Eating disorders: Overview of epidemiology, clinical features, and diagnosis

AUTHOR: Angela Guarda, MD
SECTION EDITOR: Joel Yager, MD
DEPUTY EDITOR: David Solomon, MD

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: **Oct 2023.**
This topic last updated: **May 13, 2023.**

INTRODUCTION

Eating disorders are characterized by a persistent disturbance of eating behavior that impairs health or psychosocial functioning [1]. The disorders include anorexia nervosa, avoidant/restrictive food intake disorder, binge eating disorder, bulimia nervosa, other specified feeding and eating disorder, unspecified feeding and eating disorder, pica, and rumination disorder. Diagnostic criteria are defined in the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR), which divides eating disorders into mutually exclusive categories based upon observed symptoms [1]. Some diagnoses include a dimensional component that allows clinicians to specify the severity of illness [1-4].

Sociocultural pressure for thinness may contribute to onset of eating disorders. However, these disorders are increasingly understood to have a biological basis arising from the interaction of individual genetic vulnerability and environmental factors. In addition, physiological consequences of starvation and of disordered eating behavior, disturbances in reward and appetitive neural circuits, and habitual and conditioned learning responses are recognized contributors to the compulsive nature of these disorders.

This topic reviews the epidemiology, pathogenesis, clinical features, and diagnosis of eating disorders. Treatment of eating disorders, medical complications of eating disorders and their

management, evaluation for medical complications and criteria for hospitalization, and the refeeding syndrome in anorexia nervosa are discussed separately.

- (See "Eating disorders: Overview of prevention and treatment".)
- (See "Anorexia nervosa in adults and adolescents: Medical complications and their management".)
- (See "Bulimia nervosa and binge eating disorder in adults: Medical complications and their management".)
- (See "Anorexia nervosa in adults: Evaluation for medical complications and criteria for hospitalization to manage these complications".)
- (See "Anorexia nervosa in adults and adolescents: The refeeding syndrome".)

EPIDEMIOLOGY

Meta-analyses of data from 94 studies worldwide found that the lifetime prevalence of an eating disorder in females was approximately 8 percent and in males 2 percent [5]. The most commonly ascertained eating disorder was other specified feeding and eating disorder, followed by binge eating disorder, bulimia nervosa, and anorexia nervosa. In addition, the point prevalence of all eating disorders appeared to double over the 18-year study period from 2000 to 2018, which may parallel rising rates of obesity worldwide.

Data from three nationally representative surveys of adults in the United States indicate that the lifetime prevalence of an eating disorder in females is approximately 5 percent and in males 2 percent [6]. The estimated one-year prevalence rates in females and males were 2 and 1 percent. Lifetime prevalence of any eating disorder was highest among individuals with obesity.

PATHOGENESIS

The causes of eating disorders are multifactorial and include a combination of biological, sociocultural, and psychological factors. Genetic risk appears strongest for anorexia nervosa, although bulimia nervosa and binge eating disorder also aggregate in families. Anorexia nervosa and bulimia nervosa appear highly correlated genetically based on a large Swedish twin study with a genetic correlation for broadly defined anorexia nervosa and bulimia nervosa of 0.79 and a unique environment correlation of 0.44 [7].

Risk factors for eating disorders can be nonspecific and associated with risk for psychiatric illness generally, or specific to eating disorders. Examples of nonspecific risk factors include exposure to physical and/or sexual trauma or problematic parenting behavior [8].

Following onset of eating disorders, factors that maintain/perpetuate the disorders include physiological and neurobiological complications of starvation and of binge/purge behavior, as well as conditioned learning and habit formation.

SCREENING

For patients with a normal or high body mass index, and no signs or symptoms of eating disorders, the evidence is insufficient to recommend for or against screening for eating disorders [9]. It is not known whether screening for eating disorders improves health outcomes [10], and the evidence is thus lacking to determine whether the benefits of screening outweigh the harms [9].

However, we suggest screening for eating disorders in primary care patients who are at increased risk of eating disorders, including [9,11,12]:

- Patients with a history of:
  - Adversity during childhood
  - Trauma
- Young adults
- Females
- Transgender individuals
- Athletes
- Patients who present with:
  - Signs or symptoms of eating disorders (eg, rapid weight loss, preoccupation with eating and appearance, bradycardia, or amenorrhea)
  - Anxiety disorders
  - Depressive disorders
  - Rigidity
  - Perfectionism

Screening is important because eating disorders are often undetected and thus untreated [11], and is consistent with practice guidelines from the American Psychiatric Association [13].

Clinicians can screen for eating disorders by asking the patient or family whether they have any concerns about the patient's weight, body shape, body image, or eating behaviors. In addition, relatively short and easy to interpret instruments that are suitable for screening in a primary care setting have been developed, which may help identify patients who need further evaluation [10,14,15].

If screening is implemented, we suggest the SCOFF questionnaire, which is the most commonly used instrument and is recommended by the United States Preventive Services Task Force [9,10]. The SCOFF consists of five clinician-administered questions [14,16]:

- Do you make yourself **S**ick because you feel uncomfortably full?
- Do you worry you have lost **C**ontrol over how much you eat?
- Have you recently lost more than **O**ne stone (14 pounds or 6.35 kg) in a three-month period?
- Do you believe yourself to be **F**at when others say you are too thin?
- Would you say that **F**ood dominates your life?

Answering "yes" to two or more questions is generally regarded as a positive screen; this cutoff provides good sensitivity and specificity for detecting anorexia nervosa and bulimia nervosa in young females. A positive screen should prompt further assessment to establish or rule out a diagnosis [17].

Evidence that supports using the SCOFF includes a meta-analysis of 25 studies with more than 11,000 individuals; the sensitivity was 0.86 and specificity 0.83 [18]. However, most studies enrolled young females with anorexia nervosa or bulimia nervosa and employed a case-control design [18]. Thus, the validity of the SCOFF in other populations or for other eating disorder diagnoses, such as binge eating disorder, is not established [11,12].

A reasonable alternative to the SCOFF is the clinician-administered, five-item Eating Disorder Screen for Primary Care (ESP) [12,15]:

- Are you satisfied with your eating patterns? (No is abnormal.)
- Do you ever eat in secret? (Yes is abnormal.)
- Does your weight affect the way you feel about yourself? (Yes is abnormal.)
- Have any members of your family suffered with an eating disorder? (Yes is abnormal.)

