Section 1
Introduction to Psychological Care in Severe Obesity

Chapter 1
Psychosocial Issues in Severe Obesity
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Case Vignette

Nada is a 39-year-old married woman who works as a cashier. She is a mother of a 14-year-old daughter. Her husband, Sam, works as a truck driver. Nada has struggled with her weight over the years. Today she sees her primary care physician asking to be on a medication to help with her weight.

Nada was “chubby” as a kid. She started gaining weight around the time of her parents’ divorce. At school she was bullied for being “lazy.” She experimented with marijuana and started smoking at age 14. She was able to finish high school and worked as a cashier to support herself. She tried a number of yoyo diets and weight loss shakes with initial success in losing some weight followed by regaining the weight back and more. She gained more weight after her marriage at age 24. As she was planning to get pregnant, she quit smoking and gained more weight. Her pregnancy was complicated with gestational diabetes.

She describes her life as “hectic” and states that she is “on the go” all the time. Her husband spends days away from home due to long-haul driving. She has to juggle a job and a family. Her daughter is having some academic difficulties due to reported hyperactivity. She complains of general fatigue and periods of weepiness that she attributes to stress, preoccupation with her weight, and feeling “undesirable.” She denies symptoms of cold or heat intolerance, nervousness, or irritability. She says that she has some difficulty sleeping and reports waking up feeling “unrested.”

She also complains of occasional hip stiffness after walking for prolonged periods. The hip pain limits her ability to exercise. She is on metformin for type 2 diabetes. She is frustrated with her diabetes because her sugar levels are not under control. She has seen a dietitian but has been having difficulty following the nutrition education because “I am busy all the time.”

When Nada’s eating behavior was explored, she endorsed loss of control over eating, especially at night after she comes home from work and her daughter goes to bed. She feels bored, and she finds eating to be a “way out of stress.” She eats large amounts while watching TV. She initially feels good but later feels “disgusted” with herself. She misses breakfast and eats “junk food” at the convenience store where she works.

On examination, her weight is 250 pounds and her height is 5 feet, 6 inches. Her body mass index (BMI) is 40 kg/m². Her affect is tearful, and she reports her mood as “frustrated” because of her weight struggles. She endorses sleep difficulties and low energy. She has missed work at times because of the fatigue and the hip pain. She also reports periods of irritability toward her daughter. Her thoughts are organized, and she feels demoralized and helpless to control her weight. She is not suicidal.
Introduction
This chapter provides an overview of the obesity epidemic and identifies common psychological issues facing patients with severe obesity. Prevalence rates, clinical presentations, and risk factors for a range of psychiatric disorders seen in severe obesity are discussed, including mood disorders, anxiety disorders, and eating disorders. Within the chapter, developmental factors predisposing individuals to obesity will be summarized. The chapter concludes with a summary of the role of psychosocial interventions in obesity care, specifically situating these interventions within the range of medical and surgical interventions in obesity care. The importance of psychosocial/behavioral health professionals in the management of psychological factors arising in obesity care are highlighted.

Obesity-Related Medical Comorbidities and Medical Treatments for Severe Obesity

Obesity, classified as a BMI of 30 kg/m² or greater, has reached epidemic proportions, with obesity rates nearly doubling since 1980 [1]. Data from the World Health Organization (WHO) suggest that in 2014 more than 600 million individuals had obesity worldwide [1], which compares with 78 million individuals with obesity in the United States [2]. Obesity is associated with a multitude of medical comorbidities, including hypertension, dyslipidemia, type 2 diabetes mellitus, coronary heart disease, sleep apnea, osteoarthritis, and some cancers. Moreover, obesity is associated with a significant increase in all-cause mortality even after considering metabolic status [3,4]. As a result, obesity is a well-recognized multiorgan disease with clear associations with a range of comorbidities including psychiatric illness.

Given the burden of severe obesity, several treatment options have been studied to determine their effectiveness in this patient population. Several medications have received indications for treating obesity and may offer benefit in the range of 3 to 9 percent weight loss (compared with placebo) beyond lifestyle counseling alone [5] (see Table 1.1).

