Practice Parameter for the Assessment and Treatment of Children and Adolescents With Eating Disorders

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Abstract: This Practice Parameter reviews evidence-based practices for the evaluation and treatment of eating disorders in children and adolescents. Where empirical support is limited, clinical consensus opinion is used to supplement systematic data review. The Parameter focuses on the phenomenology of eating disorders, comorbidity of eating disorders with other psychiatric and medical disorders, and treatment in children and adolescents. Because the database related to eating disorders in younger patients is limited, relevant literature drawn from adult studies is included in the discussion.

Key Words: eating disorders, anorexia nervosa, bulimia nervosa, food avoidance, binge eating

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This Practice Parameter provides an evidence-based approach to the evaluation and treatment of eating disorders in children and adolescents, including specifically anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), and avoidant restrictive food intake disorder (ARFID). The Parameter will not address feeding problems in infancy (e.g., failure to thrive), pica, rumination disorder, purging disorder, or the evaluation and treatment of obesity. Evaluation and treatment of eating disorders in children and adolescents is complex and often requires specific expertise and relevant clinical experience. This Practice Parameter is designed to help child psychiatrists accurately assess and effectively treat children and adolescents with eating disorders. This Parameter may also provide useful information for other medical and mental health professionals because the treatment of eating disorders commonly requires consultation and involvement with other experts in addition to child psychiatrists.

METHODOLOGY

The recommendations in this Practice Parameter were developed after searching literature including PubMed/Medline and using the relevant medical subject headings (MeSH terms) "eating disorders," adding limits "child: 6-12 years" and "adolescent: 13-18 years," "clinical trial," and a time period from 1985 to 2011 inclusive (yields 497 citations); Cochrane, using the relevant medical subject headings (MeSH terms) "eating disorders," adding limits "clinical trials" (yields 646 citations); and PsycINFO, using the term "eating disorders," adding limits "child: 6-12 years" and "adolescent: 13-17 years," "clinical trial," and a time period from 1985 to 2011 inclusive (yields 112 citations). In addition, the bibliographies of book chapters and treatment guideline articles were reviewed; and finally, colleagues were asked for suggested source materials.

The online search was narrowed on PubMed/Medline using delimiters and filters such as English language only, human subjects, and using the Boolean operator "AND," "OR," and "NOT" to include the following search terms: family therapy, comorbid, treatment outcome, psychopharmacology, and eating disorder not otherwise specified to reduce citations to 141. Similarly, the online search was narrowed on Cochrane by searching clinical trials, and using the Boolean operator "AND" and "OR" to include the following search terms: anorexia, bulimia, child, adolescent, and family based therapy, to reduce citations to 17. Finally, the online search was narrowed on PsycINFO by using the Boolean operator "AND" and "OR" to include the following search terms: anorexia and bulimia. The subject of this search was further specified by including treatment outcomes, family therapy, and clinical trials to reduce citations to 69 results.

For this Practice Parameter, we hand culled 91 publications for examination based on their relevance to clinical practice. In addition, 19 more recent references for 2012 to 2013 were identified by expert and member reviews.

HISTORICAL REVIEW

The first comprehensive description of a condition resembling AN was provided by Richard Morton in 1689, which he called *nervous consumption*.[1] Although there is evidence in ancient history, usually in the context of religious beliefs, of clinical problems similar to AN,[2] it was not until 1874 when Sir William Gull in England and Charles Lasegue in France coined the terms *anorexia nervosa* and *anorexia hysterique*, respectively, to describe the symptoms of self-starvation and weight preoccupations associated with AN.[3] It was not until this time that theories related to etiology and treatment began to evolve. Both Gull and Lasegue

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suggested that families likely contributed to the disorder, but Jean-Martin Charcot directly blamed families and advocated complete separation of affected individuals from their families whose influence he viewed as "pernicious."[4] Thus, treatment for AN from the late 19th century to the present has often included hospitalization and long separations from family members, where patients are treated exclusively by professionals.[5,6] Psychoanalytic approaches suggested that affected individuals suffered from a range of unconscious problems including pregnancy fears, primary narcissism, and confusion between body and emotion.[7] Hilde Bruch conceptualized AN as a disorder of suppression and neglect in childhood, leading to food refusal and the formation of AN symptoms for self-assertion. She advocated for individually oriented psychodynamic therapy for the patient to promote autonomy and independence from parents and families.[8,9] Patients were often treated in psychiatric hospitals residential treatment programs.[10] Salvador Minuchin's pioneering work in structural family therapy with psychosomatic disorders suggested that families could be important in treatment, despite the prior practices.[11] Subsequently, researchers at the Institute of Psychiatry and Maudsley Hospital in London developed a form of family therapy that was specifically designed to use parental skills to disrupt the maintaining behaviors of AN.[12] Over the past 10 years, a substantial database supports including families in the treatment of adolescents with AN.[13]