- Do you currently suffer with or have you ever suffered in the past with an eating disorder? (Yes is abnormal.)

A positive screen consists of two or more “abnormal” responses. In two studies (n = 627) of the ESP, the sensitivity ranged from 97 to 100 percent, and specificity from 40 to 71 percent [9,10].

A third screening instrument for eating disorders is the Eating Attitudes Test (EAT), which is one of the most widely used self-report eating disorder instruments [19]. The shorter 26-item version (EAT-26) (table 1) has been translated in many languages, validated in both clinical and nonclinical groups, and utilized internationally in studies of adolescents and adults. The reliability of the EAT-26 is high; however, low scores cannot be assumed to rule out an eating disorder because denial or minimization of eating disorder symptoms is common. The Ch-EAT is a modified version with simplified language for children age 8 to 13 [20].

The Primary Care Evaluation of Mental Disorders Patient Health Questionnaire (table 2) is a self-report instrument that is used to screen for and diagnose mental disorders in primary care settings [21]. It provides a categorical diagnosis for bulimia nervosa and binge eating disorder, as well as depressive, anxiety, alcohol, and somatoform disorders. The eating disorder module includes eight items. The instrument was specifically designed for use in primary care, has good diagnostic validity overall (sensitivity 75 percent, specificity 90 percent), excellent diagnostic validity for eating disorders (sensitivity 89 percent, specificity 96 percent), and the median clinician time to review the results is one to two minutes.

Additional information about assessing patients with a possible diagnosis of anorexia nervosa or bulimia nervosa is discussed separately. (See "Anorexia nervosa in adults: Clinical features, course of illness, assessment, and diagnosis", section on 'Assessment' and "Bulimia nervosa in adults: Clinical features, course of illness, assessment, and diagnosis", section on 'Assessment'.)

DIAGNOSTIC INSTABILITY

Eating disorders demonstrate significant transdiagnostic drift, such that patients with anorexia nervosa often crossover to bulimia nervosa [22]. In addition, diagnostic crossover between bulimia nervosa and binge eating disorder commonly occurs, and transition in and out of other specified feeding and eating disorder is also frequent. However, transition from anorexia nervosa to binge eating disorder is rare.

Additional information about diagnostic instability in eating disorders is discussed separately. (See "Anorexia nervosa in adults: Clinical features, course of illness, assessment, and diagnosis",

section on 'Diagnostic stability' and "Bulimia nervosa in adults: Clinical features, course of illness, assessment, and diagnosis", section on 'Diagnostic stability'.)

ANOREXIA NERVOSA

Epidemiology

- **Adults** – The estimated lifetime prevalence of anorexia nervosa in the United States adult general population is 0.8 percent, and the one-year prevalence is 0.5 percent [23]. A study of Finnish female twins found a higher lifetime prevalence of 2.2 percent [24]. Many estimates are likely to be low due to the ego syntonic nature of anorexia nervosa and the tendency of some individuals to conceal their illness [25].

  Anorexia nervosa is more common in females than males [5]. The lifetime prevalence in the general population is approximately 12 times greater in females than males (1.42 percent in females and 0.12 percent in males) [23]. In clinical settings, the ratio of females to males ranges from 10:1 [26] to 20:1 [27].

  The median age of onset for anorexia nervosa in the general population is 17 years [23].

- **Adolescents** – A nationally representative survey of adolescents (age 13 to 18 years) in the United States found that the lifetime prevalence of anorexia nervosa was 0.3 percent; the prevalence for females and males was identical (0.3 percent) [28].

Etiology and neurobiology — The etiology of anorexia nervosa includes both genetic and environmental contributions and is best thought of in terms of predisposing, precipitating, and maintaining factors.

- **Etiology**

  - **Predisposing factors** – Predisposing factors in anorexia nervosa include genetic predisposition. As with other psychiatric disorders, genetic influences are likely due to the cumulative, small effects of many distinct genes with pleiotropic effects, many of which influence vulnerability not only to anorexia nervosa but also to psychiatric disorders commonly comorbid with, or observed in relatives of probands with, anorexia nervosa [29]. These include anxiety disorders, obsessive-compulsive disorder, major depression, and substance use disorders, as well as bulimia nervosa [30,31].

    Support for genetic involvement is substantial:

- Anorexia nervosa aggregates in families, with an 11-fold risk of the disorder in female relatives of a proband with the disorder, compared with female relatives of never-ill probands [32].

- Based upon twin studies comparing monozygotic and dizygotic twins, the proportion of phenotypic variation for anorexia nervosa that is explained by genetic factors (heritability) ranges from 16 to 74 percent [31,33].

- Additional evidence supporting a genetic basis for anorexia nervosa comes from genome-wide association studies (GWAS). The first GWAS study included nearly 3500 individuals with a lifetime diagnosis of anorexia nervosa and close to 11,000 controls. This study estimated that the common genetic variant based heritability of anorexia nervosa was approximately 20 percent [34]. In addition, positive genetic correlations were noted between anorexia nervosa and neuroticism, perfectionism, schizophrenia, and educational attainment, suggesting that the same genes are involved across these phenotypes. Negative genetic correlations were found with metabolic and anthropometric traits associated with obesity, including high body mass index (BMI), fasting insulin, and fasting glucose.

  A follow-up GWAS included over 16,000 cases and 55,000 controls from 17 countries and found eight loci exceeding genome wide significance [35]. The eight loci included genes on chromosomes 1, 3, 10, and 11. The subsequent study also found positive genetic correlations between anorexia nervosa and obsessive-compulsive disorder, major depression, schizophrenia, neuroticism, and educational attainment.

Other well-established risk factors for anorexia nervosa include sociocultural pressures for thinness, elevated shape and weight concerns, dietary restraint, exercise, and family history of eating and weight control behaviors [36].

Childhood maltreatment is associated with psychiatric problems and may contribute to onset of anorexia nervosa. Several meta-analyses in patients with eating disorders have examined maltreatment before age 18 years. Compared with healthy controls, patients with eating disorders were approximately two to four times more likely to have been abused, depending upon the specific type of abuse [8]. However, it is unclear to what extent abuse is involved as a predisposing vulnerability to various eating disorders, or contributes to their onset or exacerbation.