At present, the most durable treatment for severe obesity is bariatric, or weight loss, surgery. A meta-analysis by Gloy and colleagues reported that bariatric surgery resulted in greater weight loss, remission of type 2 diabetes, and remission of metabolic syndrome compared with nonsurgical treatments [6]. Bariatric surgery includes a range of procedures that have evolved over time with advancements in surgical techniques and equipment. Bariatric surgery works through one of two mechanisms, namely, malabsorption and restriction. Weight loss from malabsorption is achieved through bypassing the first part of the small intestine, where calories are absorbed. Restriction from bariatric surgery procedures limits food intake and calories through a smaller gastric pouch. While the exact mechanisms of bariatric surgery procedures are complex and still to be determined, current nomenclature classifies the laparoscopic gastric banding (LAGB) and laparoscopic sleeve gastrectomy (LSG) as restrictive procedures and the laparoscopic Roux-en-Y gastric bypass (LRYGB) and duodenal switch as both restrictive and malabsorptive procedures. The two most common surgical procedures offered in North America are the LSG and the LRYGB. In the STAMPEDE study, bariatric surgery with intense medical management was more effective than intense medical management alone in sustained weight loss and remission of type 2 diabetes [7]. Table 1.2 lists the types of surgical treatments for severe obesity and their descriptions.
### Table 1.1 Long-Term Pharmacologic Treatments for Obesity

<table>
<thead>
<tr>
<th>Obesity pharmacotherapy</th>
<th>Mechanism of action</th>
</tr>
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<tbody>
<tr>
<td>Orlistat (Xenical)</td>
<td>Lipase inhibitor causing excretion of triglycerides in stool</td>
</tr>
<tr>
<td>Lorcaserin (Belviq)</td>
<td>Highly selective 5-HT2 C-serotonergic receptor agonist causing appetite suppression</td>
</tr>
<tr>
<td>Phentermine/topiramate-ER (Qysmia)</td>
<td>Noradrenergic and GABA-receptor activator, kainate/AMPA glutamate receptor inhibitor (note: unclear mechanism of action for weight loss)</td>
</tr>
<tr>
<td>Naltrexone-SR/bupropion-SR (Contrave)</td>
<td>Pro-opiomelanocortin (POMC) neurons stimulated by bupropion and naltrexone blocking autoinhibitory effects related to weight loss</td>
</tr>
<tr>
<td>Liraglutide (Saxenda)</td>
<td>Glucagon-like peptide receptor agonist increasing satiety and reduces food intake</td>
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</table>

* Listed agents have been approved by the US Food and Drug Administration (FDA) as treatments for obesity.

### Table 1.2 Summary of Common Bariatric Surgery Procedures

<table>
<thead>
<tr>
<th>Bariatric surgery type</th>
<th>Restrictive/ malabsorptive</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laparoscopic Roux-en-Y gastric bypass</td>
<td>R + M</td>
<td>Creation of a 20- to 30-ml gastric pouch from proximal lesser curve of stomach. A Roux-en-Y anastomosis is created from the biliopancreatic limb, alimentary limb, and remainder of the small intestine (common channel).</td>
</tr>
<tr>
<td>Laparoscopic sleeve gastrectomy</td>
<td>R</td>
<td>The majority of the greater curve of the stomach is removed to create a smaller gastric reservoir.</td>
</tr>
<tr>
<td>Laparoscopic gastric banding</td>
<td>R</td>
<td>An adjustable silicone band is placed around the cardia of stomach below the gastroesophageal junction.</td>
</tr>
<tr>
<td>Biliopancreatic diversion ± duodenal switch</td>
<td>R + M</td>
<td>This is a less common procedure, a combination of a distal gastrectomy and long Roux-en-Y reconstruction. A duodenal switch is a variant of this procedure in which the pylorus is preserved.</td>
</tr>
</tbody>
</table>

*R = restrictive; M = malabsorptive.*
Obesity-Related Psychiatric Comorbidities

Relationship between Obesity and Psychiatric Illness

A bidirectional relationship exists between obesity and psychiatric illness. Obesity results in higher rates of psychiatric disorders, and certain psychiatric disorders increase the risk of obesity independent of iatrogenic factors, such as psychotropic medications. Prevalence rates of current and lifetime psychiatric disorders vary across studies depending on the assessment setting and the method of assessment, specifically self-report versus clinician-administered diagnostic tools. Lifetime rates of psychiatric disorders in patients with severe obesity approximate 70 percent in studies where the assessment was conducted by clinicians who were independent of obesity treatment programs [8]. The most common psychiatric disorders in samples of bariatric surgery candidates are mood disorders (23 percent), binge eating disorder (17 percent), and anxiety disorders (12 percent) [9].

