SH, Heslegrave RJ. Cardiac regulation in bulimia nervosa. *J Psychiatr Res.* 1989;23(3-4):267–273
59. Messerli-Burgy N, Engesser C, Lemmenmeier E, Steptoe A, Laederach-Hofmann K. Cardiovascular stress reactivity and recovery in bulimia nervosa and binge eating disorder. *Int J Psychophysiol.* 2010;78(2):163–168
60. Nahshoni E, Yaroslavsky A, Varticovschi P, Weizman A, Stein D. Alterations in QT dispersion in the surface electrocardiogram of female adolescent inpatients diagnosed with bulimia nervosa. *Compr Psychiatry.* 2010;51(4):406–411
61. Takimoto Y, Yoshiuchi K, Kumano H, Kuboki T. Bulimia nervosa and abnormal cardiac repolarization. *J Psychosom Res.* 2006;60(1):105–107
62. Vögele C, Hilbert A, Tuschen-Caffier B. Dietary restriction, cardiac autonomic regulation and stress reactivity in bulimic women. *Physiol Behav.* 2009;98(1–2):229–234
63. Hadley SJ, Walsh BT. Gastrointestinal disturbances in anorexia nervosa and bulimia nervosa. *Curr Drug Targets CNS Neurol Disord.* 2003;2(1):1–9
64. Adson DE, Mitchell JE, Trenkner SW. The superior mesenteric artery syndrome and acute gastric dilatation in eating disorders: a report of two cases and a review of the literature. *Int J Eat Disord.* 1997;21(2):103–114
65. Fong HF, Divasta AD, Difabio D, Ringelheim J, Jonas MM, Gordon CM. Prevalence and predictors of abnormal liver enzymes in young women with anorexia nervosa. *J Pediatr.* 2008;153(2):247–253
66. Zipfel S, Sammet I, Rapps N, Herzog W, Herpertz S, Martens U. Gastrointestinal disturbances in eating disorders: clinical and neurobiological aspects. *Auton Neurosci.* 2006;129(1–2):99–106
67. Bozzato A, Burger P, Zenk J, Uter W, Iro H. Salivary gland biometry in female patients with eating disorders. *Eur Arch Otorhinolaryngol.* 2008;265(9):1095–1102
68. Price C, Schmidt MA, Adam EJ, Lacey H. Parotid gland enlargement in eating disorders: an insensitive sign? *Eat Weight Disord.* 2008;13(4):e79–e83
69. Kim HH, Park SJ, Park MI, Moon W. Acute gastric dilatation and acute pancreatitis in a patient with an eating disorder: solving a chicken and egg situation. *Intern Med.* 2011;50(6):571–575
70. Repesse X, Bodson L, Au SM, Charron C, Vieillard-Baron A. Gastric dilatation and circulatory collapse due to eating disorder. *Am J Emerg Med.* 2013;31(3):633.e633–634

Downloaded from pediatrics.aappublications.org at McMaster University on September 2, 2014

71. Mehanna HM, Moledina J, Travis J. Refeeding syndrome: what it is, and how to prevent and treat it. *BMJ*. 2008;336(7659):1495–1498
72. Setnick J. Micronutrient deficiencies and supplementation in anorexia and bulimia nervosa: a review of literature. *Nutr Clin Pract*. 2010;25(2):137–142
73. Mehler PS. Clinical practice. Bulimia nervosa. *N Engl J Med*. 2003;349(9):875–881
74. Fisher M, Simpser E, Schneider M. Hypophosphatemia secondary to oral refeeding in anorexia nervosa. *Int J Eat Disord*. 2000;28(2):181–187
75. Greenfeld D, Mickley D, Quinlan DM, Roloff P. Hypokalemia in outpatients with eating disorders. *Am J Psychiatry*. 1995;152(1):60–63
76. Wolfe BE, Metzger ED, Levine JM, Jimerson DC. Laboratory screening for electrolyte abnormalities and anemia in bulimia nervosa: a controlled study. *Int J Eat Disord*. 2001;30(3):288–293
77. Hall RC, Hoffman RS, Beresford TP, Wooley B, Tice L, Hall AK. Hypomagnesemia in patients with eating disorders. *Psychosomatics*. 1988;29(3):264–272
78. Lantzouni E, Frank GR, Golden NH, Shenker RI. Reversibility of growth stunting in early onset anorexia nervosa: a prospective study. *J Adolesc Health*. 2002;31(2):162–165
79. Misra M, Klibanski A. Neuroendocrine consequences of anorexia nervosa in adolescents. *Endocr Dev*. 2010;17:197–214
80. Misra M, Klibanski A. The neuroendocrine basis of anorexia nervosa and its impact on bone metabolism. *Neuroendocrinology*. 2011;93(2):65–73
81. Misra M, Klibanski A. Bone metabolism in adolescents with anorexia nervosa. *J Endocrinol Invest*. 2011;34(4):324–332