BN was first included in the *DSM-III* in 1980 following clinical descriptions of patients with binge eating and purging by Boskand-Lodahl (bulimarexia)[14,15] and Russell, who called it "an ominous variant" of AN.[16] Although few studies have investigated treatment for BN in adolescents, many intervention studies of adults with BN have demonstrated the effectiveness of cognitive-behavioral therapy (CBT) for this disorder.[17] In addition, antidepressant medications and interpersonal psychotherapy (IPT) are effective in adults with BN.[18] The diagnosis of binge eating disorder (BED) is now included in the *DSM-5*.[19] BED is understudied in children and adolescents but appears to be rarer in younger patients than in adults.[20] Effective treatments for adults with BED include CBT, IPT, and medications.[21,22] Pilot studies support the use of IPT for adolescents with BED.[23] Another new diagnosis in the *DSM-5* is ARFID.[19] This is a disorder found principally in children. In ARFID, food or eating is avoided, usually leading to low weight, but is not associated with shape or weight concerns, or intentional efforts to reduce weight.[24]

ANOREXIA NERVOSA (AN)

Clinical Presentation and Course

DSM-5 criteria for AN include the following: restriction of energy intake leading to low body weight; fear of gaining weight or behavior that interferes with weight gain; and self-evaluation unduly influenced by weight and body shape.[25] Denial of the seriousness of malnutrition is also a common symptom, especially in younger patients.[26] There are 2 subtypes of AN: a restricting type and a binge-eating/purging type. In the *DSM-5*, amenorrhea is no longer required to meet diagnostic criteria.[27] The *DSM-5* suggests that clinicians rate the level of severity of AN (mild to extreme) in adults based on current body mass index (BMI); in children and adolescents, severity is based on age and gender norms according to BMI percentiles.[26,28,29] A BMI below the 10th percentile is considered to be consistent with the degree of malnutrition associated with AN.[30,31] Alternatively, if longitudinal growth charts are available, deviations from individual growth trajectories can be observed.[32] At present, there is little scientific basis for using weight as a marker of severity in children and adolescents, so it remains to be seen how useful this approach will be in defining severity.

There is evidence that AN symptoms may be expressed differently in childhood and adolescence as compared to adulthood.[33] Children and adolescents are often incapable of verbalizing abstract thoughts; therefore, behaviors such as food refusal that lead to malnutrition may manifest as nonverbal representations of emotional experiences. As a result, parental reports about the child's behavior are critical, as self-report is often unreliable because of a lack of insight, minimization, and denial by the child or adolescent.[34] Children and adolescents with AN are less likely than adults to engage in binge eating and purging behaviors.[35]

Children and adolescents with AN most often present for psychiatric evaluation after a pediatrician or other medical provider suspects an eating disorder based on the patient's unexpected weight loss or failure to gain expected weight. Patients with AN often develop weight concerns and subsequent behavioral change directed toward weight loss 6 to 12 months before the full clinical diagnosis. The rate of weight loss typically escalates in the last few weeks before referral, prompting parents to seek a medical evaluation.[36] Patients sometimes report initial drive for thinness, but often claim that they are trying to eat less, avoid fattening foods, and exercise more for health reasons. Other young patients deny body image or weight concerns at assessment and insist they just "aren't hungry" or complain of abdominal discomfort. It is important, however, not to infer fear of weight gain or weight and shape concerns on the basis of developmental immaturity alone; instead, the presence of behaviors indicating avoidance of "fattening" foods or indicating fear of weight gain (such as repeated weighing, pinching skin) may confirm the diagnosis. Typically, caloric reduction increases over time as food choices become more limited (usually elimination of protein, fats, and sweets). As these dieting behaviors persist, the patient focuses more exclusively on weight and dieting to the exclusion of friends and family. Academic and athletic pursuits usually continue and sometimes become more compulsive and driven. Patients may have a compulsion to stand or move, may be exercising secretly, or may no longer have the energy to overexercise. They may dress in baggy clothing or layers and complain of feeling cold. Some patients may drink water excessively, whereas others may restrict fluid intake. Patients often appear withdrawn, depressed, and anxious. Usually they remain cognitively intact until more severe malnutrition develops. In some instances, compensatory behavior such as purging develops, but this is usually later in the course of the disorder in younger patients.[35]

