Post-Bariatric Surgery Substance Use Disorders: Prevalence, Predictors, Management, and Prevention

Karen K. Saules, PhD, and Summar Reslan, MS

ABSTRACT

- *Objective:* To review the evidence on post-bariatric surgery substance use disorders (SUDs), including a discussion of management and prevention.
- *Methods:* Review and integration of the published literature on emergence of post-bariatric SUDs with our own ongoing work in this area.
- *Results:* Emerging evidence suggests that post-bariatric surgery patients—particularly those who have had the Roux-en-Y gastric bypass and possibly gastric sleeve procedures—are at risk for developing SUDs, and about two-thirds of these cases represent new-onset SUDs, not relapses to pre-existing conditions. There are compelling physiological and psychological reasons why bariatric patients may be at risk for SUDs after surgery. Addressing patients' abilities to make myriad post-surgical adjustments without engaging in problematic behavioral patterns may be of paramount importance to optimize bariatric outcomes and prevent postoperative SUDs.
- *Conclusion:* We advocate a paradigm shift in which behavioral health providers become more involved *after* surgery in assisting bariatric patients manage dramatic adaptations regarding appearance, body image, interpersonal relationships, and health behaviors to optimize bariatric outcomes over the long term.

Prevalence rates for overweight and obesity have increased dramatically in recent decades [1], with recent estimates classifying two-thirds of American adults as overweight or obese. The World Health Organization [2] has set BMI cut-off scores for body weight classification, with a BMI ≥ 25 suggesting overweight status and BMI ≥ 30 reflecting obesity, which is further divided into Class I (30.00–34.99), Class II (35.00–39.99) and Class III (or morbid) obesity (BMI ≥ 40). Although escalating rates of overweight/obesity may be leveling off [3], it remains alarming that prevalence of clinically severe, or morbid, obesity continues to rise [4]. Specifically, from 2000 to 2005, prevalence of BMI > 30 increased by 24%; BMI > 40 increased by 50%, and BMI > 50 increased by 75% [5]. This trend has continued through 2010, although rates of morbid obesity are not increasing quite as rapidly as before 2005 [4].

Morbidly obese individuals incur greater costs associated with health care utilization, with estimates suggesting 81% greater health care costs relative to the non-obese population and 47% greater health care costs relative to their overweight, non-morbidly obese counterparts [6,7]. A more recent report indicates that the health care costs of the severely obese may be more than double those incurred by the general population [8]. In addition to physical health complications, higher BMI is associated with lower health-related quality of life [9], greater depression [10,11], and increased morbidity [12].

Dietary modification programs and pharmacologic interventions have been shown to result in modest and typically short-term weight loss [13–16], and weight loss–related improvements in obesity-related complications typically deteriorate with weight regain [17]. Because traditional weight loss interventions tend not to have enduring effects [18–20], surgical treatments have gained in popularity. Bariatric surgery, also referred to as weight loss surgery (WLS), is the most effective weight loss treatment for clinically severe or morbid obesity [21], and it is rapidly gaining popularity among the morbidly obese population. In 2008, an estimated 220,000 patients underwent WLS in the United States, and over 344,000

From Eastern Michigan University, Ypsilanti, MI.

such procedures were performed worldwide [22]. Surgical techniques are advancing rapidly and trends vary across world regions, but as of 2008 the most commonly performed procedure in the United States was the Roux-en-Y gastric bypass (RYGB; 51%), followed by adjustable gastric banding (44%) [22].

After WLS, patients lose on average 65% of excess body weight [23], and the average WLS patient will reach a peak percentage of excess weight loss (%EWL) between 18 and 24 months post-surgery, typically with some weight regain thereafter [24,25]. Recent evidence suggests better long-term weight loss outcomes for RYGB patients compared with adjustable gastric banding patients [26]. Importantly, reduced health care costs have been reported post-WLS [27]. Within 2 to 4 years post-surgery, cost savings due to lower health care utilization are estimated to offset the initial cost of the surgery, which is about $26,000. Certain conditions largely account for these cost offsets, with most patients showing fewer obesity-related conditions including diabetes, hyperlipidemia, hypertension, and sleep apnea [23]; the sustainability of these health improvements, however, depends on maintenance of weight loss over the long-term [28]. Nonetheless, improved quality of life and survival rates are well documented [29].

Despite the overall benefits associated with WLS, it is an unfortunate reality that not all WLS patients experience optimal outcomes. Several studies have observed maximal weight loss at 18 to 24 months post-WLS [24,25,30,31]. By 2 years post-WLS, two-thirds of patients begin to regain weight [32], with significant weight regain (ie, > 20% of maximum weight lost) observed in over half of patients [33,34]. Most long-term follow-up studies have very poor retention rates [35,36], and available data suggest that weight regain is associated with shame and guilt [34]. As such, weight regain may be far greater than estimated, if those with the poorest outcomes are too ashamed to return for follow-up visits. In addition to the potential for weight regain, WLS patients, RYGB patients in particular, are at risk for emergence of alcohol use disorders by 2 years post-WLS, with 16.5% of patients engaging in hazardous alcohol use and nearly 10% having an alcohol use disorder [37]. In this article, we will review the evidence on post-bariatric surgery substance use disorders (SUDs), including a discussion of management and prevention, in the context of a clinical case.

CASE STUDY

Initial Presentation

A 43-year-old Caucasian female presents for WLS at a local hospital.

History

The patient's BMI is 43 kg/m^2 (68 inches tall, 283 lb). Her medical history includes type 2 diabetes (acarbose 25 mg tid), depression (bupropion 200 mg), and headaches of unspecific origin (sumatriptan succinate 50 mg prn). Family medical history is significant for bipolar disorder and substance dependence. The patient is employed part-time in retail, enrolled in a graduate program in business, and a single mother of 3.

Pre-Surgical Psychiatric Evaluation and Support Group Attendance

The patient undergoes a pre-WLS evaluation assessing a variety of areas including developmental, psychosocial, behavioral, cognitive, emotional, and motivational domains. The patient denies a history of substance-related problems. The patient is asked to reduce her weight by 20 lb prior to receiving surgical authorization, as well as to attend pre- and postoperative WLS support groups, given her history of depression and family history of bipolar disorder. She loses 20 lb within 4 months and attends monthly psychoeducational and supportive WLS group meetings. Thereafter, the patient is authorized to undergo RYGB WLS at a local hospital.