- **Precipitating factors** – Risk factors for the onset of anorexia nervosa include premorbid low BMI and dieting behavior, which suggests that inherent

leanness/constitutional thinness precipitates the illness [37]. In addition, excessive exercise is a common symptom of anorexia nervosa, which often precedes onset of dieting behavior.

- **Maintaining factors** – Restrictive eating in anorexia nervosa initially appears to be rewarding but becomes increasingly compulsive, and may be maintained by aberrant decision making around food choice and by habit formation [38,39]. In addition, alterations in hormones and neuropeptides (eg, ghrelin, leptin and agouti-related peptide) may possibly be involved [38]. Other maintaining factors that may contribute to perpetuating restrictive eating behavior in anorexia nervosa include gastrointestinal complications of starvation or binge purge behaviors, including early satiety, gastroparesis, gastrointestinal reflux, and constipation [40]. (See "Anorexia nervosa in adults and adolescents: Medical complications and their management", section on 'Gastrointestinal'.)

- **Neurobiology** – Multiple lines of evidence demonstrate altered brain structure and function in anorexia nervosa [41]. As an example, structural and functional neuroimaging studies employing a range of food and body image stimuli suggest abnormalities and dysfunction in brain reward circuitry [38]. However, cross-sectional studies cannot determine whether abnormalities (eg, neuroimaging findings) represent etiologic causes, sequelae, neither, or both. In addition, the study samples are usually small. Some structural and functional changes reverse following weight restoration, while others persist and may represent scar effects that contribute to chronicity.

  Multiple neuroimaging studies have shown structural brain changes in patients with anorexia nervosa. (See "Anorexia nervosa in adults and adolescents: Medical complications and their management", section on 'Neurologic'.)

  Functional magnetic resonance imaging (MRI) studies suggest that abnormal functioning of different brain areas may contribute to onset or maintenance of anorexia nervosa [41]. As an example:

  - Abnormal functioning of corticolimbic reward circuits involved in appetite may contribute to anorexia nervosa. A study examined responses to tasting sucrose in patients recovered from anorexia nervosa (n = 14) and in healthy controls (n = 14); blood flow in the right anterior insula was diminished in the patients, compared with controls [42]. This suggests that restricted eating and weight loss may occur in anorexia nervosa because hunger signals are not accurately recognized (the anterior insula is thought to integrate sensory aspects of feeding).

- A second study scanned 26 females in remission from anorexia nervosa and 22 healthy controls while tasting sucrose or ionic water, either during the fasted or fed state. In the fasted state, females with remitted anorexia nervosa and control females had opposite responses to tastants in the left ventral caudate. The group with remitted anorexia nervosa responded less to tastants in the fasted state than fed state, whereas the controls responded more to tastants in the fasted state than fed state. This finding suggested that anorexia nervosa is characterized by decreased neural sensitivity to the reward value of food taste during hunger, which may facilitate food avoidance in anorexia nervosa [43].

- Another study compared inpatients with anorexia nervosa (n = 21) to healthy controls (n = 21) while they completed a food choice task in the scanner. Patients with anorexia nervosa were more likely to choose low-fat foods, and activity in the dorsal striatum was greater among patients than controls [44]. In addition, connectivity in fronto-striatal circuits differed between groups. The dorsal striatum is involved in habitual behavior, and the study suggests that maladaptive food choices and the avoidance of high-calorie foods in anorexia nervosa can become well-established behaviors subserved by fronto-striatal networks.

Neurotransmitter systems are also disrupted in anorexia nervosa. Deficits have been found in dopaminergic function, which is thought to be involved with eating behavior, motivation, and reward [41]. Other deficits have been found in serotonergic function; serotonin is involved with mood, impulse control, and obsessional behavior.

Clinical features — The clinical features of anorexia nervosa are discussed separately. (See "Anorexia nervosa in adults: Clinical features, course of illness, assessment, and diagnosis".)

Diagnosis — According to the DSM-5-TR, the diagnosis of anorexia nervosa requires each of the following (table 3) [1]:

- Restriction of energy intake that leads to a low body weight, given the patient's age, sex, developmental trajectory, and physical health

- Intense fear of gaining weight or becoming fat, or persistent behavior that prevents weight gain, despite being underweight

- Distorted perception of body weight and shape, undue influence of weight and shape on self-worth, or denial of the medical seriousness of one's low body weight

Additional information about the diagnosis is discussed separately. (See "Anorexia nervosa in adults: Clinical features, course of illness, assessment, and diagnosis", section on 'Diagnosis'.)

Medical complications and evaluation — The starvation and persistent purging that occur with anorexia nervosa can lead to many serious medical complications. (See "Anorexia nervosa in adults and adolescents: Medical complications and their management".)

All patients with anorexia nervosa should be evaluated for medical complications (table 4) [13,17]. The evaluation should include a history, physical examination, and focused laboratory testing. The evaluation for medical complications and criteria for hospitalization to manage these complications are discussed separately. (See "Anorexia nervosa in adults: Evaluation for medical complications and criteria for hospitalization to manage these complications".)

AVOIDANT/RESTRICTIVE FOOD INTAKE DISORDER

The diagnosis of avoidant/restrictive food intake disorder (ARFID) was introduced in 2013 [45]. Due to the relatively recent definition of the disorder and its heterogeneous clinical presentation, knowledge regarding the prevalence, comorbidity, and course of illness is limited [46].

Epidemiology

- **General population** – The point prevalence of ARFID in community settings is not clear because relatively few population studies have been performed. In one interview-based study of individuals age 15 years and up, the point prevalence was 0.3 percent [47]. A second study of children age 8 to 13 years used a self-report screening instrument and found a point prevalence of 3 percent [48].

- **Clinical settings** – The prevalence of ARFID is often higher in clinical settings than the general population, and varies depending upon the specific clinical setting:

  - Retrospective chart reviews of pediatric patients presenting to an eating disorders program in different countries found rates of ARFID ranging from 5 to 22 percent [49]. Higher rates of 32 to 64 percent were observed in pediatric feeding disorders programs.

  - A retrospective study of youth age 8 to 18 years who presented to gastroenterology clinics (n >2200) found ARFID in 1 percent [50]; a similar study in adults presenting to a tertiary referral neurogastroenterology clinic found a point prevalence of ARFID of 6 percent [51]. In the latter study, as well as in a study of underweight inpatients

(average age 19) with ARFID treated in an eating disorders behavioral weight restoration program (n = 27), the majority reported food avoidance symptoms related to fear of gastrointestinal consequences of eating. A common symptom was noninduced vomiting [52].