Long-term adult follow-up studies of AN suggest rates of chronicity (defined as having AN for more than 5 years) of 7% to 15% and mortality of 5% to 7%, although mortality as high as 18% has been reported in some samples.[37] Death is most often secondary to medical complications of starvation (50%) or suicide (50%).[38] The prognosis for adolescents with AN is better than in adult populations.[39,40]

Epidemiology

The prevalence of AN in the United States is reported to be 1% to 2% among females, whereas the prevalence in teenage girls has been reported to be 0.3% to 0.7%.[41,42] One study examining the rates of AN between 1939 and 1984 found evidence that the incidence of AN increased in females aged 10 to 24 years.[43,44] Little is known about the prevalence of AN in males; a ratio of 1 male case to every 10 females is commonly cited,[45] although some estimates are as high as 1:1.[46,47] Studies of males with eating disorders are limited, although some studies suggest that incidence or detection rates of AN in males are increasing.[46,48,49] The prevalence of subthreshold AN is estimated to be 1.5% in adolescent females and 0.1% in adolescent males.[42,50] There are also only sparse data related to the racial and ethnic distribution of AN, but recent studies suggest that the disorder may be less common in persons of African origin.[51] Furthermore, only 3 studies have assessed the incidence rates of AN in children 14 years or younger.[43,52] The first study reported incidence rates of 9.2 to 25.7 females per 100,000 per year in 10- to 14-year-olds and 11.9 to 69.4 females per 100,000 per year in 15- to 19-year-olds. The second study reported incidence rates of 25.7 females and 3.7 males per 100,000 of the population per year in 10- to 14-year olds and 69.4 females and 7.3 males per 100,000 of the population per year in 15- to 19-year-olds.[43] Peak incidence occurs at 14 to 18 years of age,[53] with rare cases presenting after 25 years of age.[43] The third study found an overall incidence of eating disorders of 3:100,000 between 5 and 13 years of age. Incidence rates were found to rise with age to 4.5:100,000 by age 11 years and to 9.5:100,000 by age 12. Of these, 80% had AN or a restrictive eating problem similar to AN but not meeting full diagnostic criteria.[54]

Etiology and Risk Factors

The etiology of AN is likely multifactorial and precipitated by the interaction of several risk factors, including biological, psychological, environmental/cultural, and sociological. Family aggregation studies demonstrate that AN occurs at about 5 times the expected rates in affected families. Twin studies find heritability estimates ranging from approximately 30% to 75% in AN.[55] Recent twin studies show an interaction between genes and developmental processes in children and adolescents who develop AN. One study[56] found that for 11-year-old twins, genetic influences were marginal, but in 17-year-old twins, heritability was high.[57] One possible explanation for this finding is that hormonal changes during adolescence could mediate gene expression during puberty.[58,59]

Temperament and personality type are also risk factors associated with AN,[55,60] and perfectionistic, obsessive, and avoidant personality features are likely heritable.[61,62] Studies also suggest that specific cognitive features are associated with AN, including cognitive rigidity and a bias toward detail information processing.[63,64] Some have characterized these cognitive features as an endophenotype because they are present in patients after recovery and in unaffected siblings.[65-67] Picky eating early in life has also been associated with later development of AN.[68] Developmental challenges associated with adolescence such as autonomy, self-efficacy, and intimacy are found in patients with AN, although it is uncertain whether this is a cause or a result of the disorder.[8,69] Psychosocial factors associated with the development of AN include societal pressures related to thinness and appearance that can trigger extreme dieting in vulnerable individuals.[70-72] In addition, some studies suggest that certain activities such as ballet, gymnastics, wrestling, and modeling may increase risk for eating disorders because of the role of appearance and/or weight in performance.[73,74] The westernization of culture, with an emphasis on the "thin ideal," is also associated with AN in some studies, particularly in Asian culture and immigrants to Europe and the Americas.[75-77]