Post-Surgical Follow-up

The patient loses 119 lb within the first 18 months after her surgery (BMI = 25 kg/m^2). Unfortunately, the patient regains 60 lb by approximately 24 months after WLS and began to experience symptoms of depression and alcohol-related problems. Although she continues to reliably attend monthly post-WLS support group meetings, the patient reports that she does not feel equipped to handle lifestyle and social environment changes.

By 30 months following WLS, the patient's alcohol consumption has increased markedly. The patient attempts to hang holiday decorations while intoxicated and falls off a ladder, breaking her leg. After this incident, she is voluntarily admitted to a SUD detoxification and rehabilitation program. Thereafter, she is referred for outpatient psychotherapy to address issues related to post-WLS coping and prevention of relapse to alcohol misuse.

- **What is the prevalence of post-surgical SUD?**

In a large-scale longitudinal study, rates of alcohol use disorders did not differ from 1 year before to 1 year after surgery, but by 2 years post-WLS the rate of problematic alcohol use increased significantly. Specifically, drinking ≥ 4 times per week and meeting alcohol dependence criteria both increased from about 3% to 5% of patients from baseline to year 2, and any form of alcohol use disorder increased from 7.6% to 9.6% during this same time frame. Notably, the escalation of problematic alcohol use was significant only among RYGB patients, not those who underwent adjustable gastric banding [37]. Although a 2% increase in problematic alcohol use over a 1-year period may not seem alarming, this level of escalation among a predominantly middle-aged Caucasian female sample is quite unexpected. King et al's [37] observation of a roughly 10% rate of problematic alcohol use and 5% rate of alcohol dependence contrasts dramatically with national estimates that only 1.7% of women aged 45 to 64 years meet criteria for DSM-IV alcohol abuse and only 1.15% meet dependence criteria [38].

Consistent with King et al's [37] observation, we have also observed high SUD rates in samples that were 2 years post-WLS [39] and 6 years post-WLS [40]. Although documentation of high rates of new-onset SUD among this predominantly female and middle-aged population is noteworthy, the overall SUD rate takes on additional significance in comparison with comparable but non-WLS populations. Data specifically on the incidence of SUD among middle-aged obese women are lacking, but a number of studies indicate that obesity is inversely or not at all associated with SUDs [41–44]. However, at least one report suggests that, unlike their non-bariatric counterparts, WLS candidates have relatively high lifetime SUD rates [11], so one might interpret the high post-WLS rates as reflective of persistence of or relapse to pre-existing SUD conditions. To the contrary, our research [39,40,45–47] suggests that about two-thirds of post-RYGB SUD cases are of the new-onset variant, ie, with no evidence of probable SUD prior to WLS.

Our research team began studying the emergence of SUDs among post-WLS patients when we had the opportunity to confirm clinical observations through a review of 7199 electronic medical records (EMRs) at a large SUD detoxification/rehabilitation program. From 2007 to 2009 [48], we observed that 2% of patients' EMRs reflected a WLS history, yet 6% of randomly pulled hard copy charts reflected a WLS history. Therefore, the 2% estimate was, if anything, an underestimate, due to inconsistent EMR data entry for the WLS diagnosis. We repeated this analysis with EMR data (n = 4658) from 2009 to 2011 [45] and found a 3% prevalence of WLS history among SUD inpatients. In related research, Fogger and McGuinness [49] observed that WLS patients were overrepresented in a state monitoring program for addicted nurses (alcohol and hydrocodone addictions). Specifically, 14.4% of those nurses had a history of bariatric surgery, an even higher rate than the 2% to 6% we observed in our inpatient SUD sample. These findings suggest that post-WLS SUD may be greater than estimated, given that those with SUDs severe enough to warrant inpatient rehabilitation or state monitoring may not be heavily represented in the larger-scale WLS community samples. Available estimates for the rate of WLS surgery over time and across years among the general population are variable (eg, 54.2 to 63.9 cases per 100,000 US adults [48], but they do not compare to the rate observed among SUD inpatients. Data from a subset of SUD inpatients and matched non-SUD WLS controls revealed that SUD patients had higher depression scores and poorer quality of life, highlighting the broad adverse effects of post-WLS SUD [51].

Recent data support that it is a composite of drug and alcohol use that increases during the 2-year post-surgery period for WLS patients, with those who have had the RYGB procedure at particularly high risk for escalation of alcohol use [52]. Given the higher lifetime prevalence of SUDs among those seeking bariatric surgery [11], it would not be surprising if these were cases in which patients were relapsing to problems that were minimized or undetected at the time of surgery. In fact, however, data suggest that about 60% of those who report drug and alcohol problems after WLS surgery had no prior history of problematic substance use, a group we refer to as new-onset SUD [45–47]. Remarkably consistent with our observation of a 60% new-onset rate, Fogger and McGuiness [49] reported that 68% of their post-WLS addicted nurses developed SUDs after WLS. Emergence of new-onset SUD in typically middle-aged middle-class (or above) Caucasian samples is noteworthy and merits further study, as this is not generally regarded to be an at-risk demographic.

In summary, King et al [37], Heinberg et al [53], and our own data [45,46,54] support that SUD rates increase by about 2 years post-WLS, particularly among those who have had the RYGB procedure, and many of these represent new-onset SUD cases [39,40,45,46]. Our data [45], however, and other recent reports [49,52,55] suggest that post-surgical SUD risk may extend to dependence on opiates and benzodiazepines.

• What is the etiology of post-surgical SUD?

