Chapter 10
Psychological and Psychiatric Workup

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10.1 Introduction

Bariatric surgery is the most effective treatment for morbid obesity [1], a condition that is increasingly common worldwide and that affects children particularly. Shortly, the costs of preventing and treating obesity, as well as its metabolic complications, shall not be fully covered by health systems [2]. Biliopancreatic diversion with duodenal switch (BPDDS), along with gastric bypass (RYGBP), is among the surgical techniques leading to weight loss and its maintenance over time [3]. BPDDS includes three specific components: (1) a longitudinal gastrectomy, providing caloric restriction and decreasing acid production, while maintaining normal gastric emptying; (2) a 250 cm total alimentary limb whose role is to reduce caloric absorption; and (3) a 100 cm common channel where the bolus mixes with biliopancreatic juices, resulting in decreased absorption of protein and fat [4]. Although long-term data on health-related quality of life (HRQL) after BPDDS is scarce, Aasprang et al. [5] accessed HRQL through a self-administered questionnaire, before and 1, 2, 5, and 10 years after BPDDS, showing long-term improvement in physical and mental scores. Søvik et al. [6] showed a reduction in uncontrolled and emotional eating behaviors, as well as an improvement in psychosocial function both after duodenal switch and RYGBP. Despite these results, 20% of patients undergoing bariatric surgery fail to maintain weight loss 2 years after surgery [1]. A significant number of these patients suffer from dysfunctional eating behaviors

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A. Teixeira et al. (eds.), *Duodenal Switch and Its Derivatives in Bariatric and Metabolic Surgery*, https://doi.org/10.1007/978-3-031-25828-2_10

(DEB) leading to a recurrence of obesity after bariatric surgery [7], which makes the prompt identification and treatment of DEB in bariatric patients imperative. This chapter aims to discuss the many psychological and psychiatric variables that may jeopardize BPDDS short-, medium-, and long-term outcomes. It is important to highlight that the literature on psychological and psychiatric aspects specifically related to BPDDS is still scarce; hence, many of the points discussed here stem from studies not necessarily performed with patients undergoing BPDDS.

10.2 Important Psychological Aspects on the Evaluation and Follow-Up of Candidates for Bariatric Surgery

Despite the countless benefits currently documented of bariatric surgery, not only related to significant weight loss and its long-term maintenance, but also regarding measures of quality of life and psychopathology, less is known about the origin of post-surgical undesirable psychological and behavioral outcomes affecting patients' eating behavior [8]. Data on patients undergoing BPDDS are even more scarce. Undesirable psychological and behavioral outcomes possibly reflect psychological and behavioral problems present before surgery, which, if not properly identified and treated, will certainly interfere negatively with BPDDS results. Nevertheless, it is important to remember that obesity is a complex pathological condition in which behavior is only one of the many dimensions to be addressed for obesity's suitable comprehension. Biological factors such as chronic low-grade systemic inflammation triggered by obesity [9], alterations of the intestinal microbiome [10], and brain insulin resistance [11] have been widely described as protagonists in the pathophysiology of obesity, which are known to impact the brain and the minds of patients with obesity. Such impacts are reflected, for instance, in the ability to control food consumption and adhere to physical activity programs [8]. It is possible that these factors have an even broader participation in how they modify the mind of people with obesity, altering their cognition [12], emotions [13], motivation [14], self-regulatory processes [15], and even their ability to identify emotions and other mental states in others [7].

Neuroimaging studies show that bariatric surgery can reverse anomalous recruitment and connectivity patterns in different brain areas related to both the processing of pleasure and reward associated with eating, and brain areas associated with cognitive control [16, 17]. This strongly suggests that bariatric surgery may help normalize several neuropsych pathological processes favoring DEB such as binges, emotional eating (EE), and food addiction (FA). Potential mechanisms for regulating brain activity by bariatric surgery include, in addition to improving inflammation, changes in the expression of dopaminergic receptors in key areas such as the ventral striatum, putamen, caudate, thalamus, and hypothalamus, as well as post-surgical changes in the concentration of peptides such as ghrelin, GLP-1, and peptide YY [18]. Findings like these are, however, still controversial. Therefore, the

complexity of the mechanisms through which obesity and bariatric surgery can affect the mind and behavior requires that the mental health professional working with bariatric patients have a broad knowledge about the countless variables at stake, which, when not properly controlled, may threaten the surgical results.

The processing of emotions seems to be affected by obesity, favoring BED, which interferes in the results of BPDDS and all other surgical techniques. Indeed, patients with obesity frequently say that their emotions drive or determine their eating behaviors. These patients usually state that they consume food—usually caloric ones—in order to relieve emotions—typically the unpleasant ones.

Emotions are defined as short-term affective responses triggered by environmental stimuli, situations, or events with reinforcing potential [19]. They have different motivational functions and contribute to the control of basic behavioral systems in animals and humans. Emotions may affect all eating behavior, including motivation to eat, affective responses to food, food choices, chewing, speed of eating, amount of food ingested, and even metabolism and digestion [19]. Thus, emotions and eating behavior are closely linked; however, the nature of this connection is not yet fully understood. Hunger is indeed a potent emotional modulator. In fact, hungry animals and humans tend to be more alert and irritable and diverse stimuli elicit different emotional responses in individuals with and without hunger [20]. Nevertheless, there is an individual variation in how emotions may affect eating behavior. Several experiments have shown that individuals restricting food in order to decrease or maintain weight eat more in response to fear and negative moods than individuals who do not [19]. These studies also show that emotional eaters tend to consume more sweet and fatty foods in response to emotional stress, and compulsive eaters tend to have binges when facing negative emotions [19]. Negative emotions need to be regulated and it is possible that, in at least a percentage of individuals with obesity, they will be anomalously regulated with caloric foods. Emotion regulation (ER) is a multidimensional construct encompassing the ability to respond to personal and social demands with acceptable and flexible behaviors and emotions, as well as the ability to postpone and even suppress spontaneous reactions when this is necessary or convenient. ER is achieved through psychological processes such as monitoring, appreciating, and changing the magnitude of emotional reactions [21].