Differential Diagnosis and Comorbidity

There are many possible causes of weight loss, loss of appetite, and refusal to eat. Differential diagnosis for AN includes ARFID and rumination disorder when these disorders have resulted in low weight. Any medical or psychiatric illness that leads to changes in appetite, weight loss, or changes in food intake likely increases the risk for the development of an eating disorder. In addition, chronic infection, thyroid disease, Addison's disease, inflammatory bowel disease, connective tissue disorders, cystic fibrosis, peptic ulcer disease, disease of the esophagus, celiac disease, infectious diseases, disease of the small intestine, diarrhea, diabetes mellitus, and occult malignancies can lead to weight loss, appetite loss, and refusal to eat.[78] Many of these can be ruled out with a thorough history and physical examination along with laboratory studies.

Further complicating the diagnosis of AN is the potential presence of other significant psychiatric comorbid conditions. In adolescents with AN, results from the National Comorbidity Survey Replication Adolescent Supplement (NCS-A) found that the lifetime rate of comorbidity with at least 1 other psychiatric disorder is 55.2%.[79] Psychiatric comorbidity in adults with AN includes depression, social anxiety, separation anxiety, obsessive-compulsive disorder (OCD), generalized anxiety, and substance abuse, and avoidant, dependent, obsessive-compulsive, or passive-aggressive personality disorders are also often diagnosed.[80-82] AN and OCD share obsessional preoccupations, such as over eating, food, weight, and shape obsessions, and compulsive behaviors such as restricting and counting calories, overexercising, checking behaviors, and meal-time rituals, making it difficult to differentiate these 2 disorders. When the preoccupations and compulsions emerge at the same time and are focused on food, eating, and weight, the diagnosis, however, is likely to be AN. In addition, comorbid conditions, particularly anxiety disorders, are often present before AN develops and persist after recovery.[83-85] Specific phobias, particularly those such as fear

of swallowing, can lead to weight loss and be confused with AN. Similarly, dysphoric thoughts, low self-esteem, and guilt are found in both depression and AN. With depression these features encompass common aspects of life, whereas in AN these thoughts are focused more specifically on eating and weight-related concerns.[86]

BULIMIA NERVOSA (BN)

Clinical Presentation and Course

DSM-5 criteria for BN include the following: recurrent binge-eating in which a binge is defined as consuming a very large amount of food in a discrete period of time, such as within 2 hours, and a sense of loss of control over eating during that episode; recurrent compensatory behavior, such as vomiting, fasting, exercise, laxative use, diuretic use, diet pill use; and self-evaluation unduly influenced by weight and body shape. The binge-eating and compensatory behaviors both occur, on average, at least once a week for 3 months, and do not occur exclusively during AN.[19,25] The severity of BN, mild to extreme, is based on the frequency of compensatory behaviors. Compensatory behaviors distinguish BN from BED. Patients with BN are often within a normal or high normal weight range for age, gender, and height. Secrecy, shame, and guilt tend to accompany BN in adolescents, and in turn these emotions may negatively affect the age-appropriate development of adolescents by interfering with social and interpersonal processes.[87,88] Males with BN appear to be more likely than females to present with overexercise and steroid use.[89] Risk for BN is increased in males who participate in sports such as wrestling, gymnastics, diving, and long-distance running, in which weight and appearance can affect performance.[49] There is also a limited database suggesting that homosexual males may be more at risk than their heterosexual peers for the development of BN.[90,91]

There are challenges in diagnosing BN in childhood and adolescence because of developmental differences between younger patients and adults.[33] Data suggest that loss of control may be a more valid marker than calories consumed in terms of determining whether an eating episode should be characterized as binge eating.[92] Binge eating may also occur less frequently in younger patients because children do not have access to, or control over, foods in the same way as adults with BN.[33,35] Similar limitations on abstract thinking, self-expression, and minimization as described for adolescent AN are relevant for adolescent BN as well; thus, parental interviews and other collateral reports may be essential to obtain an accurate history.[33]

The short-term course of BN is marked by fluctuating symptoms with varying cycles of remission and exacerbation. Typically individuals with BN have exhibited symptoms for nearly 5 years before seeking treatment.[93] Outcome in BN is highly variable. Of those with BN who receive treatment, 50% are symptom free 5 to 10 years later, whereas the other 50% continue to exhibit eating disorder symptoms. One long-term follow-up study suggests, however, that the number of women who continue to meet full criteria for BN declines as the duration of follow-up increases.[94] A history of low self-esteem, childhood obesity, and personality disorders predict a longer duration of illness.[95] Self-injury, substance abuse, and other impulsive and risk-taking behaviors are common in adults and older teens seeking treatment, but appear to be less common in younger adolescents with BN.[96]

