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Review
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Updates in Medical and Surgical Weight Loss

Melissa G. Davis[1], CNM, DNP, FNP, Bethany D. Sanders[1], CNM, MSN

The number of individuals with obesity is at an all-time high, and the rate of obesity continues to climb each year. Obesity is a chronic disease with widespread effects throughout the body. Midwives and perinatal care providers need an understanding of the etiology, pathophysiology, and interventions for obesity. A review of evidence-based diet and lifestyle modifications, medications, and surgical procedures is presented.
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INTRODUCTION

Obesity accounts for an excess of 1300 deaths per day in the United States.[1] Adult obesity prevalence in the United States was 41.7% in 2021 and has steadily increased over the past 20 years despite public health efforts.[2] Healthy People 2030 aims to decrease the obesity rate to 36% to reduce chronic diseases associated with excessive body weight.[3] Obesity is also associated with increased health care costs. Health care expenditures increase in a linear fashion beginning at a body mass index (BMI) of 30; each one unit costs an additional $253 per person per year on average.[4]

Despite obesity affecting approximately 2 out of 5 Americans, rates vary in different demographic groups. Black women have the highest rates of obesity compared with other groups, with a rate of 56.9%, whereas Asian non-Hispanic men and women have the lowest rates.[5] Obesity rates also vary across age groups, with the lowest prevalence of severe obesity in individuals over the age of 60 likely due to associated premature mortality.[6] Age-related weight gain also varies by sex, with individuals assigned female at birth being more likely to experience weight gain between ages 15 and 44.[7] Sexual minority groups experience obesity differently, as lesbian and bisexual women have higher rates of obesity compared with heterosexual women due to sociocultural factors.[8] Higher rates of obesity are associated with lower levels of education and lower income levels.[9] There are also geographic differences in obesity rates in the United States, with the highest prevalence occurring in southeastern states.[9]

Negative attitudes toward obesity and interest in weight loss solutions are pervasive in American culture. Implicit bias against individuals with obesity continues to rise, including among health care professionals.[10] Weight stigma and discrimination contribute to stress, activity levels, and maladaptive eating patterns and can contribute to obesity.[11] Media and consumer interest in weight loss has led to increased consumer demand for medications for weight loss, at times without regard to indication for or safety profile of the medication.[12]

Gynecologic and perinatal care providers are well positioned to identify individuals with obesity and thus need familiarity with available interventions to reduce the impact of associated chronic diseases and perinatal complications while maintaining awareness of their own weight biases.[13] The aim of this review is to provide midwives and other perinatal care providers a review of current understanding of obesity and an update on evidence-based diet and lifestyle modifications, medical, and surgical interventions for obesity.

DEFINITION OF OBESITY

The most common metric used to describe an individual's weight to height ratio is BMI. The Centers for Disease Control and Prevention defines normal BMI as 18.5 to 24.9 kg/m^2, overweight as 25 to 29.9 kg/m^2, and obese as 30 kg/m^2 and above.[14] Obesity is further defined as category 1 for BMIs 30 to 35, category 2 for BMI 35 to 40, and category 3 for BMI 40 or higher. Category 3 may also be referred to as severe obesity.[14] The use of BMI to define obesity fails to recognize the multifactorial nature of the disease and is not without controversy.[15] Body fatness, type of body fat, and body fat distribution are not accounted for by BMI.[15] BMI cutoffs do not predict health status or the risk of developing future disease processes; it is possible for individuals to have a BMI higher than 30 and remain in good health, without developing diabetes or hypertension.[16] Other obesity indices such as waist circumference may be more useful in determining risk of metabolic syndrome because BMI does not factor in lean muscle mass.[16] BMI does give a general idea of an individual's

[1]Vanderbilt University School of Nursing and Vanderbilt University Medical Center, Nashville, Tennessee

Correspondence
Melissa G. Davis
Email: Melissa.g.davis@vanderbilt.edu

ORCID
Melissa G. Davis https://orcid.org/0000-0001-5804-3976
Bethany D. Sanders https://orcid.org/0000-0001-7953-8054

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Quick Points

- Medications for weight loss are becoming more commonly prescribed and are very effective.
- Bariatric surgeries such as gastric bypass have been in use for more than 20 years and can lead to improvement or resolution of type 2 diabetes.
- Midwives and other health care practitioners should have a general understanding of the medications and surgeries used for weight loss.