There are compelling pharmacokinetic reasons why some WLS patients may develop problematic alcohol use after surgery, including evidence that RYGB patients reach higher peak blood alcohol concentrations (BACs) than controls, reach peak BAC faster, and take longer to return to baseline [56,57]. Significant pre-post RYGB WLS differences in response to alcohol have been observed, with consumption of 5 oz red wine post-RYGB yielding a peak BAC of 0.088% and detectable alcohol levels up to 88 min [58]. More specifically, in that study, after drinking 5 oz red wine, BAC was 0.024% pre-WLS, 0.059% at 3 months post-WLS, and 0.088% (ie, near or above the legal standard for intoxication, which varies by state) at 6 months post-WLS; the time to achieve sobriety (ie, BAC = 0%) was 49 minutes pre-WLS, 61 minutes at 3 months, and 88 minutes at 6 months post-WLS. Notably, patient's sex, age, and weight loss did not contribute substantially to differences in BAC. Faster absorption of ethanol, higher peak BAC, and longer duration of subjective effects in post-RYGB patients support that alcohol might serve as a stronger reinforcer in this population, thereby increasing patients' risk of developing alcohol problems.

Physiological changes resulting from gastric bypass surgery also help to explain heightened postoperative alcohol sensitivity. Specifically, the portion of the stomach that secretes alcohol dehydrogenase, an enzyme that plays a significant role in alcohol metabolism, is circumvented following the RYGB procedure [59], and rapid emptying of the gastric pouch, particularly for liquids, facilitates quick absorption of alcohol into the jejunum [60].

With respect to drug use/abuse, Sawaya et al [61] reviewed the limited literature on drug absorption among post-WLS patients and observed that many drugs are poorly absorbed after WLS; this would suggest that surgically induced physiological changes would not alter pharmacokinetics in a way that would enhance abuse liability of various drugs. To our knowledge, however, neither Sawaya et al [61] nor anyone else has directly assessed post-WLS pharmacokinetics of commonly abused prescription medications. Our data [45], that of Fogger and McGuinness [49], and a recent case report [55] suggest that post-surgical SUD risk may extend to opiates and benzodiazepines. Opioids may be attractive to WLS patients because of their effects on the gastrointestinal tract, which counter those induced by WLS. In particular, opioids decrease gastric motility and increase transit time [62], which could conceivably mitigate adverse effects associated with post-WLS "dumping syndrome" (a condition characterized by dizziness, nausea, cramps, bloating, diarrhea, fatigue, shakiness, chills, and hot flashes, particularly following consumption of sweet foods). Benzodiazepines may have abuse liability because they are rapidly absorbed in the gastrointestinal tract; the exact nature of benzodiazepine absorption among WLS patients is unknown.

Certain drugs may have post-WLS abuse liability by virtue of their ease of accessibility. That is, opioids are commonly prescribed for the management of post-surgical pain; post-WLS opioid consumption is elevated among those who are younger, male, unmarried, and who have had previous psychiatric hospitalizations [63], all of which are well documented risk factors for SUDs. Given that many post-WLS cases are middle-aged women with new-onset SUD [45,48], it is possible that opioids and benzodiazepines are attractive because (at least initially) they can be legally obtained and are commonly prescribed for valid post-WLS comorbidities.

• What support exists for the concept of "addiction transfer?"

Theoretical Support

The concept of "addiction transfer" has some credibility in popular culture, but to date has had very limited empirical support. The concept, however, does have considerable theoretical support. For example, individuals with compulsive overeating leading to morbid obesity often experience loss of control over eating, unsuccessful efforts to quit or cut down, and continued eating despite adverse consequences. These features are similar to those characterizing SUDs, eg, loss of control over intake,

unsuccessful attempts to quit or cut down, and continued use despite adverse consequences. Neurobiological mechanisms may also be shared, ie, it is generally accepted that dopaminergic brain reward circuits implicated in addiction are also activated by food reinforcement [67,68]. More specifically, activation of the mesolimbic dopamine (DA) system mediates the primary reinforcing characteristics of addictive substances [68–71], and a reduction in the availability of DA receptors has also been found among the obese [68]. While a comprehensive review of brain reward circuitry is beyond the scope of this report, reviews discussing the role of neurotransmitters in establishing and mediating reward [72], as well as the neuroanatomy and connectivity of reward circuits [73], are available.

With respect to food reward, a prominent theory by Berridge [74,75] suggests that food reward is composed of 2 distinct components: "liking," controlled by the endogenous opioid system, and "wanting," controlled by the mesolimbic DA system [75–78]. Liking, or the hedonic value of a stimulus, refers to the immediate pleasure derived from eating a given food, while wanting, or incentive salience, refers to the motivation to eat influenced by situational factors such as the exposure to food and food-related cues. This distinction between liking and wanting can also be applied to understand drug abuse [79]. Specifically, neural sensitization to incentive salience results in the brain beginning to anticipate rewarding stimuli in response to cues that have been associated with these stimuli (eg, drugs or food cues) in the past. Incentive salience thus makes the stimulus (drug or food) more desirable and attractive, so regardless of whether the drug is concomitantly "liked," "wanting" explains the development of uncontrolled addictive behavior [80–82].

Initially driven by their rewarding properties, both the overconsumption of food and initial drug use involve activation of DA [83,84] but may do so in slightly different ways. Specifically, disordered eating may activate brain reward circuitry indirectly [85], whereas drugs of abuse may activate the same circuitry through their direct chemical effects [86]. Additionally, endogenous opioids may mediate the rewarding effects of both conditions [78,87]. Therefore, some theoretical support exists for why an individual may experience difficulty in both domains (ie, substance abuse and compulsive overeating), or shift from food to substance abuse when excessive eating is no longer an option (such as in the case following bariatric surgery). In light of these shared mechanisms, Kalarchian et al [11] suggest that their observation of high lifetime SUD rates but very low current SUD rates in their bariatric candidates might be due to a shared diathesis between SUD and weight problems, such that SUD remits when eating predominates.