Epidemiology

The incidence of BN is increasing in urbanized areas and countries undergoing rapid Westernization.[41,97] Between 1% and 2% of adolescent females and 0.5% of adolescent males are estimated to meet *DSM-IV* criteria for BN.[98-100] The estimated male-to-female ratio is 1:10, although some estimates are as high as 1:3.[47,90] BN typically begins in adolescence between 14 and 22 years of age[42,101] and, for some individuals, arises after an episode of AN. BN is rarely diagnosed in children and young adolescents, although older patients presenting for treatment often pinpoint the onset of their illness to early adolescence.[35]

Etiology and Risk Factors

The etiology of BN is multifactorial, including biological, psychological, and sociological factors. BN occurs more often in first-degree relatives of those with eating disorders than in the general population.[102] Twin studies show a higher concordance rate in monozygotic twins than in dizygotic twins, with heritability threshold estimates ranging from 60% to 83%.[103-105] Family factors, abuse, posttraumatic stress disorder (PTSD), impulsive personality traits, and perfectionistic temperament have also been suggested to be risk factors for BN.[95,102,106-111] In addition, social pressures to be thin, body dissatisfaction, dieting, and negative affect are associated with the development of BN.[73,74,77] Dieting to try to accomplish a thin ideal physique is thought to lead to physical and psychological starvation.[93] Physiological starvation then leads to hunger and promotes binge eating when food becomes available. Guilt associated with binge eating and fear of weight gain lead to purging behavior and increased dieting. Thus, a cycle of deprivation, binge eating, and purging is established.

Differential Diagnosis and Comorbidity

Differential diagnosis for BN includes AN, binge/purge type, subthreshold BN, purging disorder, BED, major depressive disorder, central nervous system (CNS) tumors, Kleine-Levin syndrome, Kluver-Bucy syndrome, and gastrointestinal pathology such as obstruction and gastroparesis. Several psychiatric disorders are highly associated with BN, including affective disorders, anxiety disorders, substance use disorders, and personality disorders.[84,93,112] In adolescent BN, a recent study found a lifetime psychiatric comorbidity rate of 88%, with a majority of adolescents having at least 1 comorbid psychiatric illness.[79] In addition, significant rates of suicidal ideation (53%), plans (26%), and attempts (35%) were found among adolescents with BN.

BINGE EATING DISORDER (BED)

Clinical Presentation and Course

BED is characterized, as is BN, by recurrent binge eating episodes accompanied by a sense of loss of control over eating during the episode. In BED, these episodes are associated with at least 3 of the following: eating more rapidly, eating until uncomfortably full, eating when not hungry, eating alone because of embarrassment about the amount of food consumed, and feelings of disgust, depression, or guilt. According to the *DSM-5*, binge eating episodes need to occur, on average, at least once a week for 3 months, and must be associated with marked distress, to meet diagnostic severity levels. BED is distinguished from BN in part because the binge eating episodes are not associated with inappropriate compensatory behaviors. In addition, to meet criteria for BED, binge eating episodes cannot occur exclusively during the course of AN or BN.

There are similar challenges in diagnosing BED and BN in childhood and adolescence because of developmental differences between younger patients and adults.[33] A sense of being out of control when eating is likely more important than eating an objectively large amount of food in younger patients,[92] because younger patients often cannot gain access to food as easily as adults.[35] For these reasons, a clinical concern about BED in children and adolescents should consider using a lower threshold for the rate and duration of binge eating episodes.[33] A suggested rate of once per month, over the previous 3-month period, was recommended by a consensus group of experts in child and adolescent eating disorders.[33] In addition, as with AN and BN, children and adolescents are limited in their abstract thinking ability and self-expression. They may also minimize any discomfort or shame that they experience when binge eating. Thus, parental interviews and other collateral reports are often necessary for making a definitive diagnosis.[33] BN usually occurs in patients of normal weight or slightly overweight; BED often occurs in overweight and obese individuals. In BN, binge eating is considered to be a response to restriction of food intake,[113] whereas in BED, binge eating occurs in the context of overall chaotic and unregulated eating patterns.[114]