The high rates of psychiatric comorbidity are further supported by studies showing a positive correlation between BMI and level of psychopathology, with increases in BMI resulting in higher psychopathology in individuals with obesity [10]. This relationship is further summarized in obesity staging, which has included psychological comorbidities as part of the severity classification system for obesity. For example, the Edmonton Obesity Staging System includes “obesity-related psychological symptoms” as part of its staging system to quantify obesity severity in addition to BMI [11]. In stage 2 severity in this system, moderate psychological symptoms include depression and anxiety disorders. Thus psychiatric factors are a core component of obesity care and should be assessed in order to generate comprehensive treatment plans to support patients seeking treatment for obesity.

Pathoetiologic of Psychiatric Comorbidity in Severe Obesity

A myriad of pathoetiologic factors have been proposed to explain the bidirectional relationship between psychiatric disorders and obesity. Factors can be divided into biological factors, iatrogenic factors, and environmental factors. Biological factors consist of shared abnormalities in neurometabolism and neuroinflammation between obesity and psychiatric disorders, specifically using mood disorders as the paradigm. Iatrogenic factors primarily consist of medications that are often used in psychiatric treatment that can increase the risk of metabolic syndrome, specifically atypical antipsychotic medications and some antidepressants. Lastly, environmental factors consist of early-childhood adversity, which is known to increase the risk of psychiatric illness and also obesity in the long term. Each of these factors will be further discussed in this section.

Biological Factors. As mentioned earlier, aberrant neurometabolism and neuroinflammation responses are shared between psychiatric illness and obesity. These shared features are best described in mood disorder populations [12]. One potential mechanism is the hypothalamic-pituitary-adrenal (HPA) axis, which is dysfunctional in both obesity and mood disorders. Studies suggest that abdominal adiposity has been associated with abnormal HPA axis activation, including both higher and lower cortisol levels in response to stress [13–16]. Conversely, individuals exposed to chronic stress, which resulted in elevated cortisol levels, were found to have increased visceral adipose tissue [17]. In addition to obesity, both depression and bipolar disorder have been associated with abnormalities in
the HPA axis. Individuals with bipolar disorder have increased cortisol secretion in euthymic, manic, and depressive states and have blunted reactivity to stress [18]. Several studies have reliably demonstrated an association between remitted and symptomatic depression with an increase in cortisol levels [19,20]. It is possible that these overlapping findings related to HPA axis abnormalities explain the relationship between mood disorders and obesity.

Obesity is also associated with immune-inflammatory dysregulation. Data from a meta-analysis have shown elevated levels of pro-inflammatory markers, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), in the serum of patients with bipolar or major depressive disorder [21,22]. Moreover, obesity is also associated with pro-inflammatory states, such as increased levels of C-reactive protein [23]. Therefore, proinflammation may be a shared pathway leading to comorbidity between obesity and psychiatric illness that warrants further study.

Adipokines in the context of obesity have been associated with mood disorders and psychopathology. Previous studies have suggested that leptin resistance in the context of abdominal obesity was a risk factor for depression in an elderly patient sample [24]. Further, a longitudinal study of patients with early-stage bipolar disorder showed an inverse relationship between adiponectin and leptin levels and depressive symptoms [25]. In this study, higher adiponectin and leptin levels were also associated with higher BMI gain. Despite these findings, the relationship between adipokines and mood symptoms has been limited by confounding variables. The heterogeneity in the association between depression and adiponectin across studies may be explained by gender differences and differences in depression severity [26]. Further, the heterogeneity in the findings between leptin and depression may be explained by differences in BMI between groups with depression and healthy control individuals in these studies [26]. Thus the role of adipokines in this relationship between psychopathology and obesity warrants further study to elucidate the nature of this relationship across patient populations.