Bariatric surgery, however, is a novel situation that might set the stage for "addiction transfer" in a way that other weight loss methods do not. To be successful, traditional weight loss methods (like traditional drug/alcohol recovery efforts) typically require a series of cognitive and behavioral steps to consolidate behavior change and prevent relapse. In the case of WLS surgery, those potentially important mechanisms are bypassed through surgical constraints. As such, the WLS patient is abruptly confronted with an inability to overeat without experiencing discomfort, and if combined with limited coping skills for managing palatable food urges through other means, this may create conditions that fosters onset of SUDs. Approaches designed to provide more targeted and longer-term intervention may provide patients with a stronger sense of connection to the bariatric treatment team, offer them more effective coping strategies for managing high risk situations, and thereby provide an innovative solution to the problem of poor post-WLS outcomes. Given the rather high cost of WLS, if we optimize outcomes for even a subset of patients, there may be significant advantages to greater involvement of psychologists in the post-WLS period, rather than the current tactic of heavily utilization during the pre-WLS period.

Empirical Support

Only a small proportion of WLS patients encounter post-surgical SUDs, despite possibly elevated risk due to surgical constraints on how they now process and respond to drugs of abuse. When interviewed about the development of their drug and alcohol problems, 75% of post-WLS patients in a SUD detoxification/rehabilitation program suggested that some form of unresolved psychological problem was a contributor, and 85% believed that because they could no longer cope through eating, they substituted with drugs/alcohol, eg, "I gave up love of food, and compensated that with going out and drinking" [46]. These qualitative reports are backed up by preclinical literature indicating that overconsumption of palatable foods, particularly sugar, can result in behaviors and brain changes that are like those seen in addiction [64]. We have observed that WLS patients who endorse having had pre-surgical problems

with high sugar/low fat foods (a category predominantly comprised of sugar-sweetened beverages) are those most likely to develop post-WLS SUDs [65]. This finding is consistent with preclinical literature demonstrating that "sugar-addicted" rats, when forced to abstain, will increase consumption of available alcohol [64] or cocaine [66].

In a sample of post-RYGB patients (n = 1460, 80% female, mean age 53 years, mean 6 years since surgery) [40], we explored the possibility of addiction transfer and observed that those meeting criteria for a probable SUD endorsed a greater number of pre-surgical symptoms on the Yale Food Addiction Scale [88]. Nocturnal eating and environmental responsiveness to food cues were also associated with a probable post-WLS SUD. Thus, those who developed post-WLS SUD appeared to have stronger cognitive and behavioral responses to food, providing some support for putative pre-WLS "food addiction" and post-WLS addiction transfer. Unfortunately, for most patients, accurate data on the onset of both food addiction and SUD is unavailable, and it remains for future research to clarify whether this may be a bi-directional or cyclical process for some (ie, does early SUD transfer to food addiction for some, and perhaps back to SUD post-WLS?).

Admittedly, there is considerable debate about the concept of food addiction, in general, and addiction transfer from food to substances in the post-RYGB population. For example, Benton [89] lays out an extensive argument for the findings that would be necessary to support the plausibility of sugar addiction, and he lands on a rather strong conclusion that there is "no support" for such a phenomenon. His argument, however, tends to rest on the premise that sugar should operate in the manner that his comparator drug does (which varies, depending on the point being made), despite the fact that across alcohol and other drugs central mechanisms and behavioral phenotypes vary, sometimes greatly and sometimes slightly. For example, Benton's argument that naloxone does not precipitate sugar withdrawal does not negate the possibility of sugar addiction; naloxone does not precipitate withdrawal from anything except opioids. In addition, Benton's argument for the implausibility of food addiction also entirely fails to consider the vast amounts of sugar that people take in through sugar-sweetened beverages, while our recent work suggests this may be an important determinant [65]. Specifically, in our work, we have found WLS patients who endorsed having had a pre-WLS problem controlling intake of high sugar/low fat food (primarily soda/pop) were the ones most likely to transition to post-WLS SUD. Likewise, in animal studies, Avena and colleagues [64] have observed that it is a high sugar/low fat diet that is most likely to yield symptoms suggestive of addiction (bingeing, withdrawal, etc). Therefore, although the validity of the concept of food addiction remains hotly contested, we suggest it is a possibility that future research should continue to explore, particularly through more thorough and repeated assessment of beverage intake of all types [90].

Outpatient Psychotherapy

Upon presenting for treatment, the patient states, "Things were just different after the surgery. At first, things were going great. I had good support, and my eating and lifestyle and diet were all under control. Then, one night, I had this tiny little glass of wine, and it made me feel so fuzzy and so good. I don't know how a normal person would react to alcohol, but to me, it was just an amazing feeling. That's what started it. After that first taste of wine, I started drinking more and more. I was passing out and when I tried to detox myself, I started hallucinating." The patient identifies that reducing alcohol misuse, problematic eating behaviors, and symptoms of depression are her primary goals for treatment.

- **What are predictors of successful WLS outcomes?**

To identify suitable WLS candidates, most programs conduct pre-surgical evaluations, and some use very extensive batteries of psychological and medical screening procedures, but current assessment practices vary widely [91,92]. The American Society for Bariatric Surgery (now ASMBS) [93] published comprehensive recommendations for the pre-surgical assessment of WLS candidates, including behavioral, cognitive/emotional, developmental, psychosocial, and motivational domains. Their recently published clinical practice guidelines [94] suggest that "high-risk" RYGB patients should eliminate alcohol use due to impaired alcohol metabolism and risk of alcohol use disorder postoperatively, but unfortunately our understanding of who is actually at "high risk" remains quite limited.

It is important to note that WLS is increasingly being

considered a viable treatment option for morbidly obese adolescents and even younger children. A recent meta-analytic review [95] supports the short-term efficacy of WLS among adolescents and children, but it also notes that longer term prospective studies are lacking. A comprehensive review of WLS among children is beyond the scope of this review, but with respect to the issue of SUDs, this is a group that may be particularly vulnerable. Assessment, prevention, and management of SUD emergence in adolescent and younger WLS populations will be of paramount importance if WLS gains popularity among younger groups, as teens are well known to be highly vulnerable to substance abuse onset even if WLS is not a potentially complicating factor.