In contrast to anorexia nervosa, bulimia nervosa, or binge eating disorder, the prevalence of ARFID appears higher in males than females, and the average age of onset is younger [49,53-55]. As an example, a retrospective chart review found that among patients who presented to eating disorder programs, the average age was less in those with ARFID (n = 98) than those with anorexia nervosa (n = 98) or bulimia nervosa (n = 66; 12.9 versus 15.6 and 16.5 years) [53]. However, ARFID may onset during adulthood [49,53-55].

Etiology and neurobiology — Relatively little is known about the etiology and neurobiology underlying ARFID.

Clinical features — Although ARFID is a heterogeneous condition, the core clinical feature is restriction in the variety or amount of food consumed, which leads to a persistent failure to meet nutritional and/or energy needs [1].

Food restriction and/or avoidance is not driven by weight or body shape concerns, as is the case with other eating disorders such as anorexia nervosa. Whereas individuals with anorexia nervosa fear weight gain and tend to avoid consuming calorie dense, high-energy foods, those with ARFID often preferentially consume a limited variety of processed, calorie dense, high-energy food.

Factors that may be associated with food avoidance in ARFID include lifelong, low appetitive drive; sensory specific aversion to certain foods (taste, texture, temperature, smell, or appearance); or anxiety regarding the consequences of eating (eg, fear of gastrointestinal aversive consequences of eating) [51]. In a study of underweight inpatients with ARFID treated in an eating disorders behavioral weight restoration program (n = 27), approximately 80 percent reported food avoidance symptoms related to fear of gastrointestinal consequences of eating [52]. A common symptom was noninduced vomiting.

ARFID results in caloric and/or micronutrient nutritional insufficiency and the disorder is more severe than the common picky eating seen in children. Patients with ARFID are often underweight, although the disorder can occur at any weight [54]. Individuals with ARFID often present first to nonpsychiatric specialists, including pediatricians and gastroenterologists, rather than psychiatrists.

Among patients with ARFID, psychiatric comorbidities are common, especially anxiety disorders such as generalized anxiety disorder [54,56,57]:

- In one cross-sectional study of 74 youth with ARFID, at least one comorbidity was identified in 45 percent [58]. Generalized anxiety disorder was observed most often, followed by panic disorder and social anxiety disorder.
- A retrospective study of 98 youth with ARFID found that comorbid anxiety disorders were present in approximately 60 percent; nearly 30 percent had generalized anxiety disorder [53].

Other psychiatric comorbidities in ARFID include attention deficit hyperactivity disorder, autism spectrum disorder, mood disorders, and internet gaming disorder [49].

Medical complications — Underweight patients with ARFID are at risk of medical complications related to malnutrition, similar to those seen in anorexia nervosa, such as cardiac, endocrine, and gastrointestinal complications [56]. Children with ARFID and severe low weight are at risk for stunted growth. In addition, ARFID characterized by vomiting can lead to electrolyte abnormalities. A retrospective study of 98 patients with ARFID found that a general medical disorder or symptom was present in approximately 50 percent [53].

Course of illness — Due to the relatively recent definition of ARFID, little is known about its course of illness. However, one clearly established aspect is that the disorder can persist into adulthood [1]. As an example, one retrospective study identified 19 youth with ARFID who were less than 18 years old, and assessed outcomes at a mean age of 25 years [57]. Mean body mass index was 22, and persistent ARFID was present in 26 percent (n = 5).

Diagnosis — We suggest diagnosing ARFID according to the criteria in the DSM-5-TR [1]. The diagnosis requires each of the following:

- Avoiding or restricting food intake, which may be based upon one or more of the following:
  - Lack of interest in food or low appetitive drive
  - Aversion or disgust to sensory characteristics of certain foods or food types
  - A conditioned negative response (eg, anxiety or disgust) associated with food intake following an aversive experience (eg, choking, vomiting, or abdominal pain)

- Restriction in the amount or types of foods eaten leads to a persistent failure to meet nutritional and/or energy needs, manifested by at least one of the following:
  - Clinically significant weight loss, or in children, poor growth or failure to achieve expected weight gain
  - Nutritional deficiency
  - Supplementary enteral feeding or oral nutritional supplements are required to provide adequate intake
  - Impaired psychosocial functioning
- The eating or feeding disturbance is not due to lack of available food or associated with a culturally sanctioned practice.
- The disturbance does not occur solely during the course of anorexia nervosa or bulimia nervosa, and body weight and shape are not distorted.
- The disturbance is not due to a general medical condition (eg, gastrointestinal disease, food allergies, or occult malignancy) or another psychiatric disorder. When ARFID occurs in the context of another illness, the symptoms of ARFID are both out of proportion to what is expected for the other illness and warrant additional clinical attention.

ARFID can also be diagnosed according to the criteria in the World Health Organization's International Classification of Diseases-11th Revision (ICD-11) [59]. The ICD-11 criteria closely resemble those of the DSM-5-TR [1].

BINGE EATING DISORDER

Epidemiology

- **Adults** – Based upon pooled results from surveys of adults in 14 countries, the estimated lifetime prevalence of binge eating disorder is 1.9 percent and the 12-month prevalence is 0.8 percent [60]. In the United States, the estimated prevalence of binge eating disorder appears to be lower; a nationally representative survey found lifetime and 12-month prevalence rates of 0.9 and 0.4 percent [23].

  Studies consistently find a two- to threefold higher prevalence of binge eating in females than in males [6,23,60]. As an example, a meta-analysis of 94 worldwide studies found that

the lifetime prevalence of binge eating disorder in females was 2.8 percent and in males was 1 percent [5].

The prevalence of binge eating disorder increases with increasing weight [61]. Generally, prevalence rates are highest in obese individuals. In the population with obesity, rates of binge eating disorder are 10-fold higher in those with class III obesity (body mass index [BMI] ≥40 kg/m^2) than those with class I obesity (BMI = 30 to 34.9 kg/m^2) [6].

The median age of onset of binge eating disorder is approximately 21 years [23].