In addition, several studies have identified an increased association between attention deficit hyperactivity disorder (ADHD) and obesity. Identification of familial risk factors from the Swedish National Registry showed an increased risk of ADHD in siblings of index males with obesity [27]. ADHD has also been observed at higher rates among patients with eating disorders, including binge eating disorder (BED) [28].

Lastly, the construct of food addiction has emerged as a possible risk factor for obesity and has been linked to hedonic drive and eating psychopathology in obesity. Strong evidence initially emerged from animal models demonstrating that rats given intermittent access to sugar ignore their usual chow over time and begin to binge on sugar solution after repeated exposures to sugar [29]. Subsequently, rats displayed signs of withdrawal when sugar was removed from their diet or naloxone was administered to block the opioid receptor effects of sugar. Further, neuroimaging studies in humans have shown deficits in dopaminergic pathways and frontal-striatal systems that are also seen in individuals who use cocaine [30]. Additional risks for overeating in obesity include genetic attenuation of dopamine transmission as a result of reduced dopamine D2 receptor (DDR) availability, which is observed in patients addicted to substances of abuse. The Taq1A polymorphism for the A1 allele has been proposed as a genetic variant resulting in decreased DDR [31]. However, a meta-analysis has refuted the association between the Taq1A polymorphism and obesity, thus suggesting that parallels for these genetic associations cannot be made for food addiction [32]. In summary, these vulnerabilities and activation of reward pathways by...
highly processed foods may predispose some individuals to develop eating patterns reflective of food addiction symptomatology; however, further exploration is required to identify clear mechanisms and risk factors.

Iatrogenic Factors. Treatment of psychiatric comorbidity in patients with severe obesity may warrant the use of psychotropic medications. These medications may predispose individuals to develop metabolic side effects including weight gain and diabetes. Antipsychotic medications, such as olanzapine, clozapine, and quetiapine, carry increased metabolic risks in the long term [33,34]. Similarly, antidepressant medications, specifically mirtazapine, paroxetine, and tricyclic antidepressants, can also result in increased weight in patients with depression and anxiety [34]. Further, patients with autoimmune disorders and chronic obstructive pulmonary disease (COPD) may require treatment with steroid medications, such as prednisone, which can increase the risk of weight gain and metabolic syndrome. Therefore, pharmacotherapy for obesity-related physical and mental health comorbidities is a potential factor contributing to the co-occurrence of obesity and psychiatric illness.

Environmental Factors. Early-childhood adversity has been associated with the development of comorbid obesity and psychological distress. In the seminal Adverse Childhood Experiences study involving 13,177 patients from a large health organization in California, a history of childhood abuse was more strongly associated with a BMI of 40 kg/m² or greater than a BMI of 30 kg/m² or greater as an adult [35]. Moreover, individuals with a history of sexual abuse have higher rates of severe obesity compared with control individuals [36]. Data from a large meta-analysis consisting of 23 cohort studies involving 112,708 participants demonstrated that all types of childhood abuse were associated with adult obesity: physical (odds ratio [OR] = 1.28), emotional (OR = 1.36), sexual (OR = 1.31), and general abuse (OR = 1.45) [37]. Weight discrimination and bullying in early childhood also have been associated with a range of psychological sequelae in children and youth with obesity, including maladaptive eating behaviors, anxiety, depression, and suicidal ideation [38]. In addition, patients’ early-childhood parental figures and attachment relationships may shape their perception of the world, including their relationship with healthcare providers caring for them during their obesity care (see Chapter 4). An insecure attachment (relationship) style can also contribute to disordered eating, difficulties engaging with obesity care teams, and attenuated weight loss outcomes [39]. This evidence provides compelling support that childhood adversity and attachment relationships further contribute to the development of comorbid psychiatric illness and obesity.

Specific Psychiatric Disorders in Severe Obesity

Mood Disorders

Substantial research has identified a bidirectional relationship between obesity and depression, which has been established early in life. A meta-analysis of studies examining depression rates in children and adolescents who are obese and not obese found a positive association between childhood and adolescent obesity and depression (OR = 1.34), as well as higher depressive symptoms in the patient groups with obesity [40]. A meta-analysis involving longitudinal studies showed that individuals with depression had a 58 percent
increased risk of developing obesity, and individuals with obesity had a 55 percent increased risk of developing depression [41].