Although pre-WLS screening is largely devoted to assessing psychological and psychosocial factors, for the most part such variables are not strongly associated with post-WLS outcomes [31]. The most consistent predictors of successful weight loss outcomes are younger age at time of surgery [34], male gender [96], Caucasian race [97,98], and lower preoperative BMI [97,99–101]. In addition, our recent work suggests that post-surgical SUD may adversely affect weight loss outcomes [47], although perhaps not among those at the most severe (inpatient) end of the SUD spectrum [51]. Predictors of weight loss and SUD outcomes, however, are not identical; among a host of variables assessed, we have only observed family history of SUD [102] to be associated with both post-WLS weight loss and SUD outcomes. In fact, family history (but not personal history) of SUD was found to be the strongest predictor of a post-WLS SUD [47].

Currently, mental health practitioners have a heavy presence in the pre-surgical evaluation process, despite the fact that an abundance of studies have not found strong relationships between pre-surgical variables and post-WLS outcomes. Given the vast amount of effort (and expense) that has gone into identifying pre-WLS predictors of treatment outcomes, with disappointingly limited findings, we argue that the role of the mental health practitioner in bariatric care may not be best allocated to conducting comprehensive pre-WLS assessments of variables with no or minimal relationship to WLS outcomes. Instead, mental health practitioners may be best utilized by being enlisted to deliver peri- and post-ssurgical interventions designed to prevent untoward outcomes of SUD and poor weight loss and/or regain. Future work to engage patients in post-WLS treatment will be warranted, possibly by engaging third-party payers to consider novel reimbursement strategies to promote additional treatment engagement when it is most needed after WLS. It is not necessarily the case that patients must be intrinsically motivated to benefit from treatment; an abundant literature on outcomes from compulsory treatment of severe mental illness [103], mandated treatment for college drinking problems [104], contingency management [105–107], and financial incentives [108] to enhance treatment engagement supports that important gains can be made under appropriate contingencies As such, incentives for more intensive post-WLS treatment may be warranted.

- **What behavioral health issues are associated with WLS outcomes?**

Addressing patients' ability to make myriad post-WLS adjustments without falling into problematic patterns (eg, SUD, weight regain) may be of paramount importance, but to date, it is a possibility that has been largely neglected. Further, support groups tend to be psychoeducational and supportive in nature, rather than relying on empirically supported treatments for the problems experienced by those who must make major life adjustments. Such problems include emotion regulation in the face of novel stressors, learning new ways of eating, managing changes in relationships and novel social situations, and cognitively and affectively processing changes in appearance and related responses from others in the social environment. These changes are numerous, and without adequate resources to cope with them, it would not be surprising that a patient might veer into maladaptive coping strategies, such as substance use, excess eating, and/or physical inactivity. More intensive efforts by behavioral health treatment providers may be warranted with respect to (1) enlisting motivation to change, (2) identifying potential obstacles to successful outcomes, (3) developing proactive strategies for managing them if such obstacles arise, (4) promoting more effective coping skills, and (5) optimizing outcomes, both with respect to SUD and weight. If future research supports such efforts, there may be a paradigm shift in which behavioral health providers may become more involved in assisting WLS patients to manage the rather dramatic adaptations that they must make in terms

of appearance, body image, interpersonal relationships, and health behaviors to optimize WLS outcomes over the long term.

- **What interventions are available for treatment?**

Currently, there are no empirically supported interventions to treat co-occurring maladaptive eating behavior, problematic alcohol use, and depression among post-WLS patients. The Unified Protocol (UP) for Transdiagnostic Treatment of Emotional Disorders [109] was selected to assist the patient in accomplishing her treatment goals. The UP is rooted in key principles of cognitive behavioral therapy [110], integrated with techniques to enhance emotion regulation [111]. The UP has been validated for treating most anxiety disorders and unipolar depression [109]. During session one, needs and goals for treatment were identified, motivational interviewing [112] was utilized to identify motivation for and barriers to change, and psychoeducation was provided about the nature of emotions (UP Modules 1 and 2). During sessions two and three, learning to observe emotions in the present moment, identify thoughts associated with emotions, modify thinking patterns, increase cognitive flexibility, and recognize patterns of emotional avoidance were the foci (UP Modules 3 and 4). During the final session, ways to maintain treatment gains and relapse prevention were discussed (UP Module 8).

Case Conclusion

Since receiving the Unified Protocol structured intervention, the patient has maintained abstinence from alcohol, has lost 20 lb, and has not been re-hospitalized. She also reports a greater ability to cope with distress and improved social relationships.

Corresponding author: Karen K. Saules, PhD, Eastern Michigan University, Psychology Clinic, 611 W. Cross St., Ypsilanti, MI 48197, ksaules@emich.edu.

Financial disclosures: None.

REFERENCES

1. Flegal KM, Carroll MD, Ogden CL, Curtin LR. Prevalence and trends in obesity among US adults, 1999-2008. JAMA 2010;303:235–41.
2. World Health Organization (WHO). BMI classification. 2013 [cited 2013 May 24]; Available at http://apps.who.int/bmi/index.jsp?introPage=intro_3.html.
3. Flegal KM, Carroll MD, Kit BK, Ogden CL. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999-2010. JAMA 2012;307:491–7.
4. Sturm R, Hattori A. Morbid obesity rates continue to rise rapidly in the United States. Int J Obes (Lond) 2013;37:889–91.
5. Sturm R. Increases in morbid obesity in the USA: 2000-2005. Public Health 2007;121:492–6.
6. Arterburn DE, Maciejewski ML, Tsevat J. Impact of morbid obesity on medical expenditures in adults. Int J Obes (Lond) 2005;29:334–9.
7. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999-2000. JAMA 2002;288:1723–7.
8. Keating CL, Moodie ML, Bulfone L, et al. Healthcare utilization and costs in severely obese subjects before bariatric surgery. Obesity (Silver Spring) 2012;20:2412–9.
9. Fontaine KR, Barofsky I. Obesity and health-related quality of life. Obes Rev 2001;2:173–82.
10. Castres I, Folope V, Dechelotte P, et al. Quality of life and obesity class relationships. Int J Sports Med 2010;31:773–8.
11. Kalarchian MA, Marcus MD, Levine MD, et al. Psychiatric disorders among bariatric surgery candidates: relationship to obesity and functional health status. Am J Psychiatry 2007;164:328–34.
12. Sturm R, Wells KB. Does obesity contribute as much to morbidity as poverty or smoking? Public Health 2001;115:229–35.
13. Apfelbaum M, Vague P, Ziegler O, et al. Long-term maintenance of weight loss after a very-low-calorie diet: a randomized blinded trial of the efficacy and tolerability of sibutramine. Am J Med 1999;106:179–84.
14. Delinsky SS, Latner JD, Wilson GT. Binge eating and weight loss in a self-help behavior modification program. Obesity (Silver Spring) 2006;14:1244–9.
15. Wadden TA, Butryn ML. Behavioral treatment of obesity. Endocrinol Metab Clin North Am 2003;32:981–1003.
16. Wing R. Behavioral approaches to the treatment of obesity. In: Bray GA, Bouchard C, editors. Handbook of obesity: Clinical applications. New York: Marcel Dekker; 2004.
17. Engel SG, Crosby RD, Kolotkin RL, et al. Impact of weight loss and regain on quality of life: mirror image or differential effect? Obes Res 2003;11:1207–13.
18. Safer DJ. Diet, behavior modification, and exercise: a review of obesity treatments from a long-term perspective. South Med J1991;84:1470–4.
19. Wadden TA, Butryn ML, Byrne KJ. Efficacy of lifestyle modification for long-term weight control. Obes Res 2004;12 Suppl:151S–62S.
20. Wadden TA, Sternberg JA, Letizia KA, et al. Treatment of obesity by very low calorie diet, behavior therapy, and their combination: a five-year perspective. Int J Obes 1989;13 Suppl 2:39–46.