Epidemiology

BED may be the most common eating disorder, affecting 3.5% of females and 2% of males among adults.[47] Rates of BED in children and adolescents are estimated to be 2.3% in adolescent females and 0.8% in adolescent males. Prevalence of subthreshold BED is estimated to be 2.3% in female adolescents and 2.6% in male adolescents.[42] Some studies suggest that among obese adolescents, about 1% meet criteria for BED, and 9% have objective binge episodes.[20] A high percentage of adults and adolescents who present with BED are moderately overweight or obese. Prevalence rates of BED are higher among women enrolled in weight-control programs compared to those who are not.[115]

Etiology and Risk Factors

Comparatively little is known about the specific risk factors for BED in children and adolescents, as it is a relatively rare disorder in younger populations. Onset of binge eating typically begins in late adolescence or early adulthood, often after a period of significant dieting or weight loss. Some studies suggest that restrictive dieting, pressure to be thin, body dissatisfaction, emotional eating, low self-esteem, and poor social support are risk factors for the onset of adolescent binge eating.[116] Other studies suggest increased eating related to specific psychopathology, depressed mood, and increased anxiety are associated with the initiation of binge eating in adolescents.[117]

Differential Diagnosis and Comorbidity

Differential diagnosis includes BN, AN, and subthreshold BED, night eating syndrome, and nocturnal sleep-related eating disorder. Medical reasons for binge eating should be considered including CNS tumors, Kleine-Levin syndrome, Kluver-Bucy syndrome, Prader-Willi syndrome, and gastrointestinal pathology. In adults, BED is associated with significant comorbid psychopathology, including depressive disorders, anxiety disorders, PTSD, impulse control disorders, substance use disorders, and personality disorders.[50,118]

AVOIDANT RESTRICTIVE FOOD INTAKE DISORDER (ARFID)

Clinical Presentation

Diagnostic criteria for ARFID[24,119,120] include food restriction or avoidance without shape or weight concerns or intentional efforts to lose weight that results in significant weight loss and nutritional deficiencies, and are associated with disturbances in psychological development and functioning. Some patients present with highly selective eating, neophobia (the fear of new things) related to food types, or hypersensitivity to food texture, appearance, and taste.[120-123] For some patients, fear of swallowing or choking contributes to food avoidance; a specific event can sometimes be identified as triggering that fear. ARFID also applies to individuals who have a lack of interest in eating or who have low appetite.

Epidemiology

There are no epidemiological studies available for this new diagnosis.

Etiology and Risk Factors

Specific etiologic risk factors for ARFID are unknown. Patients with autism spectrum disorder (ASD) frequently display selective eating patterns. Anxiety disorders and anxious traits, as well as depressive symptoms, often pre-date the development of ARFID. Neglect, abuse, and developmental delays may increase the risk for chewing and spitting associated with ARFID.

Differential Diagnosis

ARFID can be confused with AN, but distinguishing features include a lack of fear of weight gain in ARFID, no shape and weight concerns in ARFID, and no specific focus on weight loss in ARFID. It is essential to obtain collateral history from parents, who will usually indicate no avoidance of high-calorie food in

ARFID. The patients are aware that they are low weight and may express a wish to eat more and gain weight, but their anxiety and fear prevent them from actually consuming enough.[24] ARFID can sometimes be confused with ASD, other neurodevelopmental disorders, and anxiety disorders.

OTHER SPECIFIED FEEDING OR EATING DISORDERS

In previous iterations of the *DSM,* an eating disorder not otherwise specified (EDNOS) was the broad diagnostic category that encompassed atypical and subthreshold presentations of AN, BN, BED and other atypical eating problems. In the *DSM-5*, many of the patients who would formerly have been diagnosed with EDNOS will now meet the revised criteria for AN, BN, BED, and ARFID. The remaining patients will be diagnosed under the general grouping of "other specified feeding or eating disorders" and will include specified diagnostic groupings such as atypical AN, which meets all criteria for AN, except that despite signficant weight loss, the individual's weight remains within or above the normal range; BN, with symptoms of low frequency and/or limited duration; BED, with symptoms of low frequency and/or limited duration; purging disorder, recurrent purging to influence body shape or weight in the absence of binge eating; and night eating syndrome, recurrent episodes of eating at night that lead to impairment and/or distress. Finally, there is a diagnosis called "unspecifed feeding and eating disorder" for cases in which the clinician chooses not to specify the reason that criteria are not met for a specific feeding or eating disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis. The rationale for this reorganization in the *DSM-5* was based in part on studies that suggested that many younger patients with AN and BN would receive more appropriate clinical diagnoses based on *DSM-5* criteria.[122]

Epidemiology, Etiology, and Risk Factors

The epidemiology of this new grouping of specified eating disorders in *DSM-5* is unknown, particularly in children and adolescents. For subthreshold AN and BN cases, the same etiologic and risk factors for full syndrome disorders are likely pertinent.