Among bariatric surgery candidates, rates of depression based on clinician-administered assessment approximate 19 percent, with some large multisite studies reporting rates as high as 39 percent [9,42]. Following bariatric surgery, depression tends to improve in the first year postoperatively; however, long-term data suggest that depressive symptoms can gradually increase over time [9,43]. It should be noted that depressive symptoms remain better than before surgery for most patients, although there are several reports of increased self-harm and suicide in comparison with the general population after bariatric surgery, which may be related to worsening depression [44,45]. Some studies suggest that early postoperative depression is associated with worse long-term weight loss outcomes after bariatric surgery; however, further research is needed to replicate these early findings [9,46]. Based on the current evidence, preoperative assessment and stability of depressive symptoms should be a focus of psychosocial assessment and early intervention. This should be followed by continuous monitoring and early introduction of psychological interventions after bariatric surgery to support patients’ behavior changes and psychological well-being.

Several factors have been reported to increase the association between depression and obesity. Lower socioeconomic status has been associated with an increased risk of comorbid depressive disorder and obesity [47]. Additional clinical factors increasing the risk of comorbid depression and obesity include more severe obesity, binge eating and other dysregulated eating behaviors, increased physical health issues [47], body image issues such as prominent shape and weight concerns and body image dissatisfaction [47], the presence of weight-related stigma [48,49], and interpersonal stressors and decreased social activity [50,51]. Although psychological factors such as self-esteem, hostility, and maladaptive schemas also influenced the relationship between obesity and depression, gender differences emerged regarding the impact of specific psychological factors [47]. The relationship between obesity and depression was stronger for men with low interpersonal effectiveness, poor conflict resolution, and loneliness [52]. In comparison, the relationship between obesity and depression was stronger for women with greater anger, sadness, and excitement. Based on these variables, patients identified as being at high risk should be educated on potential risks. Moreover, patients who are at an increased risk of having comorbid depression and obesity may benefit from psychological interventions aimed at improving modifiable variables, such interpersonal effectiveness and self-esteem, for example.

In addition to depression, obesity has also been associated with bipolar disorder and known to influence mood outcomes in this population. A study of 644 patients with bipolar 1 and 2 disorder from the United States and Europe identified obesity and severe obesity in 21 and 5 percent of the sample, respectively [53]. Data from the National Comorbidity Survey Replication involving 9,125 respondents noted that obesity was significantly associated with a lifetime diagnosis of bipolar disorder (OR = 1.47) and had an even stronger association with a diagnosis of bipolar disorder within the last 12 months (OR = 1.61) [54]. These studies reinforce the strong link between bipolar disorder and severe obesity.

Obesity has also been associated with a worse course of illness and increased risk of mood relapse in patients with bipolar disorder. In a study comparing treatment naïve female patients with bipolar disorder with control individuals without bipolar disorder, patients with bipolar disorder had higher abdominal adiposity compared with control individuals [55]. Moreover, patients with bipolar disorder who are obese have higher rates of mood...
recurrence compared with nonobese patients with bipolar disorder [56]. In addition, higher BMI has been associated with longer duration of illness and chronic course in bipolar disorder [57]. The impact of obesity on bipolar illness outcomes is further reinforced by neuroimaging data from patients recovered from their first manic episode, which showed a relationship between elevated BMI and gray and white matter reductions in the frontal, temporal, and subcortical limbic areas, which are all implicated in the pathophysiology of bipolar disorder. In addition to illness-related effects on obesity, it should be noted that many of the mood-stabilizing medications, such as atypical antipsychotics, lithium, and valproate, are associated with increased weight gain in the long term [58]. Therefore, treatment of obesity should be a priority in patients with bipolar disorder given the impact on mood disorder outcomes.