21. North American Association for the Study of Obesity and the National Heart, Lung, and Blood Institute. Practical guide to the identification, evaluation, and treatment of overweight and obesity in adults. Bethesda, MD: National Institutes of Health; 2000.
22. Buchwald H, Oien DM. Metabolic/bariatric surgery Worldwide 2008. Obes Surg 2009;19:1605–11.
23. Buchwald H, Avidor Y, Braunwald E, et al. Bariatric surgery: a systematic review and meta-analysis. JAMA 2004;292:1724–37.
24. Suter M, Calmes JM, Paroz A, Giusti V. A 10-year experience with laparoscopic gastric banding for morbid obesity: high long-term complication and failure rates. Obes Surg 2006;16:829–35.
25. Valezi AC, Mali Junior J, de Menezes MA, et al. Weight loss outcome after silastic ring Roux-en-Y gastric bypass: 8 years of follow-up. Obes Surg 2010;20:1491–5.
26. Spivak H, Abdelmelek MF, Beltran OR, et al. Long-term outcomes of laparoscopic adjustable gastric banding and laparoscopic Roux-en-Y gastric bypass in the United States. Surg Endosc 2012;26:1909–19.
27. Cremieux PY, Buchwald H, Shikora SA, et al. A study on the economic impact of bariatric surgery. Am J Manag Care 2008;14:589–96.
28. Laurino Neto RM, Herbella FA, Tauil RM, et al. Comorbidities remission after Roux-en-Y gastric bypass for morbid obesity is sustained in a long-term follow-up and correlates with weight regain. Obes Surg 2012;22:1580–5.
29. O'Brien PE. Bariatric surgery: mechanisms, indications and outcomes. J Gastroenterol Hepatol 2010;25:1358–65.
30. Christou NV, Look D, Maclean LD. Weight gain after short- and long-limb gastric bypass in patients followed for longer than 10 years. Ann Surg 2006;244:734–40.
31. Hsu LK, Benotti PN, Dwyer J, et al. Nonsurgical factors that influence the outcome of bariatric surgery: a review. Psychosom Med 1998;60:338–46.
32. de Zwaan M, Hilbert A, Swan-Kremeier L, et al. Comprehensive interview assessment of eating behavior 18-35 months after gastric bypass surgery for morbid obesity. Surg Obes Relat Dis 2010;6:79–85.
33. Abu Dayyeh BK, Lautz DB, Thompson CC. Gastrojejunal stoma diameter predicts weight regain after Roux-en-Y gastric bypass. Clin Gastroenterol Hepatol 2011;9:228–33.
34. Kruseman M, Leimgruber A, Zumbach F, Golay A. Dietary, weight, and psychological changes among patients with obesity, 8 years after gastric bypass. J Am Diet Assoc 2010;110:527–34.
35. Obeid A, Long J, Kakade M, et al. Laparoscopic Roux-en-Y gastric bypass: long term clinical outcomes. Surg Endosc 2012;26:3515–20.
36. Odom J, Zalesin KC, Washington TL, et al. Behavioral predictors of weight regain after bariatric surgery. Obes Surg 2010;20:349–56.
37. King WC, Chen JY, Mitchell JE, et al. Prevalence of alcohol use disorders before and after bariatric surgery. JAMA 2012;307:2516–25.
38. Grant BF, Dawson DA, Stinson FS, et al. The 12-month prevalence and trends in DSM-IV alcohol abuse and dependence: United States, 1991-1992 and 2001-2002. Drug Alcohol Depend 2004;74:223–34.
39. Ivezaj V. An examination of psychological risk factors for the development of substance abuse among post-bariatric surgery patients [dissertation]. Ypsilanti, MI: Eastern Michigan University; 2011.
40. Reslan S. Relationships between food reinforcement and eating behaviors to bariatric surgery weight loss and substance abuse outcomes [dissertation]. Ypsilanti, MI: Eastern Michigan University; 2012.
41. Barry D, Petry NM. Associations between body mass index and substance use disorders differ by gender: results from the National Epidemiologic Survey on Alcohol and Related Conditions. Addict Behav 2009;34:51–60.
42. Bluml V, Kapusta N, Vyssoki B, et al. Relationship between substance use and body mass index in young males. Am J Addict 2012;21:72–7.
43. McIntyre RS, McElroy SL, Konarski JZ, et al. Substance use disorders and overweight/obesity in bipolar I disorder: preliminary evidence for competing addictions. J Clin Psychiatry 2007;68:1352–7.
44. Petry NM, Barry D, Pietrzak RH, Wagner JA. Overweight and obesity are associated with psychiatric disorders: results from the National Epidemiologic Survey on Alcohol and Related Conditions. Psychosom Med 2008;70:288–97.
45. Wiedemann A, Saules K, Ivezaj V. Examination of post-bariatric patients who develop problematic substance use after surgery: prevalence and comparison of New Onset versus Relapser Groups. Clinical Obesity. In press.
46. Ivezaj V, Saules KK, Wiedemann AA. "I didn't see this coming.": Why are postbariatric patients in substance abuse treatment? Patients' perceptions of etiology and future recommendations. Obes Surg 2012;22:1308–14.
47. Reslan S, Saules K, Greenwald M, Schuh L. Development of probable substance use disorder following Roux-en-Y gastric bypass surgery. Subst Use Misuse. In press.
48. Saules KK, Wiedemann A, Ivezaj V, et al. Bariatric surgery history among substance abuse treatment patients: prevalence and associated features. Surg Obes Relat Dis 2010;6:615–21.
49. Fogger SA, McGuinness TM. The relationship between addictions and bariatric surgery for nurses in recovery. Perspect Psychiatr Care 2012;48:10–5.
50. Nguyen NT, Masoomi H, Magno CP, et al. Trends in use of bariatric surgery, 2003-2008. J Am Coll Surg 2011;213:261–6.
51. Pulcini M, Saules K, Schuh L. Psychosocial and weight loss outcomes of Roux-en-Y gastric bypass patients in inpatient treatment for substance use disorders: A matched controlled study. Clin Obes. In press.
52. Conason A, Teixeira J, Hsu CH, et al. Substance use following bariatric weight loss surgery. JAMA Surg 2013;148:145–50.
53. Heinberg LJ, Ashton K, Coughlin J. Alcohol and bariatric surgery: review and suggested recommendations for assessment and management. Surg Obes Relat Dis 2012;8:357–63.