Many patients compare their relationship with food with that displayed by addicts to psychoactive substances, a similarity with an irresistible intuitive appeal, since individuals who consider themselves addicted on food present behavioral phenomena such as cravings, feelings of loss of control, excessive consumption, tolerance, and even signs of food withdrawal. Indeed, obesity and addictions share neurobiological processes that result in compulsive consumption, which are consequences of problems on the suitable functioning of reward processing circuits, where dopamine plays an essential role.

The particularly reinforcing character of food in obesity characterizes its *addictive dimension* [22]. The neurobiological factors traditionally studied in both conditions include three interconnected brain systems that control eating behavior: the hypothalamus (which responds to internal signals about the energy balance); limbic

structures such as the amygdala, the hippocampus, the insula, the orbitofrontal cortex (OFC), and the nucleus accumbens or ventral striatum (which are involved in learning/memory and coding the incentive or salience value of food and other environmental stimuli); and the prefrontal cortex (related to cognitive control and self-regulation) ([23]). Impairments in the ability to exert self-control are critical psychopathological elements in any addictive behavior. Self-control can be defined as the set of efforts that an individual makes to modify thoughts, feelings, and behaviors, in order to reach long-term goals or interests. Such efforts allow the coordination of lower-level automatic or implicit cognitive processes, ensuring that our behavior is in line with our aspirations [24]. Individual differences in the functioning of these three systems explain why some people are more inclined to weight gain and substance use. Such differences may stem from inheritable traits; for instance, people who prefer more concentrated sugar solutions are more likely to have a family history of alcoholism [25]. In this sense, a fundamental neuropsychological variable is impulsivity, which can be defined as a predisposition for rapid and unplanned reactions to internal or external stimuli, without concern for their negative consequences, and which results from impaired unconscious information processing [26]. Impulsivity occurs in conditions where conscious processes of reflection and self-control are impaired, being common in mania, addictions, attention deficit hyperactivity disorder, personality disorders, binge eating disorder, and obesity, among many others [23, 26]. Neurobiological processes leading to obesity and addictions result from the interaction between a tendency to produce responses of greater magnitude to potential rewards of the environment (what is called *reward sensitivity*) and damage to self-control, which explains why more impulsive individuals are also more vulnerable to weight gain when exposed to obesogenic environments [23].

Several authors have studied how psychological variables such as emotional overload, ER, impulsivity, anxiety, depression, temperament, and reward sensitivity may negatively affect bariatric surgery outcomes. For instance, Benzerouk et al. [27] studied candidates for bariatric surgery over 1 year, showing that emotional deficits lie behind binges presented by many individuals of this population, who could benefit from programs composed of ER strategies in order to avoid postoperative less than desired loss of weight. In the same sense, Lavender et al. [28] showed significant correlations between emotional dysregulation, emotion intensity, negative urgency, cognitive control, reward sensitivity, and eating pathology over 7 years in adults submitted to RYGBP and laparoscopic adjustable gastric band. Efferdinger et al. [29], in turn, evaluated ER strategies in patients before and 6 months after RYGBP or Sleeve gastrectomy and, despite not showing a significant relationship between ER strategies before and after surgery, they recorded greater patient satisfaction with their ER strategies 6 months after surgery, as well as that their greater satisfaction was associated with postoperative improvement in psychosocial functioning. Williamson et al. [30] examined the moderating effect of ER on symptoms of attention deficit and hyperactivity disorder (ADHD), impulsivity-hyperactivity, inattention, and reward sensitivity, in a cohort of bariatric patients (90% undergoing Sleeve gastrectomy), showing that worse ER strategies are

associated with more modest weight loss. These findings reinforce those of other authors, who have documented impairments in ER strategies perpetuating DEB in patients with other psychiatric conditions, whether they are bariatric [31, 32] or not [33].

Individuals with obesity may display higher scores on instruments measuring alexithymia and greater difficulty in identifying emotions than lean individuals. Alexithymia is a transdiagnostic dimension expressing impairments in the abilities to identify and describe one's own emotions, associated with an externally oriented cognitive style [21]. Individuals with obesity and greater alexithymia tend to regulate unpleasant emotions with food, particularly if they show higher scores on instruments measuring externally oriented thinking [34]. Externally oriented thinking is characterized by a style of perceiving and thinking that is disconnected from one's emotions and, therefore, characterized by a low tendency to reflect on conflicts and unpleasant feelings [35]. Therefore, these patients have a more concrete thinking, as well as a low capacity for introspection and may respond poorly to insight psychotherapies.

Individuals with alexithymia may have deficits in the interpretation of bodily signals from the periphery which, when processed by the cerebral cortex, give rise to conscious feelings or emotions [36]. It is also possible (and quite likely) that the interoceptive deficits seen in alexithymia would be not restricted to those specifically linked to emotions, but also involve non-affective interoceptive deficits, such as difficulties in interpreting signs of hunger, proprioception, tiredness, and temperature. In this sense, Brewer et al. [37] investigated whether alexithymia is specifically associated with affective interoception or with a more generalized interoceptive impairment. The authors evaluated the subjects' ability to discriminate between affective and non-affective interoception, concluding that alexithymia encompasses a more generalized interoceptive impairment, where there is a high degree of perceived similarity between affective and non-affective interoception. In this sense, one could imagine that people with alexithymia would have greater difficulty in differentiating anger or tiredness from hunger, which, in turn, would facilitate the recruitment of DEB as emotional regulators.