- **Adolescents** – A nationally representative survey of adolescents (age 13 to 18 years) in the United States found that the lifetime prevalence of binge eating disorder was 1.6 percent. As with adults, the prevalence was two to three times greater in females than males (2.3 versus 0.8 percent) [28].

Etiology, pathogenesis, and neurobiology — A genetic component appears to be involved in the etiology of binge eating disorder. One review of twin studies estimated that the proportion of phenotypic variation for binge eating disorder explained by genetic factors (heritability) is 39 to 45 percent [31].

Childhood maltreatment is associated with psychiatric problems and may contribute to onset of binge eating disorder. Several meta-analyses in patients with eating disorders have examined maltreatment before age 18 years. Compared with healthy controls, patients with eating disorders were approximately two to four times more likely to have been abused, depending upon the specific type of abuse [8]. However, it is unclear to what extent abuse is involved as a predisposing vulnerability to various eating disorders, or contributes to their onset or exacerbation.

Although the pathogenesis of binge eating disorder is poorly understood, multiple studies have found reduced inhibitory control and higher impulsivity towards salient palatable food stimuli in patients with binge eating disorder, compared to non-binge eating controls with obesity [62,63]. These traits may contribute to the compulsive nature of binge eating behavior and the inability to interrupt a binge. In addition, an increase in reward driven hedonic drive to eat (which overrides homeostatic hunger and satiety signaling) is proposed to contribute to binge behavior [64].

Neuroimaging studies indicate that compared with overweight or healthy controls, individuals with binge eating disorder are more responsive to food cues and show increased blood-oxygen-level-dependent activation in reward-related brain regions when presented with food stimuli

[65]. It is unclear whether this hyper-responsiveness is trait-related or is a consequence of binge eating behavior, which in turn may contribute to maintaining the behavior once established.

One magnetic resonance imaging (MRI) study found evidence of structural brain changes with increased grey matter volumes in the anterior cingulate cortex and the medial orbitofrontal cortex, brain regions involved in reward-learning [66].

Clinical features

- **Core features** – The core features of binge eating disorder are [1]:
  - Binge eating – Recurrent episodes of consuming a large amount of food (eg, >1000 kcal in a discrete period of time [eg, less than two hours]).
  - Binge eating episodes occur at least weekly.
  - Regular compensatory behaviors (fasting, purging, or excessive exercise) are not present.
  - Antecedents to binge eating often include distressing emotions or environmental or interpersonal stressors.
- **Associated features** – The associated features of binge eating disorder are [1]:
  - Binge eating disorder is more common in individuals with obesity but can occur at normal weights [6]. Among individuals in the community with binge eating disorder, approximately 50 percent are overweight or obese, similar to the community at large [67,68]. The remaining individuals with binge eating disorder are of normal weight, and are less likely to present for treatment [69,70].
  - Psychosocial impairment is common. Surveys from 14 countries, including the United States, identified individuals with binge eating disorder during the past 12 months, and found that impairment (mild, moderate, or severe) occurred in approximately 50 percent [23,60]. Severe impairment was reported by approximately 15 and 25 percent, depending upon the specific study.
  - Negative emotions (eg, anger or dysphoria) and difficulties with emotion regulation are frequently observed in individuals with binge eating disorder and often appear to precede binge eating episodes. Impulsivity and negative urgency, or the tendency to act rashly in response to emotion, also appear to be common in binge eating disorder [71,72].

Comorbidity

- **Psychiatric** – Comorbid psychopathology often occurs in binge eating disorder. Nearly 80 percent of individuals with binge eating disorder have a lifetime history of at least one other psychiatric disorder, and nearly 50 percent have a lifetime history of three or more comorbid disorders [67].

  In a nationally representative survey from the United States, the frequency of specific psychiatric comorbidities among individuals with binge eating disorder exceeded that in the general population for each comorbid condition assessed and was as follows [73]:

  - Unipolar major depression – 66 percent
  - Any anxiety disorder – 59 percent
  - Any personality or conduct disorder – 56 percent
  - Alcohol use disorder – 52 percent
  - Posttraumatic stress disorder – 32 percent

  The frequency of specific personality disorders was examined in a meta-analysis of nine studies, that included 838 patients with binge eating disorder, nearly all of whom were assessed with structured clinical interviews [74]:

  - Any personality disorder – 29 percent of patients
  - Avoidant personality disorder – 12 percent
  - Borderline personality disorder – 10 percent
  - Obsessive-compulsive personality disorder – 10 percent

- **Nonpsychiatric** – Nonpsychiatric (general medical) disorders are common in binge eating disorder and are not fully explained by comorbid obesity or psychiatric comorbidity.

  The association between binge eating disorder and nonpsychiatric disorders has been observed in multiple studies that included community and clinical samples from different countries, and compared individuals with binge eating disorder to controls without binge eating disorder [60,73,75,76]. Some of the analyses were adjusted for potential confounding factors such as age, sex, and comorbid psychiatric illnesses. In at least two studies, the following nonpsychiatric disorders were more likely to occur in individuals with binge eating disorder than controls:

  - Arthritis or other musculoskeletal system diseases (odds ratios 1.5 and 1.7)
  - Asthma and other respiratory diseases (odds ratios 1.3 and 2.1)
  - Chronic pain (odds ratios ranging from 1.5 to 2)

- Diabetes mellitus (odds ratios ranging from 1.6 to 5.8)
- Hypertension or other circulatory system diseases (odds ratios ranging from 1.4 to 2)

Among patients with binge eating disorder who are obese, the effect of binge eating cessation on subsequent weight loss and/or medical comorbidities of obesity is not clearly established [77].

Course of illness — Development of binge eating disorder occurs in a variety of ways. A study of 284 patients with binge eating disorder found that becoming overweight usually occurred first, compared with dieting or binge eating first (63 versus 21 and 16 percent of patients) [78].

The course of illness among patients with binge eating disorder can be chronic [67]. In a retrospective study of 62 patients who were hospitalized for binge eating disorder and assessed 12 years later, 31 percent still met criteria for an eating disorder diagnosis [79]. Psychiatric comorbidity, body dissatisfaction (undue influence of body weight or shape), impulsivity, and a history of sexual abuse are associated with a poor outcome [79,80].