54. Saules K, Reslan S, Schuh L. Which weight loss surgery patients are at risk for development of post-surgical substance use disorders? Poster presentation. Annual meeting of The Obesity Society; Sept 2012; San Antonio, TX.
55. Wendling A, Wudyka A. Narcotic addiction following gastric bypass surgery--a case study. Obes Surg 2011;21:680–3.
56. Hagedorn JC, Encarnacion B, Brat GA, Morton JM. Does gastric bypass alter alcohol metabolism? Surg Obes Relat Dis 2007;3:543–8.
57. Klockhoff H, Naslund I, Jones AW. Faster absorption of ethanol and higher peak concentration in women after gastric bypass surgery. Br J Clin Pharmacol 2002;54:587–91.
58. Woodard GA, Downey J, Hernandez-Boussard T, Morton JM. Impaired alcohol metabolism after gastric bypass surgery: a case-crossover trial. J Am Coll Surg 2011;212:209–14.
59. Lee SL, Chau GY, Yao CT, et al. Functional assessment of human alcohol dehydrogenase family in ethanol metabolism: significance of first-pass metabolism. Alcohol Clin Exp Res 2006;30:1132–42.
60. Horowitz M, Collins PJ, Harding PE, Shearman DJ. Gastric emptying after gastric bypass. Int J Obes 1986;10:117–21.
61. Sawaya RA, Jaffe J, Friedenberg L, Friedenberg FK. Vitamin, mineral, and drug absorption following bariatric surgery. Curr Drug Metab 2012;13:1345–55.
62. Chan L. Opioid analgesics and the gastrointestinal tract. Pract Gastroenterol 2008;64-65:37–50.
63. Weingarten TN, Sprung J, Flores A, et al. Opioid requirements after laparoscopic bariatric surgery. Obes Surg 2011;21:1407–12.
64. Avena NM, Rada P, Hoebel BG. Evidence for sugar addiction: behavioral and neurochemical effects of intermittent, excessive sugar intake. Neurosci Biobehav Rev 2008;32:20–39.
65. Fowler L, Saules K. An examination of the relationship of problematic food types to the development of substance use disorders in post-bariatric surgery patients. In preparation.
66. Carroll ME, Anderson MM, Morgan AD. Regulation of intravenous cocaine self-administration in rats selectively bred for high (HiS) and low (LoS) saccharin intake. Psychopharmacology (Berl) 2007;190:331–41.
67. Beaver JD, Lawrence AD, van Ditzhuijzen J, et al. Individual differences in reward drive predict neural responses to images of food. J Neurosci 2006;26 :5160–6.
68. Wang GJ, Volkow ND, Thanos PK, Fowler JS. Similarity between obesity and drug addiction as assessed by neurofunctional imaging: a concept review. J Addict Dis 2004;23:39–53.
69. James GA, Gold MS, Liu Y. Interaction of satiety and reward response to food stimulation. J Addict Dis 2004;23:23–37.
70. Koob GF. Neurobiology of addiction. Toward the development of new therapies. Ann N Y Acad Sci 2000;909:170–85.
71. Salamone JD, Correa M, Mingote S, Weber SM. Nucleus accumbens dopamine and the regulation of effort in food-seeking behavior: implications for studies of natural motivation, psychiatry, and drug abuse. J Pharmacol Exp Ther 2003;305:1–8.
72. Schultz W. Multiple reward signals in the brain. Nat Rev Neurosci 2000;1:199–207.
73. Baxter MG, Murray EA. The amygdala and reward. Nat Rev Neurosci 2002;3:563–73.
74. Berridge KC. Food reward: brain substrates of wanting and liking. Neurosci Biobehav Rev 1996;20:1–25.
75. Berridge KC. 'Liking' and 'wanting' food rewards: brain substrates and roles in eating disorders. Physiol Behav 2009;97:537–50.
76. Berridge KC. The debate over dopamine's role in reward: the case for incentive salience. Psychopharmacology (Berl) 2007;191:391–431.
77. Havermans RC. "You say it's liking, I say it's wanting ..." On the difficulty of disentangling food reward in man. Appetite 2011;57:286–94.
78. Pecina S, Smith KS. Hedonic and motivational roles of opioids in food reward: implications for overeating disorders. Pharmacol Biochem Behav 2010;97:34–46.
79. Berridge KC, Robinson TE, Aldridge JW. Dissecting components of reward: 'liking', 'wanting', and learning. Curr Opin Pharmacol 2009;9:65–73.
80. Robinson TE, Berridge KC. The neural basis of drug craving: an incentive-sensitization theory of addiction. Brain Res Brain Res Rev 1993;18:247–91.
81. Robinson TE, Berridge KC. The psychology and neurobiology of addiction: an incentive-sensitization view. Addiction 2000;95 Suppl 2:S91–117.
82. Robinson TE, Berridge KC. Review. The incentive sensitization theory of addiction: some current issues. Philos Trans R Soc Lond B Biol Sci 2008;363:3137–46.
83. Volkow ND, Wang GJ, Fowler JS, Telang F. Overlapping neuronal circuits in addiction and obesity: evidence of systems pathology. Philos Trans R Soc Lond B Biol Sci 2008;363:3191–200.
84. Volkow ND, Wise RA. How can drug addiction help us understand obesity? Nat Neurosci 2005;8:555–60.
85. Abizaid A, Gao Q, Horvath TL. Thoughts for food: brain mechanisms and peripheral energy balance. Neuron 2006;51:691–702.
86. Hyman SE, Malenka RC, Nestler EJ. Neural mechanisms of addiction: the role of reward-related learning and memory. Annu Rev Neurosci 2006;29:565–98.
87. Pelchat ML. Of human bondage: food craving, obsession, compulsion, and addiction. Physiol Behav 2002;76:347–52.
88. Gearhardt AN, Corbin WR, Brownell KD. Preliminary validation of the Yale Food Addiction Scale. Appetite 2009;52:430–6.
89. Benton D. The plausibility of sugar addiction and its role in obesity and eating disorders. Clin Nutr 2010;29:288–303.
90. Hedrick VE, Savla J, Comber DL, et al. Development of a brief questionnaire to assess habitual beverage intake (BEVQ-15): sugar-sweetened beverages and total beverage energy intake. J Acad Nutr Diet 2012;112:840–9.
91. Sogg S, Mori DL. The Boston interview for gastric bypass: determining the psychological suitability of surgical candidates. Obes Surg 2004;14:370–80.
92. Wadden TA, Sarwer DB. Behavioral assessment of candidates for bariatric surgery: a patient-oriented approach.