In addition to alexithymia, other problems in emotion recognition have been described in individuals with obesity. Deficits in emotion recognition and in recognition of the others' facial affect have been documented in children and adolescents with obesity [38, 39]. Such deficits may reflect primary impairments in the identification of one's own emotions, according to the Simulation Theory of Emotion Recognition [40]. This theory proposes that we use our own mental states to infer those of others. We judge not only how other people feel, but also how they think and what they want, through the interpretation of their actions and their facial expressions, for instance. Emotions, particularly the basic ones (happiness, surprise, fear, disgusted anger, and sadness) present us with characteristic facial expressions [41]. Faces are a salient stimulus for our species, since they inform us about identity, gender, age, emotions, and even complex social attributes of our conspecifics [42]. Brain areas associated with the processing of emotions expressed by other human faces include the insula, the OFC, the amygdala, the superior temporal sulcus, and

the superior temporal gyrus [43]. OFC seems to contextualize the emotional information, as well as to modulate the activity of the amygdala, which, in turn, directs the gaze to strategic face areas for the identification of emotions [44]. Insula is known to be involved in the processing of interoceptive information and, through its activity of elaborating interoceptive maps of the individual, it may additionally assist in the elaboration of others' interoceptive maps through the observation of their postures, attitudes, and movements [45], in an interoceptive mirroring mechanism. In this way, the insula could be identified as a brain area also associated with the simulation of the mental states of others and even with mechanisms of empathic simulation, which involve cognitive mirroring.

The difficulty in identifying, naming, and reflecting on their own emotional states present in patients with obesity and alexithymia putatively lie behind problems in regulating these states through strategies such as cognitive reappraisal or expressive suppression. Such problems may lead to ER through food, alcohol, and other substances and behaviors with addictive potential, such as gambling, shopping, social media, and pornography.

The most commonly used psychological interventions designed to address ER and DEB in patients with obesity include acceptance-based therapies, dialectical behavioral therapy (developed to reinforce the ability to deal with stress in order to decrease the frequency of dysfunctional behaviors [46]), and cognitive-behavioral therapy, performed individually or in groups [47]. Although these intervention programs include self-monitoring and other basic aspects of self-regulation, psychotherapeutic or rehabilitation strategies specifically addressed to ER have not been sufficiently studied so far in bariatric populations.

An area of increasing interest for mental health professionals working in multidisciplinary team is the relationship between personality traits and bariatric surgery outcomes [48]. This area reflects the role that individual factors such as temperament play in increasing the chances of DEB. Traits such as low *conscientiousness* (the ability to organize and control [49]), poor *impulse control*, and high *neuroticism* (tendency to experience negative mental states [49]) have been associated with an increased risk for obesity, while the trait *persistence* (ability to pursue a goal despite obstacles and frustrations) has been considered a predictor of good postoperative results [48].

10.3 Important Psychiatric Aspects on the Evaluation and Follow-Up of Candidates for Bariatric Surgery

Individuals with morbid obesity are significantly more likely to have any mood disorder, anxiety, substance use, or personality disorders, as well as higher levels of stress, depression, "food cravings," DEB, low self-esteem, and worse quality of life [50].

DEB seem to be behavioral markers of bariatric surgery undesirable outcomes and, in fact, patients with binges tend to have worse mid- and long-term postsurgical results. Binges are defined as episodes of food intake more intense than is commonly tolerated by most people, associated with a sense of loss of control over the amount of food eaten, and with feelings of guilt or shame [51].

Another major concern for psychiatrists and psychologists evaluating and treating bariatric patients is the popular phenomenon of Transference of Addictions (TA). This term suggests that, being no longer able to binge eat, some bariatric patients would abuse alcohol, other substances, as well as behaviors with addictive potential, such as gambling, shopping, or pornography. However, it has not yet been established whether these cases result from increased substance use or from engaging in addictive behaviors by individuals who had already been experiencing problems like these before surgery, from relapses in individuals with substance and/or behavioral dependence, or whether they represent new cases of problematic use of substances or addictive behaviors [52]. Due to their importance in the evaluation and follow-up of bariatric patients, these topics will be discussed in detail below.

10.4 Impulsivity and Compulsivity

Patients with obesity frequently report compulsive eating behaviors, which need to be properly evaluated and treated, mostly in candidates for bariatric surgery, since the maladaptive eating behaviors included by the patients under the term "compulsive" represent risks of undesirable postsurgical outcomes. "Compulsivity" is a nonspecific term, without a diagnostic meaning, being widely used both by patients and by untrained professionals in the diagnosis of eating disorders.

Patients who identify themselves as compulsive often experience Binge Eating Disorder (BED), Night Eating Syndrome (NES), Emotional Eating (EE), Food Addiction (FA), and/or grazing. All of these conditions somewhat include impulsive and/or compulsive components, similar to that which individuals with substance and behavioral addictions show. Such components comprise increased motivation to consume palatable foods and greater pleasure related to the consumption of such foods, gradual increase in the amount of food necessary to maintain satiety, loss of control over food consumption, greater use of time in obtaining and/or consuming food, stress and dysphoria when they are on diets or unable to eat as they usually do, eating quickly or too much in the absence of hunger, overeating despite its adverse physical and psychological consequences, and feelings of guilt, demoralization, or depression associated with eating [53, 54]. Impulsivity and compulsivity are behavioral phenotypes, or endophenotypes [53]. They are hereditary and variable in the general population [55]. Impulsivity is defined as the predisposition for rapid and unplanned reactions to internal or external stimuli, without concern for its negative consequences, and which results from impaired unconscious information

processing [26]. In a more colloquial way, an impulsive person acts without thinking. Impulsive people show flaws in conscious processes of reflection and self-control, as well as a tendency to produce responses of greater magnitude to potential rewards of the environment (reward sensitivity) [23, 26]. Compulsivity, in turn, is defined by impairments in the ability to interrupt an ongoing behavior when it is necessary [53].