82. Chan JL, Mantzoros CS. Role of leptin in energy-deprivation states: normal human physiology and clinical implications for hypothalamic amenorrhoea and anorexia nervosa. *Lancet*. 2005;366(9479):74–85
83. Bouquegneau A, Dubois BE, Krzesinski JM, Delanaye P. Anorexia nervosa and the kidney. *Am J Kidney Dis*. 2012;60(2):299–307
84. Kanbur N, Katzman DK. Impaired osmoregulation in anorexia nervosa: review of the literature. *Pediatr Endocrinol Rev* . 2011;8(3):218–221
85. Li Cavoli G, Mule G, Rotolo U. Renal involvement in psychological eating disorders. *Nephron Clin Pract*. 2011;119(4):c338–341; discussion c341
86. Winston AP. The clinical biochemistry of anorexia nervosa. *Ann Clin Biochem*. 2012;49(pt 2):132–143
87. Chui HT, Christensen BK, Zipursky RB, et al. Cognitive function and brain structure in females with a history of adolescent-onset anorexia nervosa. *Pediatrics*. 2008;122(2):e426–e437
88. Chowdhury U, Gordon I, Lask B, Watkins B, Watt H, Christie D. Early-onset anorexia nervosa: is there evidence of limbic system imbalance? *Int J Eat Disord*. 2003;33(4):388–396
89. Fitzpatrick KK, Darcy A, Colborn D, Gudorf C, Lock J. Set-shifting among adolescents with anorexia nervosa. *Int J Eat Disord*. 2012;45(7):909–912
90. Frank GK, Kaye WH. Current status of functional imaging in eating disorders. *Int J Eat Disord*. 2012;45(6):723–736
91. Kaye WH, Bailer UF. Understanding the neural circuitry of appetitive regulation in eating disorders. *Biol Psychiatry*. 2011;70(8):704–705
92. Kaye WH, Wierenga CE, Bailer UF, Simmons AN, Wagner A, Bischoff-Grethe A. Does a shared neurobiology for foods and drugs of abuse contribute to extremes of food ingestion in anorexia and bulimia nervosa? *Biol Psychiatry*. 2013;73(9):836–842
93. Lawson EA, Holsen LM, Desanti R, et al. Increased hypothalamic-pituitary-adrenal drive is associated with decreased appetite and hypoactivation of food-motivation neurocircuitry in anorexia nervosa. *Eur J Endocrin*. 2013;169(5):639–647
94. Lock J, Garrett A, Beenhakker J, Reiss AL. Aberrant brain activation during a response inhibition task in adolescent eating disorder subtypes. *Am J Psychiatry*. 2011;168(1):55–64
95. Nunn K, Frampton I, Fuglset TS, Törzsök-Sonnevend M, Lask B. Anorexia nervosa and the insula. *Med Hypotheses*. 2011;76(3):353–357
96. Nunn K, Frampton I, Gordon I, Lask B. The fault is not in her parents but in her insula—a neurobiological hypothesis of anorexia nervosa. *Eur Eat Disord Rev*. 2008;16(5):355–360
97. Nunn K, Frampton I, Lask B. Anorexia nervosa—a noradrenergic dysregulation hypothesis. *Med Hypotheses*. 2012;78(5):580–584
98. Oberndorfer TA, Frank GK, Simmons AN, et al. Altered insula response to sweet taste processing after recovery from anorexia and bulimia nervosa. *Am J Psychiatry*. 2013;170(10):1143–1151
99. Sato Y, Saito N, Utsumi A, et al. Neural basis of impaired cognitive flexibility in patients with anorexia nervosa. *PLoS ONE*. 2013;8(5):e61108
100. Shott ME, Filoteo JV, Bhatnagar KA, et al. Cognitive set-shifting in anorexia nervosa. *Eur Eat Disord Rev*. 2012;20(5):343–349
101. Van Autreve S, De Baene W, Baeken C, van Heeringen C, Vervaet M. Do restrictive and bingeing/purging subtypes of anorexia nervosa differ on central coherence and set shifting? *Eur Eat Disord Rev*. 2013;21(4):308–314
102. Lambe EK, Katzman DK, Mikulis DJ, Kennedy SH, Zipursky RB. Cerebral gray matter volume deficits after weight recovery from anorexia nervosa. *Arch Gen Psychiatry*. 1997;54(6):537–542
103. Wagner A, Greer P, Bailer UF, et al. Normal brain tissue volumes after long-term recovery in anorexia and bulimia nervosa. *Biol Psychiatry*. 2006;59(3):291–293
104. Casper RC. Depression and eating disorders. *Depress Anxiety*. 1998;8(suppl 1):96–104
105. Franko DL, Keel PK. Suicidality in eating disorders: occurrence, correlates, and clinical implications. *Clin Psychol Rev*. 2006;26(6):769–782