Surg Obes Relat Dis 2006;2:171–9.
93. American Society for Bariatric Surgery. Suggestions for the pre-surgical psychological assessments of bariatric surgery candidates. 2004 [cited 2011 September 28]; Available at www.asbs.org/html/pdf/PsychPreSurgicalAssessment.pdf.
94. Mechanick JI, Youdim A, Jones DB, et al. Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient--2013 update: cosponsored by American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery. Obesity (Silver Spring) 2013;21 Suppl 1:S1–27.
95. Black JA, White B, Viner RM, Simmons RK. Bariatric surgery for obese children and adolescents: a systematic review and meta-analysis. Obes Rev 2013;14:634–44.
96. Sarwer DB, Wadden TA, Moore RH, et al. Preoperative eating behavior, postoperative dietary adherence, and weight loss after gastric bypass surgery. Surg Obes Relat Dis 2008;4:640–6.
97. Carlin AM, O'Connor EA, Genaw JA, Kawar S. Preoperative weight loss is not a predictor of postoperative weight loss after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis 2008;4:481–5.
98. Harvin G, DeLegge M, Garrow DA. The impact of race on weight loss after Roux-en-Y gastric bypass surgery. Obes Surg 2008;18:39–42.
99. Chen EY, McCloskey MS, Doyle P, et al. Body mass index as a predictor of 1-year outcome in gastric bypass surgery. Obes Surg 2009;19:1240–2.
100. Dallal RM, Quebbemann BB, Hunt LH, Braitman LE. Analysis of weight loss after bariatric surgery using mixed-effects linear modeling. Obes Surg 2009;19:732–7.
101. Livhits M, Mercado C, Yermilov I, et al. Is social support associated with greater weight loss after bariatric surgery?: a systematic review. Obes Rev 2011;12:142–8.
102. Broermann C, Ivezaj V, Saules K, et al. Family history of substance abuse predicts percent excess weight loss in bariatric patients. Poster presentation at the Cleveland Clinic Obesity Summit; Cleveland, OH: October 2011.
103. Kisely S, Preston N, Xiao J, et al. An eleven-year evaluation of the effect of community treatment orders on changes in mental health service use. J Psychiatr Res 2013;47:650–6.
104. Borsari B, Hustad JT, Mastroleo NR, et al. Addressing alcohol use and problems in mandated college students: a randomized clinical trial using stepped care. J Consult Clin Psychol 2012;80:1062–74.
105. Hartzler B, Lash SJ, Roll JM. Contingency management in substance abuse treatment: a structured review of the evidence for its transportability. Drug Alcohol Depend 2012;122(1-2):1–10.
106. Ratliff JC, Palmese LB, Tonizzo KM, et al. Contingency management for the treatment of antipsychotic-induced weight gain: a randomized controlled pilot study. Obes Facts 2012;5:919–27.
107. Stitzer M, Petry N. Contingency management for treatment of substance abuse. Annu Rev Clin Psychol 2006;2:411–34.
108. Ries NM. Financial incentives for weight loss and healthy behaviours. Healthc Policy 2012;7:23–8.
109. Barlow DH, Farchione CP, Ellard CL, et al. The unified protocol for transdiagnostic treatment of emotional disorders: Therapist guide. New York: Oxford University Press; 2011.
110. Barlow DH, Craske MG. Mastery of your anxiety and panic. 4th ed: Therapist guide. New York: Oxford University Press; 2006.
111. Fairholme CP, Boisseau CL, Ellard KK, et al. Emotions, emotion regulation, and psychological treatment: A unified perspective. In: A. M. Kring, D. M. Sloan, editors. Emotion regulation and psychopathology: A transdiagnostic approach to etiology and treatment. New York: Guildford Press; 2010.
112. Miller WR. Motivational interviewing with problem drinkers. Behav Psychother 1983;11:147–72.