Individuals with obesity may "decide" to eat without reflection (due to their impulsivity) and, once they start, they have a hard time stopping eating (due to their compulsivity), even though they acknowledge that they should do it. Impulsivity and compulsivity result from failures of the top-down control exerted by the dorsolateral prefrontal cortex over structures such as the ventral striatum and the dorsal striatum, associated, respectively, with impulsivity and compulsivity. Impulsivity and compulsivity recruit different neuronal circuits: the former, a learning system through reward and motivation located in the ventral striatum; the latter, a more dorsal striatal circuit, related to habit development [53, 56]. In some substance additions and also in obesity, the consumption of a caloric and palatable substance is initially mediated by the ventral striatum and, therefore, started impulsively. The repetitive use of that substance or palatable food—primarily subject to voluntary but impulsive control—causes a migration of neuronal activity from ventral circuits to more dorsal striatal circuits, through neuroadaptation and neuroplasticity processes, what causes loss of control over the consumption [53]. Such cellular modifications may correlate with obesity-induced inflammation. In this sense, young women suffering from obesity may have significantly worse scores on measures of attention and impulsivity when compared to women without obesity, a phenomenon that may be mediated by low-grade systemic inflammation associated with obesity, since younger individuals are not usually exposed to other mechanisms related to cognitive decline in obesity, such as hypertension, metabolic dysfunction, and cardiovascular abnormalities, which are known to alter brain structure. Additionally, cognitive impairments commonly observed in young women with obesity may indicate the beginning of an early and persistent cognitive decline associated with obesity itself.

10.5 Binge Eating Disorder

BED is the most prevalent eating disorder, even though it is underdiagnosed and undertreated [57]. BED is essentially defined by the recurrence of binges, as defined above. Patients with BED do not show binges associated with inappropriate compensatory behaviors as in bulimia (for instance, the use of laxatives and/or diuretics, induction of vomits, or exaggerated physical exercises). BED is common in obesity, although not all patients with obesity suffer from this condition. People with obesity and BED usually have more psychiatric comorbidities and are more refractory to conventional treatments for psychiatric comorbidities [54]. When compared to individuals with obesity without BED, patients with obesity and BED have a greater

sense of lack of control, greater reward sensitivity, impulsivity associated with eating stimuli, as well as feelings of guilt and shame associated with intense binges [57].

BED is a relatively common disorder, with a lifetime prevalence in the general population of 1.4%, although its prevalence may increase greatly among individuals with obesity, without noticeable differences between genders [58]. Comorbidities with other psychiatric disorders are common, such as depression, anxiety, substance abuse, and even personality disorders [58, 59]. Between 64% and 79% of BED patients have some psychiatric comorbidity throughout their lives, with mood and anxiety disorders being the most prevalent [59]. Individuals with BED also have disturbing concerns about food, weight, and body image, in addition to deficits in emotion identification and ER, as well as several interpersonal problems [7]. As discussed above, negative emotions and non-adaptive ER strategies play an important role in the initiation and maintenance of BED, particularly negative feelings associated with interpersonal relationships, such as romantic disappointments and loneliness [7, 58]. High levels of depression are related to more severe binges; for instance, binges are usually more often associated with lower mood and lower energy levels. Nonetheless, emotions other than depression and sadness tend to lie behind the compulsivity of patients with BED: anger, frustration, guilt, irritability, fury, resentment, and envy, emotions very present in interpersonal contexts, which would be less tolerated by patients with BED or would be experienced by them in a different, more aversive way.

10.6 Night Eating Syndrome

NES is characterized by recurrent episodes of night eating, which can be defined either by eating after waking up at night or by excessive consumption of food after an evening meal, causing stress or impaired functioning and is not explained by other mental disorders [54]. The condition frequently affects individuals with morbid obesity and may be explained as a circadian rhythm dysfunction where there is a dissociation between sleep and eating [60]. Other symptoms include morning anorexia, a strong need to eat between dinner and bedtime and/or during the night or dawn, as well as the belief that it is not possible to sleep without eating [61].

The prevalence of NES in the general population is usually low (between 0.5 and 1.5%) and tends to increase in individuals with obesity (in this population it may reach up to 25%) [61]. Sixty percent of the candidates for bariatric surgery may display NES, whose symptoms usually overlap with those of other eating disorders. Patients with obesity, NES, and other eating disorders are also at increased risk for mood disorders, anxiety, and sleep disorders [62]. Although individuals with NES appear to have similar patterns of onset, end, and duration of sleep when compared to healthy individuals, they wake up an average of 3.6 times per night and engage in eating behaviors in order to fall asleep again [61].

NES typically begins in early adulthood and is long-lasting, with periods of remission and relapses, frequently associated with stressful life events [62]. Some

authors suggest that the motivation to eat differs in individuals with NES when compared to that of those with BED, since night eaters eat in order to being able to sleep [62]. NES must be differentiated from Sleep-Related Eating Disorder, a parasomnia in which there are episodes of involuntary food and drink intake during sleep [62].

10.7 Emotional Eating

The relationship between emotions, ER, and DEB was discussed in detail above. ER impairments are usually associated with various psychiatric conditions, such as depression, bipolar disorder, anxiety disorders, borderline personality disorder, and eating disorders [33]. Further evidence is emerging that not only eating symptoms such as binges but also restrictive behaviors common in anorexia nervosa serve as dysfunctional alternatives to regulate or suppress unpleasant emotions. Women with bulimia nervosa, BED, and anorexia nervosa report greater difficulties in perceiving their emotions, greater tendency to avoid them, as well as poorer ability to manage them, when compared to healthy women [33].

10.8 Food Addiction

FA is a term encompassing a set of behaviors related to the consumption of palatable foods that is very similar to those observed in substance disorders. From a scientific perspective, however, the mere similarity of some eating behaviors with substance use disorders would not allow their labeling as an addiction [63]. Some researchers claim that, despite the similarity that certain eating behaviors have with substance addictions, such as the presence of cravings, loss of control, excessive consumption, tolerance, abstinence, stress, functional impairment, and even the findings of alterations in mesolimbic dopaminergic systems in patients with FA [64, 65], the addictive substance putatively present in palatable foods has not yet been identified [63], which would not authorize the use of the term *food addiction*. To date, it is not possible to ensure that a specific nutrient, be it sugar or a combination of sugar and fat, acts directly on the brain, triggering reward-motivated behaviors [63]. On the other hand, evidence that obesity causes important impacts on the activity of different brain areas [66], including those related to reward processing, as well as the fact that the great majority of individuals fulfilling FA criteria (about 88%) are obese [67], bring even more controversy to the topic. It is still not possible to identify precisely whether the changes observed by neuroimaging studies in the connectivity and activity of brain areas related to reward and cognitive control in FA are in fact due to food or specifically associated with obesity.