Bariatric surgery outcomes have been explored for patients with bipolar disorder given the high prevalence of obesity, potential benefit on mood symptoms, and limited treatments for severe obesity in this patient population. Research has been limited by small sample sizes and short-term follow-up. A matched cohort study comparing 13 patients with bipolar disorder who underwent bariatric surgery with patients with bipolar disorder who had not undergone bariatric surgery over a 2.17-year mean follow-up period showed no effect of bariatric surgery on psychiatric hospitalization rates or outpatient psychiatric utilization [59]. A retrospective study comparing patients with bipolar disorder, other psychiatric conditions, and no psychiatric illness showed no difference in weight loss outcomes one year after surgery [60]. Moreover, a prospective cohort study showed no difference in weight loss outcomes and physical quality of life one year after surgery between patients with complex psychiatric illness (including bipolar disorder), other psychiatric illness, and no psychiatric illness; however, mental quality of life was reduced in the group with more complex psychiatric illness [61]. Despite these findings suggesting that bariatric surgery does not result in worse psychiatric stability or reduced weight loss, patients with bipolar spectrum disorders are still less likely to be deemed “ready” or eligible for bariatric surgery compared with other patient groups [62]. Therefore, greater focus on psychosocial factors and treatments is needed to better support patients through this process and to anticipate potential challenges with bipolar disorder treatments and follow-up bariatric surgery care.

Anxiety Disorders

In addition to depression, anxiety is a common comorbidity in patients with obesity. Estimated prevalence rates of anxiety disorders are approximately 12 percent, although lifetime rates are as high as 31 percent [9,42]. The most common anxiety disorders in this population are specific phobias and social anxiety disorder, with lifetime rates of the latter approximating 6 percent. The Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5) modified the criteria for social anxiety disorder to allow for the diagnosis of social anxiety even if the fears were related to a medical condition such as obesity [63].

Assessment of anxiety in the context of severe obesity is an important component of care, especially given data suggesting that anxiety can drive maladaptive eating behaviors. For example, grazing eating patterns in bariatric surgery candidates were associated with anxiety disorders in addition to BED [64]. Anxiety symptoms also follow a similar course to depressive symptoms after bariatric surgery, specifically the “honeymoon” phase with reduced anxiety within the first 6 to 12 months after massive weight loss, followed by increased anxiety beyond the initial year. Ten-year data from the Swedish Obesity Study
showed an initial reduction in anxiety symptoms at one year after surgery (37 percent), but this improvement was attenuated at six years (20 percent) and at ten years after surgery (23 percent) [65]. In addition, greater improvements in anxiety, depressive symptoms, and mental quality of life after bariatric surgery predicted improvement in employment functioning [66]. Data on the long-term effects of anxiety relapse have not predicted postoperative weight loss, although there are some data that anxious relationship (attachment) style is associated with poor adherence to postoperative prescribed vitamin supplementation after LRYGB [67].

Eating Disorders

Given the high prevalence of eating disorders in patients with severe obesity, a growing body of literature has summarized the rates of eating psychopathology in patients with severe obesity and during the course of severe obesity care. Rates of BED in patients with severe obesity are estimated to be 13 to 17 percent; however, subthreshold symptoms are more prevalent [9,42]. Prevalence rates for BED have ranged from 2 to 49 percent across studies including a broad range of assessment measures that extend beyond structured psychiatric assessment and interviews [68]. When using the DSM-IV-TR diagnostic category of “eating disorder not otherwise specified” (EDNOS), approximately 27 percent of bariatric surgery candidates meet criteria for EDNOS, which captures these subthreshold eating disorder symptoms [42]. The prevalence of night eating syndrome (NES), characterized by a shift in the circadian pattern of eating that results in nocturnal hyperphagia and increased frequency of nocturnal ingestions, ranges from 2 to 20 percent across studies due to varying measures and definitions of night eating [69]. Rates of emotional eating and grazing eating patterns have ranged from 38 to 59 percent and 20 to 60 percent, respectively, among bariatric surgery candidates [70].

Both BED and NES have been associated with obesity. Studies have demonstrated higher rates of obesity in patients with BED. In the WHO World Mental Health Survey study, the percentage of patients with a lifetime diagnosis of BED who had a BMI of 35 kg/m² or greater was 62 percent, compared with 25 percent in patients without a diagnosis of an eating disorder [71]. Additional studies have also supported a positive association between NES and obesity [72]. Moreover, a study of correlates of NES in bariatric surgery candidates showed that NES was positively correlated with BMI and BED, which highlights that these two distinct diagnostic entities may co-occur in patient populations with severe obesity [73]. Studies suggest that the rates of comorbid BED and NES range from 7 to 25 percent [74,75]. Nonetheless, based on data comparing normal-weight individuals and those with obesity, the risk of obesity related to NES and BED appears to increase with age [76].