Differential Diagnosis

The differential diagnosis of disorders likely to be found in this group includes AN and BN, as well as the range of medical and psychological problems that may cause eating and weight changes in children and adolescents. The most common psychological problems include changes in appetite related to depression, anxiety and phobia secondary to choking, difficulty swallowing, gastric upset, gastrointestinal motility problems, and fear of vomiting and food intake after viral gastroenteritis. Common medical problems include diabetes mellitus, celiac disease, irritable bowel syndrome, and allergies. In addition to these types of presentations, a clinical syndrome associated with women athletes has been described and is called the female triad syndrome. The female triad syndrome consists of 3 elements: low dietary energy availability from disordered eating, amenorrhea, and low bone density. Although not a specific eating disorder, presentations consistent with this triad should be considered at high risk for an eating disorder and monitored closely for worsening medical problems and eating disorder symptoms. Studies suggest that among female high school athletes, 18.2% met criteria for disordered eating, 12.5% met criteria for menstrual irregularity, and 21.8% met criteria for low bone mass.[124,125]

EVIDENCE BASE FOR PRACTICE PARAMETERS

In this Parameter, recommendations for best assessment and treatment practices are stated in accordance with the strength of the underlying empirical and/or clinical support, as follows:

Clinical Standard [CS] is applied to recommendations that are based on rigorous empirical evidence, such as meta-analyses, systematic reviews, individual randomized controlled trials, and/or overwhelming clinical consensus;

Clinical Guideline [CG] is applied to recommendations that are based on strong empirical evidence, such as nonrandomized controlled trials, cohort studies, case-control studies, and/or strong clinical consensus;

Clinical Option [OP] is applied to recommendations that are based on emerging empirical evidence, such as uncontrolled trials or case series/reports, or clinical opinion, but lack strong empirical evidence and/or strong clinical consensus;

Not Endorsed [NE] is applied to practices that are known to be ineffective or contraindicated.

The strength of the empirical evidence is rated in descending order as follows:

Randomized, Controlled Trial [rct] is applied to studies in which patients are randomly assigned to 2 or more treatment conditions;

Controlled Trial [ct] is applied to studies in which patients are nonrandomly assigned to 2 or more treatment conditions;

Uncontrolled Trial [ut] is applied to studies in which patients are assigned to 1 treatment condition;

Case Series/Report [cs] is applied to a case series or a case report.

RECOMMENDATIONS

Recommendation 1. Mental health clinicians should screen all child and adolescent patients for eating disorders [CS]

All preteen and adolescent patients should be asked about eating patterns and body satisfaction. In addition, height and weight should be obtained and plotted on growth

curves. If there is a concern about the ratio of height to weight or evidence of growth failure, referral for further evaluation is warranted. The Eating Disorder Examination-Questionnaire (EDE-Q), Eating Disorder Inventory (EDI), and Eating Attitudes Test (EAT) are validated, short, self-report measures that can be useful screening instruments for eating disorders.[126-128] The Kids' Eating Disorder Survey (KEDS), ChEDE-Q, the EDI-C, and the Child-Eating Attitudes Test (CHEAT) are all validated measures for use in younger children.[129]

Recommendation 2. A positive screening should be followed by a comprehensive diagnostic evaluation, including laboratory tests and imaging studies as indicated [CS]