Obesity and addictions share neurobiological processes that result in compulsivity, which, in turn, are consequences of impairments of brain reward areas

functioning. As discussed above, the particularly reinforcing character of food in obesity characterizes its additive dimension [22]. Impairments in the ability to exert self-control are essential psychopathological elements in any addiction. Self-control allows the coordination of lower-level, more automatic cognitive processes, adapting behavior to objectives [24]. The similarity between addictions and obesity is not exclusively phenomenological and psychobiological, but also involves family history, beginning in adolescence or early adulthood, chronic evolution with relapses, and even the possibility of spontaneous resolution [68].

10.9 Grazing

Grazing, picking, nibbling, and snack eating are synonyms defining the behavior of continuously eating small portions of food [69]. A review of different conceptualizations of grazing [69] concluded that the criteria most frequently endorsed by experts include its repetitive character, the consumption of small amounts of food and the lack of planning. The loss of control was not considered by all authors as a behavioral dimension of grazing, since, for some, loss of control would differentiate BED from grazing. Grazing appears to be more frequent in bariatric patients who had BED preoperatively, and some authors hypothesize that it is a subsyndromal BED.

10.10 Addiction Transfer After Bariatric Surgery

There is a belief that a phenomenon called "addiction transfer" would be propitiated by bariatric surgery, that is, operated patients, no longer able to abuse food, would be prone to start abusing alcohol, other substances, and addictive behaviors, such as gambling, shopping, Internet, or pornography.

However, it is not well established whether these events result from increased substance use or from engaging in behavior with high addictive potential by individuals who already had problems like these before surgery or whether, in fact, they are new cases of problematic use of substances or addictive behaviors [52].

Substance and behavioral addictions are defined by their cardinal components: salience, mood changes, tolerance, abstinence, conflict, and relapse [70]. These components are as important from the diagnostic point of view as quantitative variables such as the amount of alcohol or caloric foods used per day or the time spent on social networks or consuming pornography on the Internet. Salience refers to the importance a substance or addictive behavior has in a patient's life, becoming what is most important to him. Addictive substances or behaviors induce emotional arousal or relieve aversive feelings, demand increasing amounts of a substance (or longer amounts of time involved with addictive behaviors) to achieve the same effect of arousal or relief (tolerance), and may develop

withdrawal symptoms if exposure to the drug/behavior decreases (abstinence). Patients with addictions frequently experience situations of personal or interpersonal conflict related to their addiction and usually report relapses after trying to resist it [70]. Risk factors for the development of addictive behaviors include genetic factors (e.g., children of parents with alcoholism are 2 to 4 times more likely to develop alcoholism), lack of parental/family support, and the presence of psychosocial stressors. Personality traits such as nonconformity, novelty seeking, impulsiveness, low self-esteem, aggressiveness, emotional lability, inattention, antisocial behaviors, and stubbornness are common in addictions, however, there is still no consistent evidence that an "addictive personality" actually exists [70]. Therefore, given the nosological and etiological complexity of substance and behavioral addictions, the idea that bariatric surgery "creates" new dependents may seem a little simplistic. The emergence of new cases of chemical dependencies after surgery may be just an illusion of cause and effect. For instance, the percentage of patients who admit to continuing to consume alcohol or having trouble controlling alcohol use after surgery is much higher than that of those who acknowledge having problems dealing with alcohol consumption before surgery [52]. This could reflect either a worsening of alcohol use patterns occurring after surgery (and supposedly being induced by it) or (more likely) simply the fact that patients who have alcohol-related problems omit them in the psychiatric evaluation. Except in patients with chronic and severe alcoholism, in which physical signs of the disease are apparent, the identification of problematic use of alcohol and substances can be a real challenge, since the evaluation of problems related to substance use is influenced by limits imposed by the self-report. However, despite the notion shared by most mental health professionals working with bariatric surgery candidates, that problematic alcohol use is risk factor for undesirable outcomes associated to the procedure, some authors have demonstrated better rates of weight loss among some patients with a past of abuse of substances in relation to those without a previous history of these disorders [52]. It is assumed that these surprising results are explained as a result of using the same skills employed in solving problems with substances to deal with life changes after bariatric surgery. This contradicts the idea of TA, a phenomenon still widely discussed among experts. Many of them do not admit its existence and argue that, for there to be a transference of addiction, it is first necessary to accept that in obesity there is an addition to the food and second that this addition takes on a different form after surgery. Furthermore, the lack of consensus between the meaning of addiction makes the discussion even more confusing. For many, the meaning of addiction is similar to compulsivity, a vague term [71], widely used by laypeople, which include various types of behavior, from drinking to gambling or compulsive buying, while for experts it comprises a medical term regarding substance problems, which must be defined in a standardized way [71], covering the cardinal components discussed above. Biochemical evidence suggesting a "kinship" between food and addictive substances compulsivity involves, for instance, the role of an alleged dopamine deficiency in the brain of people with obesity, perpetuating DEB, which compensates for the decreased activation of dopaminergic circuits

[72]. Many neuroimaging studies show that people with obesity have brain responses to food intake or even visual or auditory food cues that are very different from those presented by thin individuals. These responses involve several regions of the brain, already discussed above.

10.11 Final Considerations

Patients undergoing BPDDS benefit from weight loss and maintenance after surgery. However, they need to be properly evaluated regarding pre- and post-operative presence of DEB, which usually put the medium- and long-term outcomes of the procedure at risk. DEB can result not only from difficulties in emotional processing, particularly in ER processes, but also from personality variables, which can be predictive of both weight gain (low awareness, poor impulse control, and high neuroticism), as well as success after surgery (high persistence).