Although both BED and NES have been associated with severe obesity, the direction of this relationship remains unclear. Regardless, it is clear that features of BED and NES contribute to weight gain and obesity in some patients. Loss of control over eating is a core feature of BED and can persist after bariatric surgery despite restrictive procedures. Persistence of loss of control over eating after surgery can lead to problematic eating, attenuated weight loss, weight regain, and poorer quality of life [77]. Bariatric candidates with loss of control over eating have significantly higher levels of night eating, depressive symptoms, and eating disorder psychopathology with lower mental quality of life [78].

Research examining the impact of bariatric surgery on eating psychopathology has yielded mixed results. According to an observational study analyzing data from 36 patients
with preoperative BED, bariatric surgery resulted in a reduction in BED symptoms in the first year after surgery [79]. This study contrasts with other studies that have shown an initial reduction in symptoms within the first 6 to 12 months after surgery with a gradual increase in BED symptoms over time. For example, one study showed an initial reduction in binge eating episodes at six months after RYGB (61.2 percent preoperatively to 30.7 percent), followed by an increase from 6 to 12 months after surgery (36.4 percent) and a further increase at 24 months after surgery (39.4 percent) [77]. In a recent review examining changes in eating psychopathology from before to after surgery, the authors concluded that several “fair-rated” studies showed significant reemergence of BED symptomatology after an initial reduction during the first 6 to 12 months after surgery [80]. Although several systematic reviews suggest that BED and NES before surgery are not robust predictors of weight loss after surgery [8,9], some studies suggest that preoperative eating psychopathology, such as loss of control over eating, may increase the risk of similar eating psychopathology after bariatric surgery [77]. Further research is needed to examine changes in BED and NES symptoms in the long term and across additional weight loss surgery populations, specifically LSG patients.

Despite these inconclusive findings, it is clear that a proportion of patients undergoing bariatric surgery will continue to have a spectrum of disordered eating. In fact, trends in severe obesity parallel evidence from previous reviews in uncomplicated obesity demonstrating improvement in eating psychopathology with weight loss. A systematic review of 134 studies in uncomplicated obesity reported an improvement in eating psychopathology, specifically cognitive restraint, control over eating, and binge eating [81]. With respect to severe obesity, approximately 25 percent of bariatric surgery patients endorsed subjective binges up to two years after surgery; however, only 3.4 percent fulfilled criteria for BED [82].

In a Longitudinal Assessment of Bariatric Surgery (LABS) Research Consortium three-year follow-up study, eating psychopathology, specifically global disordered eating, loss of control over eating, and regular evening hyperphagia, decreased in the initial year after surgery, but cravings and subjective eating binges were unchanged [83]. These improvements in global disordered eating, loss of control over eating, and evening hyperphagia continued at three years after surgery. Ten-year follow-up studies examining psychological factors related to eating after bariatric surgery have shown persistent improvements in disinhibited eating and hunger and to a lesser degree in cognitive restraint of eating [84]. Overall, the evidence to date suggests that most patients who undergo bariatric surgery experience improvements in eating psychopathology from before to after surgery.

Patients can develop serious eating psychopathology after surgery, however, and some of these conditions may be related to surgical complications or inadequate weight loss outcomes. Although vomiting may be a sign of disordered eating, it is not uncommon for patients to experience vomiting early postoperatively due to gastric discomfort or to complications such as strictures. In a sample of postoperative bariatric surgery patients, 63 percent endorsed vomiting due to gastric discomfort whereas 12 percent attributed vomiting to shape- and weight-related concerns [82]. Rare but distressing reports of anorexia nervosa emerging after bariatric surgery have been reported, and these cases generally occur in the context of poor weight loss outcomes after surgery [85]. Moreover, patients may resume emotional eating after surgery, which can trigger nonadherence to postoperative nutrition regimens and potentially increase the risk of weight regain [86]. Postoperative psychosocial interventions have been shown to assist in reducing emotional eating in the postoperative period and will be discussed in more detail in Chapters 6, 9, 10, and 11.