Nevertheless, course of illness in binge eating disorder appears to be more favorable in the short-term than that of anorexia nervosa or bulimia nervosa, with remission rates in binge eating disorder ranging from approximately 20 to 75 percent [81]. In addition, a six-year, prospective follow-up of 283 outpatients with binge eating disorder who were initially treated with cognitive-behavioral therapy found that nearly 60 percent recovered and remained stable [22].

Suicide attempts are common in binge eating disorder. A nationally representative survey of adults in the United States estimated that the prevalence of suicide attempts in individuals with binge eating disorder was 23 percent [82]. In addition, suicide attempts were four to five times more likely to occur in those with binge eating disorder than those without an eating disorder.

Adolescents with binge eating disorder are also at increased risk of suicide attempts [28].

When to suspect the disorder — Binge eating disorder can be difficult to detect because patients often feel ashamed by their behavior [83]. The presence of the disorder is suggested by clues such as:

- Greater than expected weight dissatisfaction
- Large weight fluctuations
- Depressive symptoms

Inquiring sensitively but directly about experiencing a sense of loss of control over eating, examples of the quantity and type of food eaten during a recent binge, and other diagnostic

features is important to making the diagnosis.

Diagnosis — In the DSM-5-TR, the diagnosis of binge eating disorder requires each of the following [1]:

- Episodes of binge eating, defined as consuming an amount of food in a discrete period (eg, two hours) that is clearly larger than what most people would eat in a similar amount of time under similar circumstances. During episodes, patients feel they lack control over their eating (eg, patients feel they cannot stop eating or control the amount or what they are eating).
- Binge eating episodes are marked by at least three of the following:
  - Eating more rapidly than normal
  - Eating until feeling uncomfortably full
  - Eating large amounts of food when not feeling physically hungry
  - Eating alone because of embarrassment by the amount of food consumed
  - Feeling disgusted with oneself, depressed, or guilty after overeating
- Patients experience marked distress regarding binge eating
- Episodes occur, on average, at least once a week for three months.
- No regular use of inappropriate compensatory behaviors (eg, purging, fasting, or excessive exercise) as are seen in anorexia nervosa and bulimia nervosa.
- Binge eating does not occur solely during the course of bulimia nervosa or anorexia nervosa.

The current level of severity is based upon the number of binge eating episodes per week:

- Mild – 1 to 3
- Moderate – 4 to 7
- Severe – 8 to 13
- Extreme – 14 or more

Binge eating disorder can also be diagnosed according to the criteria in the ICD-11 [59]. The ICD-11 criteria closely resemble those of the DSM-5-TR [1]. However, one difference is the requirement in ICD-11 that the symptoms are not better accounted for by another psychiatric disorder (eg, unipolar major depression) or nonpsychiatric disorder (eg, Prader-Willi syndrome).

The diagnosis of binge eating disorder may change over time. A national registry study of patients with binge eating disorder found that over one year, 16 percent transitioned to another eating disorder diagnosis, most commonly bulimia nervosa or other specified feeding and eating disorder [84].

The diagnostic stability of adolescent binge eating disorder is low. A prospective study of adolescents (n >1500) found that binge eating disorder at age 14 was associated with bulimia nervosa at age 17, and that binge eating disorder at age 17 was associated with bulimia nervosa at age 20 [85].

BULIMIA NERVOSA

Epidemiology

- **Adults** – Based upon pooled results from surveys of adults in 14 countries, the estimated lifetime prevalence of binge eating disorder is 1 percent, and the 12-month prevalence is 0.4 percent [60]. In the United States, a nationally representative epidemiologic study found a lifetime prevalence rate of 0.28 percent and a 12-month prevalence of 0.14 percent [23]. Prevalence estimates of bulimia nervosa are likely low due to the tendency of some individuals to conceal their illness out of shame or perceived stigma [25].

  Bulimia nervosa is more common in females than males [60]. Nationally representative surveys in the United States have found that the lifetime prevalence of bulimia nervosa is at least three times higher in females than males [5,6,23,67]. One such survey found that the lifetime prevalence in females was approximately 0.5 percent and in males 0.1 percent [23]. In clinical settings, the ratio of females to males with a first-time diagnosis is much higher (13 to 1), as males are less likely to present for treatment [26].

  The median age of onset of bulimia nervosa is 16 years [23].

- **Adolescents** – A nationally representative survey of adolescents (age 13 to 18 years) in the United States found that the lifetime prevalence of bulimia nervosa was 0.9 percent; the prevalence was greater in females than males (1.3 versus 0.5 percent) [28].

Etiology and neurobiology — A genetic component appears to be involved in the etiology of bulimia nervosa. One review of twin studies estimated that the proportion of phenotypic variation for bulimia nervosa explained by genetic factors (heritability) is 28 to 83 percent [31].

Disinhibition, increased salience of palatable food stimuli, and impairments in emotional self-regulation are characteristic of bulimia nervosa. In addition, an increase in reward driven

hedonic drive to eat (which overrides homeostatic hunger and satiety signaling) is proposed to contribute to binge behavior in bulimia nervosa [64].

Well-established risk factors for bulimia nervosa include sociocultural pressures for thinness, elevated shape and weight concerns, dietary restraint, exercise, and family history of eating and weight control behaviors [36].

Childhood maltreatment is associated with psychiatric problems and may contribute to onset of bulimia nervosa. Several meta-analyses in patients with eating disorders have examined maltreatment before age 18 years. Compared with healthy controls, patients with eating disorders were approximately two to four times more likely to have been abused, depending upon the specific type of abuse [8]. However, it is unclear to what extent abuse is involved as a predisposing vulnerability to various eating disorders, or contributes to their onset or exacerbation.

Multiple studies demonstrate altered brain structure and function in bulimia nervosa [86]. As an example, structural and functional neuroimaging studies employing a range of food and body image stimuli suggest abnormalities and dysfunction in brain reward circuitry [38]. In addition, the studies collectively support a relationship between illness severity and neural changes. However, it is not clear whether any of the observed changes are etiologic and lead to bulimia nervosa, or whether the changes represent consequences of the disorder.

Magnetic resonance imaging (MRI) studies have shown structural brain changes in bulimia nervosa, including frontal and temporoparietal areas:

- One study examined gray and white matter volumes in patients with active bulimia nervosa (n = 20) and in healthy controls (n = 24); after controlling for potential covariates (eg, age, anxiety, depression, and medications), the analyses found that the medial orbitofrontal cortex and antero-ventral insula was larger in patients than controls [87]. In addition, temporal and parietal areas were reduced in patients.