References

1. Taube-Schiff M, Van Exan J, Tanaka R, Wnuk S, Hawa R, Sockalingam S. Attachment style and emotional eating in bariatric surgery candidates: the mediating role of difficulties in emotion regulation. Eat Behav. 2015;18:36–40. https://doi.org/10.1016/j.eatbeh.2015.03.011.
2. Gellner R, Domschke W. Epidemiologie der Adipositas [Epidemiology of obesity]. Chirurg. 2008;79(9):807–10, 812–6, 818. https://doi.org/10.1007/s00104-008-1534-6.
3. Duarte MI, Bassitt DP, Azevedo OC, Waisberg J, Yamaguchi N, Pinto Junior PE. Impact on quality of life, weight loss and comorbidities: a study comparing the biliopancreatic diversion with duodenal switch and the banded Roux-en-Y gastric bypass. Arq Gastroenterol. 2014;51(4):320–7. https://doi.org/10.1590/S0004-28032014000400010.
4. Biertho L, Lebel S, Marceau S, Hould FS, Julien F, Biron S. Biliopancreatic diversion with duodenal switch: surgical technique and perioperative care. Surg Clin North Am. 2016;96(4):815–26. https://doi.org/10.1016/j.suc.2016.03.012.
5. Aasprang A, Andersen JR, Våge V, Kolotkin R, Natvig GK. Ten-year changes in health-related quality of life after biliopancreatic diversion with duodenal switch. Surg Obes Relat Dis. 2016;12(8):1594–600. https://doi.org/10.1016/j.soard.2016.04.030. Epub 2016 May 5.
6. Søvik TT, Karlsson J, Aasheim ET, Fagerland MW, Björkman S, Engström M, Kristinsson J, Olbers T, Mala T. Gastrointestinal function and eating behavior after gastric bypass and duodenal switch. Surg Obes Relat Dis. 2013;9(5):641–7. https://doi.org/10.1016/j.soard.2012.06.006. Epub 2012 Jun 28.
7. Tonelli H, de Siqueira Rotenberg L. Emotion perception and theory of mind in obesity: a systematic review on the impact of social cognitive deficits on dysfunctional eating behaviors. Surg Obes Relat Dis. 2020;17(3):618–29. https://doi.org/10.1016/j.soard.2020.10.016.
8. Jumbe S, Hamlet C, Meyrick J. Psychological aspects of bariatric surgery as a treatment for obesity. Curr Obes Rep. 2017;6(1):71–8. https://doi.org/10.1007/s13679-017-0242-2.
9. Liu R, Nikolajczyk BS. Tissue immune cells fuel obesity-associated inflammation in adipose tissue and beyond. Front Immunol. 2019;10:1587. https://doi.org/10.3389/fimmu.2019.01587.
10. Sanmiguel C, Gupta A, Mayer EA. Gut microbiome and obesity: a plausible explanation for obesity. Curr Obes Rep. 2015;4(2):250–61. https://doi.org/10.1007/s13679-015-0152-0.

11. Maciejczyk M, Żebrowska E, Chabowski A. Insulin resistance and oxidative stress in the brain: what's new? Int J Mol Sci. 2019;20(4):874. https://doi.org/10.3390/ijms20040874.
12. Calderone A, Calabro PF, Lippi C, Jaccheri R, Vitti J, Santini F. Psychopathological behaviour and cognition in morbid obesity. Recent Pat Endocr Metab Immune Drug Discov. 2017;10(2):112–8. https://doi.org/10.2174/1872214810666161226162823.
13. Fernandes J, Ferreira-Santos F, Miller K, Torres S. Emotional processing in obesity: a systematic review and exploratory meta-analysis. Obes Rev. 2018;19(1):111–20. https://doi.org/10.1111/obr.12607. Epub 2017 Oct 10.
14. Sinha R. Role of addiction and stress neurobiology on food intake and obesity. Biol Psychol. 2018;131:5–13. https://doi.org/10.1016/j.biopsycho.2017.05.001. Epub 2017 May 4.
15. Willem C, Gandolphe MC, Roussel M, Verkindt H, Pattou F, Nandrino JL. Difficulties in emotion regulation and deficits in interoceptive awareness in moderate and severe obesity. Eat Weight Disord. 2019;24(4):633–44. https://doi.org/10.1007/s40519-019-00738-0. Epub 2019 Jun 26.
16. Tonelli H, Sartor FM, Marchesini JCD, Marchesini JB, Tonelli DG. Effects of bariatric surgery on the central nervous system and eating behavior in humans: a systematic review on the neuroimaging studies. J Bras Psiquiatr. 2013;62(4):297–305. https://doi.org/10.1590/S0047-20852013000400007.
17. Tonelli H. Efeitos da cirurgia bariátrica no sistema nervoso central. In: Tonelli H, Pereira M, Marchesini JCD, editors. Saúde Mental e Cirurgia Bariátrica. 1ª Edição. São José do Rio Preto: DLR Press; 2020. p. 43–71.
18. Lin Z, Qu S. Legend of weight loss: a crosstalk between the bariatric surgery and the brain. Obes Surg. 2020;30(5):1988–2002. https://doi.org/10.1007/s11695-020-04474-8.
19. Macht M. How emotions affect eating: a five-way model. Appetite. 2008;50(1):1–11.
20. Vögele C, Gibson EL. Mood, emotions, and eating disorders. In: Agras WS, editor. The Oxford handbook of eating disorders. Oxford University Press; 2010. p. 180–205.
21. Casagrande M, Boncompagni I, Forte G, Guarino A, Favieri F. Emotion and overeating behavior: effects of alexithymia and emotional regulation on overweight and obesity. Eat Weight Disord. 2019;25(5):1333–45. https://doi.org/10.1007/s40519-019-00767-9.
22. Volkow ND, Wang GJ, Tomasi D, Baler RD. The addictive dimensionality of obesity. Biol Psychiatry. 2013;73(9):811–8. https://doi.org/10.1016/j.biopsych.2012.12.020. Epub 2013 Jan 29.
23. Michaud A, Vainik U, Garcia-Garcia I, Dagher A. Overlapping neural endophenotypes in addiction and obesity. Front Endocrinol (Lausanne). 2017;8:127. https://doi.org/10.3389/fendo.2017.00127.
24. Krug MK, Carter CS. Anterior cingulate cortex contributions to cognitive and emotional processing: a general-purpose mechanism for cognitive control and self-control. In: Hassin RR, Ochsner KN, Trope Y, editors. Self-control in society, mind and brain. Oxford University Press; 2010. p. 3–26.
25. Swann AC. Impulsivity and affective regulation. In: Agras WS, editor. The Oxford handbook of eating disorders. Oxford University Press; 2010. p. 245–61.
26. Veldhuizen MG, Rudenga KJ, Small DM. The pleasure of taste, flavor and food. In: Kringelbach ML, Berridge KC, editors. Pleasures of the brain. Oxford University Press; 2010. p. 146–68.
27. Benzerouk F, Djerada Z, Bertin E, Barrière S, Gierski F, Kaladjian A. Contributions of emotional overload, emotion dysregulation, and impulsivity to eating patterns in obese patients with binge eating disorder and seeking bariatric surgery. Nutrients. 2020;12(10):3099. https://doi.org/10.3390/nu12103099.
28. Lavender JM, King WC, Kalarchian MA, Devlin MJ, Hinerman A, Gunstad J, Marcus MD, Mitchell JE. Examining emotion-, personality-, and reward-related dispositional tendencies in relation to eating pathology and weight change over seven years in the Longitudinal Assessment of Bariatric Surgery (LABS) study. J Psychiatr Res. 2020;120:124–30. https://doi.org/10.1016/j.jpsychires.2019.10.014. Epub 2019 Oct 19.