106. Godart NT, Flament MF, Perdereau F, Jeammet P. Comorbidity between eating disorders and anxiety disorders: a review. *Int J Eat Disord*. 2002;32(3):253–270
107. Golden NH, Attia E. Psychopharmacology of eating disorders in children and adolescents. *Pediatr Clin North Am*. 2011;58(1):121–138, xi
108. Halmi KA. Anorexia nervosa: an increasing problem in children and adolescents. *Dialogues Clin Neurosci*. 2009;11(1):100–103
109. Nicholls D, Hudson L, Mahomed F. Managing anorexia nervosa. *Arch Dis Child*. 2011;96(10):977–982
110. Peebles R, Wilson JL, Lock JD. Self-injury in adolescents with eating disorders: correlates and provider bias. *J Adolesc Health*. 2011;48(3):310–313
111. Kreipe RE. Eating disorders among children and adolescents. *Pediatr Rev*. 1995;16(10):370–379
112. le Grange D, Lock J, Loeb K, Nicholls D. Academy for Eating Disorders position paper: the role of the family in eating disorders. *Int J Eat Disord*. 2010;43(1):1–5
113. Katzman DK, Peebles R, Sawyer SM, Lock J, Le Grange D. The role of the pediatrician in family-based treatment for adolescent eating disorders: opportunities and challenges. *J Adolesc Health*. 2013;53(4):433–440
114. Stiles-Shields C, Hoste RR, Doyle PM, Le Grange D. A review of family-based treatment for adolescents with eating disorders. *Rev Recent Clin Trials*. 2012;7(2):133–140

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115. Lock J. Treatment of adolescent eating disorders: progress and challenges. *Minerva Psichiatr.* 2010;51(3):207–216
116. Lock J, le Grange D, Forsberg S, Hewell K. Is family therapy useful for treating children with anorexia nervosa? Results of a case series. *J Am Acad Child Adolesc Psychiatry.* 2006;45(11):1323–1328
117. le Grange D, Crosby RD, Rathouz PJ, Leventhal BL. A randomized controlled comparison of family-based treatment and supportive psychotherapy for adolescent bulimia nervosa. *Arch Gen Psychiatry.* 2007;64(9):1049–1056
118. le Grange D, Doyle P, Crosby RD, Chen E. Early response to treatment in adolescent bulimia nervosa. *Int J Eat Disord.* 2008;41(8):755–757
119. Le Grange D, Lock J, Dymek M. Family-based therapy for adolescents with bulimia nervosa. *Am J Psychother.* 2003;57 (2):237–251
120. Loeb KL, le Grange D. Family-based treatment for adolescent eating disorders: current status, new applications and future directions. *Intl J Child Adolesc Health.* 2009;2(2):243–254
121. Berkman ND, Bulik CM, Brownley KA, et al. Management of eating disorders. *Evid Rep Technol Assess (Full Rep).* 2006; (135):1–166
122. Loeb KL, Lock J, Grange DL, Greif R. Transdiagnostic theory and application of family-based treatment for youth with eating disorders. *Cognit Behav Pract.* 2012;19(1):17–30
123. Manikam R, Perman JA. Pediatric feeding disorders. *J Clin Gastroenterol.* 2000;30 (1):34–46
124. Tang B, Piazza CC, Dolezal D, Stein MT. Severe feeding disorder and malnutrition in 2 children with autism. *J Dev Behav Pediatr.* 2011;32(3):264–267
125. Dimitropoulos G, Freeman VE, Bellai K, Olmsted M. Inpatients with severe anorexia nervosa and their siblings: non-shared experiences and family functioning. *Eur Eat Disord Rev.* 2013;21(4):284–293
126. Arcelus J, Mitchell AJ, Wales J, Nielsen S. Mortality rates in patients with anorexia nervosa and other eating disorders. A meta-analysis of 36 studies. *Arch Gen Psychiatry.* 2011;68(7):724–731
127. Silber TJ. Anorexia nervosa in children and adolescents: diagnosis, treatment and the role of the pediatrician. *Minerva Pediatr.* 2013;65(1):1–17
128. Attia E, Kaplan AS, Walsh BT, et al. Olanzapine versus placebo for out-patients with anorexia nervosa. *Psychol Med.* 2011; 41(10):2177–2182
129. Kafantaris V, Leigh E, Hertz S, et al. A placebo-controlled pilot study of adjunctive olanzapine for adolescents with anorexia nervosa. *J Child Adolesc Psychopharmacol.* 2011;21(3):207–212