- A second study compared age matched patients (n = 34) and healthy controls (n = 34), and found that the cerebral surface of the frontal and temporoparietal cortical areas was reduced in patients [88]. In addition, reductions were greater in patients with more episodes of bingeing and purging, more preoccupation with shape and weight, and a longer duration of illness. Reduction of cerebral surfaces was also associated with poorer neuropsychological functioning.

- A study of 62 females with full and subthreshold bulimia nervosa examined subcortical structures associated with reward circuits and found that many structures exhibited

localized surface deformations [89]. The deformations were more pronounced in older patients, and were associated with greater symptom severity, suggesting these shape abnormalities may serve as biomarkers of the condition and have prognostic significance.

In addition, abnormal functioning of corticolimbic circuits involved in appetite may contribute to bulimia nervosa. A functional MRI study examined responses to tasting sucrose in patients who recovered from bulimia nervosa (n = 14) and in healthy controls (n = 14); blood flow in the right anterior insula was elevated in the patients, compared with controls [42]. This suggests that overeating may occur in bulimia nervosa because hunger and satiety states are not accurately recognized (the anterior insula is thought to integrate sensory aspects of feeding).

Further, alterations in hormones and neuropeptides (eg, ghrelin, leptin, and agouti-related peptide) may possibly be involved in the pathogenesis of bulimia nervosa [38].

Clinical features — The clinical features of bulimia nervosa are discussed separately. (See "Bulimia nervosa in adults: Clinical features, course of illness, assessment, and diagnosis".)

Diagnosis — The DSM-5-TR criteria for bulimia nervosa include recurrent episodes of both binge eating and inappropriate compensatory behavior to prevent weight gain, occurring on average at least once per week for three months (table 5) [1]. (See "Bulimia nervosa in adults: Clinical features, course of illness, assessment, and diagnosis", section on 'Diagnosis'.)

Medical complications and evaluation — Persistent purging can lead to many medical complications, including dehydration, hypokalemia, menstrual irregularities, Mallory-Weiss syndrome, ipecac-induced myopathy, and erosion of dental enamel [90-93]. Electrocardiogram changes can occur as well. The medical complications of bulimia nervosa and their management are discussed in detail separately. (See "Bulimia nervosa and binge eating disorder in adults: Medical complications and their management".)

Patients with bulimia nervosa should be evaluated for medical complications that are secondary to persistent purging [90,93]. The evaluation includes a history, physical examination, and laboratory testing. The evaluation for medical complications is discussed separately, and the criteria for hospitalization to manage these complications are discussed separately in the context of anorexia nervosa. (See "Bulimia nervosa and binge eating disorder in adults: Medical complications and their management" and "Anorexia nervosa in adults: Evaluation for medical complications and criteria for hospitalization to manage these complications", section on 'Inpatient hospitalization'.)

PICA

- **Epidemiology** – The limited epidemiological studies on pica include one with 1430 youth age 7 to 13 years and another with 804 youth age 7 to 14 years; both found a similar prevalence of 5 percent [94,95]. Onset most often occurs during childhood but can also occur during adolescence or adulthood [1,59]. Risk factors for pica include neglect and developmental delay.

  The prevalence of pica in males and females is similar [59].

- **Clinical features** – Pica is associated with higher rates of eating disorder pathology, including body dissatisfaction, fear of weight gain, eating restraint, and binge eating or purging behaviors; also, those with elevated pica symptoms are more likely to exhibit symptoms of avoidant/restrictive food intake disorder [94]. (See 'Avoidant/restrictive food intake disorder' above.)

  Pica may be a clinical manifestation of iron deficiency anemia. (See "Causes and diagnosis of iron deficiency and iron deficiency anemia in adults", section on 'Pica and ice craving'.)

  In addition, pica is common in pregnancy, particularly in areas where nutritional deficiencies are more prevalent. The epidemiology, potential consequences, and management of pica in pregnancy is discussed separately. (See "Pica in pregnancy".)

- **Course of illness** – The course of illness in pica varies and in some patients is chronic [1]. Among patients with a variable course, increased stress and anxiety may precede pica behavior [59].

- **Diagnosis** – In the DSM-5-TR, the diagnosis of pica requires each of the following [1]:

  - Repeated eating of nonfood substances (eg, cloth, dirt, gum, hair, metal, paint, paper, or soap) that are not nutritional, for at least one month.

  - The eating behavior is:

    - Inappropriate for the patient's developmental level
    - Not culturally supported or socially normal

  - If the eating behavior occurs solely in the context of another mental disorder (eg, autism, intellectual disability, or schizophrenia) or general medical condition (including pregnancy), the eating behavior is sufficiently severe to warrant additional clinical attention.

The DSM-5-TR distinguishes pica from nonsuicidal self-injurious behaviors in which patients swallow potentially harmful objects (eg, batteries, knives, or needles) [1].

Pica can also be diagnosed according to the criteria in the ICD-11 [59]. The ICD-11 criteria resemble those of the DSM-5-TR [1]. However, one difference is the requirement in ICD-11 that the symptoms are not due to a nonpsychiatric disorder (eg, nutritional deficiency).

RUMINATION DISORDER

Rumination syndrome is a functional disorder characterized by effortless regurgitation of recently ingested food into the mouth after most meals. The material is either spat out or reswallowed [1]. (See "Rumination syndrome".)

The DSM-5-TR diagnosis of rumination disorder requires each of the following [1]:

- Repeated effortless regurgitation of recently ingested food, which may be rechewed, reswallowed, or spit out; the eating disturbance occurs for at least one month.
- Regurgitation of food is not due to a general medical condition, such as gastroesophageal reflux disease or postviral gastroparesis. (See "Clinical manifestations and diagnosis of gastroesophageal reflux in adults" and "Approach to the infant or child with nausea and vomiting".)
- Regurgitation does not occur solely during the course of avoidant/restrictive food intake disorder, anorexia nervosa, binge eating disorder, or bulimia nervosa.
- If the eating behavior occurs in the context of another mental disorder (eg, intellectual disability) or general medical condition (including pregnancy), the severity of the eating behavior warrants additional clinical attention.