29. Efferdinger C, König D, Klaus A, Jagsch R. Emotion regulation and mental well-being before and six months after bariatric surgery. Eat Weight Disord. 2017;22(2):353–60. https://doi.org/10.1007/s40519-017-0379-8. Epub 2017 Apr 7.
30. Williamson TM, Campbell TS, Telfer JA, Rash JA. Emotion self-regulation moderates the association between symptoms of ADHD and weight loss after bariatric surgery. Obes Surg. 2018;28(6):1553–61. https://doi.org/10.1007/s11695-017-3037-3.
31. Cella S, Fei L, D'Amico R, Giardiello C, Allaria A, Cotrufo P. Binge eating disorder and related features in bariatric surgery candidates. Open Med (Wars). 2019;14:407–15. https://doi.org/10.1515/med-2019-0043.
32. Micanti F, Iasevoli F, Cucciniello C, Costabile R, Loiarro G, Pecoraro G, Pasanisi F, Rossetti G, Galletta D. The relationship between emotional regulation and eating behaviour: a multidimensional analysis of obesity psychopathology. Eat Weight Disord. 2017;22(1):105–15. https://doi.org/10.1007/s40519-016-0275-7. Epub 2016 Apr 11.
33. Berking M, Wupperman P. Emotion regulation and mental health: recent findings, current challenges, and future directions. Curr Opin Psychiatry. 2012;25(2):128–34. https://doi.org/10.1097/YCO.0b013e3283503669. Review.
34. Li X, Lu J, Li B, Li H, Jin L, Qiu J. The role of ventromedial prefrontal cortex volume in the association of expressive suppression and externally oriented thinking. J Affect Disord. 2017;222:112–9. https://doi.org/10.1016/j.jad.2017.06.054.
35. Wiebe A, Kersting A, Suslow T. Deployment of attention to emotional pictures varies as a function of externally-oriented thinking: an eye tracking investigation. J Behav Ther Exp Psychiatry. 2017;55:1–5. https://doi.org/10.1016/j.jbtep.2016.11.001. Epub 2016 Nov 5.
36. Damasio A. Self comes to mind. Constructing the conscious brain. Pantheo Books; 2010.
37. Brewer R, Cook R, Bird G. Alexithymia: a general deficit of interoception. R Soc Open Sci. 2016;3(10):150664. https://doi.org/10.1098/rsos.150664.
38. Percinel I, Ozbaran B, Kose S, Simsek DG, Darcan S. Increased deficits in emotion recognition and regulation in children and adolescents with exogenous obesity. World J Biol Psychiatry. 2018;19(2):112–8. https://doi.org/10.1080/15622975.2016.1265147.
39. Koch A, Pollatos O. Reduced facial emotion recognition in overweight and obese children. J Psychosom Res. 2015;79(6):635–9. https://doi.org/10.1016/j.jpsychores.2015.06.005.
40. Apperly IA. Beyond simulation-theory and theory-theory: why social cognitive neuroscience should use its own concepts to study "theory of mind". Cognition. 2008;107(1):266–83. Epub 2007 Sep 14.
41. Adolphs A, Janowski V. Emotion recognition. In: Decety, Cacioppo (eds), The Oxford handbook of social neurosciences. 2015. p. 525–41.
42. Hugenberg K, Wilson JP. Faces are central to social cognition. In: Carlston DE (ed), The Oxford handbook of social cognition. 2014. p. 167–193.
43. Adolphs R, Gosselin F, Buchanan TW, Tranel D, Schyns P, Damasio AR. A mechanism for impaired fear recognition after amygdala damage. Nature. 2005;433(7021):68–72.
44. Corneille O, Mauduit S, Holland RW, Strick M. Liking products by the head of a dog: perceived orientation of attention induces valence acquisition. J Exp Psychol. 2009;45:234–7.
45. Bartholow BD, Dickter CL. Person perception. In: Decety, Cacioppo (eds), The Oxford handbook of social neurosciences. 2015. p. 404–18.
46. Himes SM, Grothe KB, Clark MM, Swain JM, Collazo-Clavell ML, Sarr MG. Stop regain: a pilot psychological intervention for bariatric patients experiencing weight regain. Obes Surg. 2015;25(5):922–7. https://doi.org/10.1007/s11695-015-1611-0.
47. Anderson LM, Chacko TP. Bariatric outcomes: self-management for sustained surgical success: a multicomponent treatment for dysregulated overeating in bariatric surgery patients. J Addict Nurs. 2018;29(1):32–42. https://doi.org/10.1097/JAN.0000000000000210.
48. Sogg S, Lauretti J, West-Smith L. Recommendations for the presurgical psychosocial evaluation of bariatric surgery patients. Surg Obes Relat Dis. 2016;12(4):731–49. https://doi.org/10.1016/j.soard.2016.02.008. Epub 2016 Feb 12.