Evaluation of a child or adolescent who screens positive for an eating disorder should include complete psychiatric evaluation and physical examination. A comprehensive evaluation, in addition to components typically included in a psychiatric assessment, should include evaluation of weight and height, changes in body weight, menstrual history in females, body image concerns, presence of dieting and calorie counting, as well as the amount, type, and frequency of exercise. A registered dietician can be helpful in assessing specific dietary intake. Percentage of weight loss and rapidity of weight loss should also be evaluated.[130] In addition, binge eating or purging behaviors should be assessed including frequency, amount consumed, and duration of the behaviors. Evaluation of comorbid symptoms including depressive symptoms, anxiety symptoms, obsessional thoughts, self-injurious behavior, and suicidality is also necessary. Screening for psychotic symptoms and drug use is also recommended. Inquiry into label checking and self-weighing can be helpful if other symptoms of body image preoccupation are denied. Obtaining a history from parents is critical to corroborate symptoms.[26] Percentile BMIs are necessary to estimate levels of malnutrition in children and adolescents, given the inaccuracy of standard BMI calculations in this age group.[30] Methods for calculating percentile BMIs are available using the Centers for Disease Control and Prevention (CDC) age and gender growth charts.[31,131] Efforts should be made to obtain longitudinal growth charts, and deviations from individual growth trajectories should be evaluated.

The best-characterized and most commonly used structured interview for assessing disordered eating behaviors and eating-related psychopathology is the Eating Disorder Examination (EDE).[132,133] The EDE is reliable for patients down to 12 years of age. A child version for evaluating children and adolescents less than 14 years of age is also available.[134] There is also a self-report version (EDE-Questionnaire), which takes about 5 minutes to complete and is reliable in adolescents.[128,135,136] It is noteworthy that for adolescent AN, studies suggest that minimization and denial may produce unusually low scores on the EDE, despite the clinical presence of clear behaviors consistent with AN.[26] In this instance, parental report using the EDE interview provides scores similar to clinician ratings.[34] In addition to the EDE, several other commonly used questionnaires are available, including the Eating Attitudes Test (EAT),[126] Children's version of the Eating Attitudes Test (CHEAT),[137] and Eating Disorder Inventory (EDI).[138]

For adolescents with BN, the EDE appears to provide a good measure of eating-disordered behaviors and constructs, even without parent report.[34,132] Another measure specific to BN that may be considered is the Bulimia-Test–Revised (BULIT-R). Several assessment instruments are relevant for AN, BN, and BED, such as EDE, CHEAT,[126,127] EAT, and EDI.[132,137] A critical review of the strengths of these various instruments is available.[139]

In those patients in whom there is evidence of malnutrition or purging behaviors, initial laboratory testing typically includes a complete blood count, chemistry profile including electrolytes, blood urea nitrogen, creatinine, glucose, and liver functions including aspartate aminotransferase (AST) and alanine aminotransferase (ALT). These tests also can be used to monitor treatment. A thyroid-stimulating hormone (TSH) test should be ordered to rule out underlying thyroid dysfunction. Further blood testing should include measuring calcium, magnesium, phosphate, total protein, albumin, erythrocyte sedimentation rate (ESR), and amylase (as some studies suggest that elevations of amylase provide evidence that the patient is vomiting), B_{12}, and lipid profiles; in females, luteinizing hormone (LH), follicle-stimulating hormone (FSH), and estradiol levels should also be tested. If indicated, a pregnancy test using β–human chorionic gonadotropin (B-hCG) should also be considered to evaluate amenorrhea. Electrocardiograms are often necessary to further evaluate bradycardia and risk of cardiac arrhythmias. Dual-energy x-ray absorptiometry (DEXA) of bone should initially be conducted in females with amenorrhea lasting more than 6 months and yearly if amenorrhea persists.[140] All males with significant weight loss should also have a DEXA scan. Results of these data provide patients and families with guidance about the clinical impact of starvation on physical health and growth. Over time, improvements in these physical health parameters can help benchmark clinical progress. For example, for females with AN, normalization of estrogen levels can be a marker of healthy weight. At the same time, in more chronically ill patients with eating disorders, there can be a normalization of laboratory values that can suggest better health than is present.

Recommendation 3. Severe acute physical signs and medical complications need to be treated [CS]

Adverse physical effects of eating disorders include cardiac arrhythmias, bradycardia, hypotension, hypothermia, dehydration, congestive heart failure, kidney failure, pancreatitis, amenorrhea or irregular menses, low bone mineral density, neurological and cognitive impairments, delay in growth or growth impairment, pubertal delay or interruption, hormonal imbalances, and fluid and electrolyte abnormalities. In addition, clinical signs of malnutrition include hair loss, lanugo hair, dry skin, dependent edema, muscle weakness, and cramps.[78] Most physical abnormalities are reversible with adequate diet