OTHER SPECIFIED FEEDING OR EATING DISORDER

Diagnosis — Other specified feeding or eating disorder applies to patients with symptoms that cause significant distress or impair psychosocial functioning, but do not meet the full criteria for a specific feeding and eating disorder [1]. Clinicians record the diagnosis “other specified feeding or eating disorder,” followed by the reason that the presentation does not meet full criteria for an eating disorder. Examples of syndromes that can be specified when using the diagnosis other specified feeding or eating disorder include:

- Atypical anorexia nervosa – All the criteria for anorexia nervosa (table 3) are met, except that body mass index is ≥18.5 kg/m^2. One example is a patient with obesity who demonstrates the signs and symptoms of anorexia nervosa during rapid weight loss to a normal weight; the diagnosis is "other specified feeding or eating disorder, atypical anorexia nervosa."

  The medical complications of atypical anorexia nervosa can be comparable to those of anorexia nervosa. A randomized trial enrolled adolescent inpatients with either anorexia nervosa (n = 66) or atypical anorexia nervosa (n = 50) to study of meal-based refeeding for weight restoration [96]. Findings from the study included the following:

  - Eating disorder psychopathology was greater in the group with atypical anorexia nervosa than the group with anorexia nervosa.

  - Independent of admission weight, faster weight loss prior to admission was associated with lower heart rate and greater weight loss. Longer duration of loss prior to admission was also associated with lower serum phosphate. These findings indicate that significant medical risks of malnutrition are observed in atypical anorexia nervosa as well as anorexia nervosa across a range of body weights and are related to recent rate of weight loss and duration of loss [96].

- Bulimia nervosa of low frequency and/or limited duration – All of the criteria for bulimia nervosa (table 5) are met, except that episodes of binge eating and inappropriate compensatory behavior occur, on average, less than once per week and/or for less than three months.

- Binge eating disorder of low frequency and/or limited duration – All of the criteria for binge eating disorder are met, except that episodes of binge eating occur, on average, less than once per week and/or for less than three months.

- Purging disorder – Recurrent episodes of purging (self-induced vomiting, or misuse of laxatives, diuretics, or enemas) to influence body weight or shape, in the absence of binge eating.

- Night eating syndrome – Recurrent episodes of night eating, defined as eating after awakening from sleep or eating excessively after the evening meal. The night eating is not explained by changes in the sleep-wake cycle (eg, night shift work), medication effects, binge eating disorder, substance use disorders, or general medical disorders.

Mortality — Mortality may be increased in patients with the DSM-5-TR diagnosis of other specified feeding or eating disorder. However, the relevant studies of mortality examined patients diagnosed with "eating disorder not otherwise specified," a broader diagnosis included in previous versions of the DSM, which overlaps only partially with other specified feeding or eating disorder. (See 'Diagnosis' above.)

UNSPECIFIED FEEDING OR EATING DISORDER

The diagnosis "unspecified feeding or eating disorder" applies to patients with symptoms of a feeding and eating disorder that cause significant distress or impair psychosocial functioning, but do not meet the full criteria for a specific eating disorder [1]. This diagnosis is used when clinicians decide to not specify the reason that the presenting syndrome does not meet the full criteria for an eating disorder, including situations in which there is insufficient information to make a more specific diagnosis (eg, in the emergency department).

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Eating disorders".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Anorexia nervosa (The Basics)" and "Patient education: Bulimia nervosa (The Basics)")

SUMMARY

- **Screening** – The SCOFF is a five-item, clinician-administered measure that can be used to screen for anorexia nervosa and bulimia nervosa. (See 'Screening' above.)

- **Anorexia nervosa** – The estimated lifetime prevalence of anorexia nervosa in the general population of females is approximately 1 percent and in males 0.1 percent. The diagnosis requires each of the criteria listed in the table (table 3). Complications include bradycardia, hypotension, electrolyte abnormalities, functional hypothalamic amenorrhea, osteoporosis, gastroparesis, constipation, antenatal and postpartum problems and in severe cases myocardial atrophy, mitral valve prolapse and pericardial effusion (table 4). (See 'Anorexia nervosa' above.)

- **Avoidant/restrictive food intake disorder** – The point prevalence of avoidant/restrictive food intake disorder in eating disorder programs is approximately 5 to 20 percent. The diagnostic criteria are as follows:

  - Avoiding or restricting food intake, which leads to a persistent failure to meet nutritional and/or energy needs

  - The eating disturbance is not due to lack of available food or associated with a culturally sanctioned practice

  - The disturbance does not occur solely in the course of anorexia nervosa or bulimia nervosa, and body weight and shape are not distorted

  - The disturbance is not due to a general medical condition or another mental disorder

  (See 'Avoidant/restrictive food intake disorder' above.)

- **Binge eating disorder** – The lifetime prevalence of binge eating disorder in adults is 1.9 percent, and the 12-month prevalence is 0.8 percent. Binge eating disorder is characterized by the following:

  - Episodes of eating an amount of food in a discrete period of time that is definitely larger than what most people would eat in a similar period of time under similar circumstances

  - The episodes of binge eating are associated with a lack of control and with distress over the eating

- These episodes occur on average at least once a week for three months

Binge eating disorder is accompanied by significant functional impairment in most affected individuals, and is more frequent in those with obesity, but can also be found in normal weight individuals. (See 'Binge eating disorder' above.)

- **Bulimia nervosa** – The lifetime prevalence of bulimia nervosa in the United States in females is approximately 0.5 percent and in males is 0.1 percent. The diagnostic criteria include binge eating (consuming a large amount of food in a discrete period of time) and inappropriate compensatory behavior to prevent weight gain, both occurring on average at least once per week for three months (table 5). Complications include dehydration, hypokalemia, menstrual irregularities, Mallory-Weiss syndrome, and erosion of dental enamel. (See 'Bulimia nervosa' above.)

- **Pica** – Pica involves repeated eating of nonfood substances that is inappropriate to the patient's developmental level and warrants clinical attention. (See 'Pica' above.)

- **Rumination disorder** – Rumination syndrome is a functional disorder characterized by effortless regurgitation of recently ingested food into the mouth after most meals. The material is either spat out or reswallowed. (See 'Rumination disorder' above.)

ACKNOWLEDGMENT

The UpToDate editorial staff acknowledges Sara F Forman, MD, who contributed to an earlier version of this topic review.

Topic 2093 Version 42.0

→