49. Goldberg LR. An alternative "description of personality": the big-five factor structure. J Pers Soc Psychol. 1990;59(6):1216–29. https://doi.org/10.1037//0022-3514.59.6.1216.
50. Norris L. Psychiatric issues in bariatric surgery. Psychiatr Clin North Am. 2007;30(4):717–38. https://doi.org/10.1016/j.psc.2007.07.011.
51. Kaye W. Neurobiology of anorexia and bulimia nervosa. Physiol Behav. 2008;94(1):121–35. https://doi.org/10.1016/j.physbeh.2007.11.037. Epub 2007 Nov 29.
52. Heinberg LJ, Ashton K, Coughlin J. Alcohol and bariatric surgery: review and suggested recommendations for assessment and management. Surg Obes Relat Dis. 2012;8(3):357–63. https://doi.org/10.1016/j.soard.2012.01.016. Epub 2012 Feb 2.
53. Stahl SM. Stahl's essential psychopharmacology. Neuroscientific basis and practical applications. 4th ed. Cambridge University Press; 2008.
54. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Washington, DC: DSM-V. American Psychiatric Publishing; 2013.
55. Kendler KS, Neale MC. Endophenotype: a conceptual analysis. Mol Psychiatry. 2010;15(8):789–97. https://doi.org/10.1038/mp.2010.8. Epub 2010 Feb 9.
56. Ziauddeen H, Fletcher PC. Is food addiction a valid and useful concept? Obes Rev. 2013;14(1):19–28. https://doi.org/10.1111/j.1467-789X.2012.01046.x. Epub 2012 Oct 12.
57. Kornstein SG, Kunovac JL, Herman BK, Culpepper L. Recognizing binge-eating disorder in the clinical setting: a review of the literature. Prim Care Companion CNS Disord. 2016;18(3). https://doi.org/10.4088/PCC.15r01905.
58. Amianto F, Ottone L, Abbate Daga G, Fassino S. Binge-eating disorder diagnosis and treatment: a recap in front of DSM-5. BMC Psychiatry. 2015;15:70. https://doi.org/10.1186/s12888-015-0445-6.
59. Dingemans A, Danner U, Parks M. Emotion regulation in binge eating disorder: a review. Nutrients. 2017;9(11):1274. https://doi.org/10.3390/nu9111274.
60. Colles SL, Dixon JB, O'Brien PE. Loss of control is central to psychological disturbance associated with binge eating disorder. Obesity (Silver Spring). 2008;16(3):608–14. https://doi.org/10.1038/oby.2007.99.
61. Cleator J, Abbott J, Judd P, Sutton C, Wilding JP. Night eating syndrome: implications for severe obesity. Nutr Diabetes. 2012;2(9):e44. https://doi.org/10.1038/nutd.2012.16.
62. McCuen-Wurst C, Ruggieri M, Allison KC. Disordered eating and obesity: associations between binge-eating disorder, night-eating syndrome, and weight-related comorbidities. Ann N Y Acad Sci. 2018;1411(1):96–105. https://doi.org/10.1111/nyas.13467. Epub 2017 Oct 16.
63. Fletcher PC, Kenny PJ. Food addiction: a valid concept? Neuropsychopharmacology. 2018;43(13):2506–13. https://doi.org/10.1038/s41386-018-0203-9. Epub 2018 Sep 6. Erratum in: Neuropsychopharmacology. 2018.
64. Small DM, Zatorre RJ, Dagher A, Evans AC, Jones-Gotman M. Changes in brain activity related to eating chocolate: from pleasure to aversion. Brain. 2001;124(Pt 9):1720–33. https://doi.org/10.1093/brain/124.9.1720.
65. Yokum S, Gearhardt AN, Harris JL, Brownell KD, Stice E. Individual differences in striatum activity to food commercials predict weight gain in adolescents. Obesity (Silver Spring). 2014;22(12):2544–51. https://doi.org/10.1002/oby.20882. Epub 2014 Aug 25.
66. Carnell S, Gibson C, Benson L, Ochner CN, Geliebter A. Neuroimaging and obesity: current knowledge and future directions. Obes Rev. 2012;13(1):43–56. https://doi.org/10.1111/j.1467-789X.2011.00927.x. Epub 2011 Sep 8.
67. Pedram P, Wadden D, Amini P, Gulliver W, Randell E, Cahill F, Vasdev S, Goodridge A, Carter JC, Zhai G, Ji Y, Sun G. Food addiction: its prevalence and significant association with obesity in the general population. PLoS One. 2013;8(9):e74832. https://doi.org/10.1371/journal.pone.0074832.
68. Yau YH, Potenza MN. Gambling disorder and other behavioral addictions: recognition and treatment. Harv Rev Psychiatry. 2015;23(2):134–46. https://doi.org/10.1097/HRP.0000000000000051.

69. Conceição EM, Mitchell JE, Engel SG, Machado PP, Lancaster K, Wonderlich SA. What is “grazing”? Reviewing its definition, frequency, clinical characteristics, and impact on bariatric surgery outcomes, and proposing a standardized definition. Surg Obes Relat Dis. 2014;10(5):973–82. https://doi.org/10.1016/j.soard.2014.05.002. Epub 2014 May 15.
70. Rosenberg KP, Feder LC. An introduction to behavioral addictions. In: Rosenberg KP, Feder LC, editors. Behavioral addictions. Criteria, evidence, and treatment. Academic Press; 2014. p. 1–17.
71. Sogg S. Alcohol misuse after bariatric surgery: epiphenomenon or “Oprah” phenomenon? Surg Obes Relat Dis. 2007;3(3):366–8. https://doi.org/10.1016/j.soard.2007.03.004. Epub 2007 Apr 23.
72. Holden C. ‘Behavioral’ addictions: do they exist? Science. 2001;294(5544):980–2. https://doi.org/10.1126/science.294.5544.980.