130. Norris ML, Spettigue W, Buchholz A, et al. Olanzapine use for the adjunctive treatment of adolescents with anorexia nervosa. *J Child Adolesc Psychopharmacol.* 2011;21(3):213–220
131. Couturier J, Lock J. What is recovery in adolescent anorexia nervosa? *Int J Eat Disord.* 2006;39(7):550–555
132. Jarman M, Walsh S. Evaluating recovery from anorexia nervosa and bulimia nervosa: integrating lessons learned from research and clinical practice. *Clin Psychol Rev.* 1999; 19(7):773–788
133. Morgan HG, Hayward AE. Clinical assessment of anorexia nervosa. The Morgan-Russell outcome assessment schedule. *Br J Psychiatry.* 1988;152:367–371
134. Pike KM. Long-term course of anorexia nervosa: response, relapse, remission, and recovery. *Clin Psychol Rev.* 1998;18(4):447–475
135. Couturier J, Lock J. What is remission in adolescent anorexia nervosa? A review of various conceptualizations and quantitative analysis. *Int J Eat Disord.* 2006;39(3):175–183
136. Meguerditchian C, Samuelian-Massat C, Valéro R, et al. The impact of weight normalization on quality of recovery in anorexia nervosa. *J Am Coll Nutr.* 2009;28(4):397–404
137. Lo Sauro C, Castellini G, Lelli L, Faravelli C, Ricca V. Psychopathological and clinical features of remitted anorexia nervosa patients: a six-year follow-up study. *Eur Eat Disord Rev.* 2013;21(1):78–83
138. Le Grange D, Doyle PM, Swanson SA, Ludwig K, Glunz C, Kreipe RE. Calculation of expected body weight in adolescents with eating disorders. *Pediatrics.* 2012; 129(2):e438–e446
139. O'Toole J. Determining ideal body weight. Kartini Eating Disorder Blog 2013. Available at: www.kartiniclinic.com/blog/post/determining-ideal-body-weight/. Accessed August 8, 2014
140. Weltzin TE, Fernstrom MH, Hansen D, McConaha C, Kaye WH. Abnormal caloric requirements for weight maintenance in patients with anorexia and bulimia nervosa. *Am J Psychiatry.* 1991;148(12):1675–1682
141. Agostino H, Erdstein J, Di Meglio G. Shifting paradigms: continuous nasogastric feeding with high caloric intakes in anorexia nervosa. *J Adolesc Health.* 2013;53(5):590–594
142. Garber AK, Mauldin K, Michihata N, Buckelew SM, Shafer MA, Moscicki AB. Higher calorie diets increase rate of weight gain and shorten hospital stay in hospitalized adolescents with anorexia nervosa. *J Adolesc Health.* 2013;53(5):579–584
143. Garber AK, Michihata N, Hetnal K, Shafer MA, Moscicki AB. A prospective examination of weight gain in hospitalized adolescents with anorexia nervosa on a recommended refeeding protocol. *J Adolesc Health.* 2012;50(1):24–29
144. Golden NH, Keane-Miller C, Sainani KL, Kapphahn CJ. Higher caloric intake in hospitalized adolescents with anorexia nervosa is associated with reduced length of stay and no increased rate of refeeding syndrome. *J Adolesc Health.* 2013;53(5):573–578
145. Kohn MR, Madden S, Clarke SD. Refeeding in anorexia nervosa: increased safety and efficiency through understanding the pathophysiology of protein calorie malnutrition. *Curr Opin Pediatr.* 2011;23(4):390–394
146. Whitelaw M, Gilbertson H, Lam PY, Sawyer SM. Does aggressive refeeding in hospitalized adolescents with anorexia nervosa result in increased hypophosphatemia? *J Adolesc Health.* 2010;46(6):577–582
147. Shaw H, Stice E, Becker CB. Preventing eating disorders. *Child Adolesc Psychiatr Clin N Am.* 2009;18(1):199–207
148. Jones M, Völker U, Lock J, Taylor CB, Jacobi C. Family-based early intervention for anorexia nervosa. *Eur Eat Disord Rev.* 2012;20(3):e137–e143
149. Neumark-Sztainer D. Integrating messages from the eating disorders field into obesity prevention. *Adolesc Med State Art Rev.* 2012;23(3):529–543

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Pediatrics 2014;134;582; originally published online August 25, 2014;
DOI: 10.1542/peds.2014-0194

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