size via a standardized formula, but other methods should be considered to determine if obesity is a disease versus a condition.[16]

ETIOLOGY AND PATHOPHYSIOLOGY OF OBESITY

Research into the mechanisms underlying obesity has evolved beyond the traditional understanding that caloric intake beyond that needed for the body's activities leads to storage of excessive energy in fat cells.[17] As a result of this emphasis on excessive calories as the driver of obesity, negative and harmful assumptions were made about individuals with obesity.[11] The rote advice to consume fewer calories than the person burns in a given day is outdated and inadequate, especially at higher BMIs. The amount of time spent on physical activity to create a negative energy balance is not a reasonable expectation; likewise, even with perfect adherence to a prescribed diet, the weight loss achieved is often not significant enough to lead to improved health.[18] Research suggests that obesity is the result of a complex interaction of environmental and genetic factors and that the role of personal responsibility has been previously overstated.[19] Single gene variants for obesity have been identified but are rare; however, family and twin studies have found that 40% to 70% of BMI is inherited, suggesting that epigenetics likely contributes to obesity.[20] Current understanding expands upon the energy balance model to create a more comprehensive theory including influences from a carbohydrate-insulin model, the oxidation-reduction model, and the obesogen model.

Within the energy balance model, weight is the result of hormonal and metabolic systems regulated by central mechanisms within the brain and peripheral signals.[21] According to this model, appetite regulation and energy expenditure arise from the hypothalamus in response to neuropeptide hormones.[22] Other parts of the brain respond to sensory input, pleasure, memory, and cognitive processes to contribute to energy balance and regulation.[22] Interactions between the gastrointestinal microbiome, peptides within the digestive tract such as glucagon-like peptide 1 (GLP-1), and the autonomic nervous system also contribute to energy regulation.[21]

The carbohydrate-insulin model focuses on the effects of hyperinsulinemia on adipose tissue deposition.[23,24] Insulin promotes glucose uptake by cells, suppresses adipose tissue from breaking down into fatty acids by inhibiting lipoprotein lipase, and supports lipogenesis through the deposition of glycogen and fat.[24] In the carbohydrate-insulin model, metabolism of high-glycemic carbohydrates results in a rapid rise in insulin secretion and downregulation of the insulin receptor, leading to insulin resistance.[23] The resulting hyperinsulinemia has an anabolic effect of glucose levels on adipose tissue rather than being available for catabolism within lean tissue.[24] Individuals with hyperinsulinemia may experience increased appetite with weight gain, as glucose is stored rather than consumed.[24]

The oxidation-reduction model is a newer theory of obesity and focuses on how modern foods and environmental exposures result in inaccurate signals about energy status from tissues, resulting in altered metabolism and obesity. The oxidation-reduction model considers how all the metabolic organs collaborate through a network of circulating metabolites created through transfer of electrons from molecules to decrease the oxidative state of the reactants.[25] Many of these reactants reside within the mitochondria of cells; however, the metabolites circulate among the body and are shared cofactors in various reactions. The oxidative-reduction system is sensitive to changes in the supply or demand of energy sources, and changes in mitochondrial content and protein expression may occur in response to environmental changes such as elevated glucose levels, food additives, or disease states such as diabetes.[25] For example, reactive oxygen species can promote either lipogenesis or lipolysis in adipocytes depending on the amount of insulin available.[23] This model suggests that epigenetic changes may occur over time due to changes in the oxidation-reduction system from environmental factors, which could account in part for the growing rates of obesity over the past decades.[23]

The obesogen model focuses on environmental influences on weight gain. Obesogens are defined as chemicals that are ingested, inhaled, or internalized through skin contact and cause adipocyte differentiation and the accumulation of white adipose tissue.[26] Exposure to these chemicals can disrupt endocrine function through numerous mechanisms such as impaired signaling and dysbiosis of gut microbiota, which has been demonstrated in animal and human studies.[26] In utero exposure to certain obesogens may result in epigenetic changes that predispose individuals to obesity later in life.[23] The list of obesogens continues to grow and includes chemicals found in pesticides, prescription drugs, food preservatives, flame retardants, plastics, nonstick coating, air pollutants, personal care products, and can linings.[23]

No single model provides a clear explanation of the origin of obesity; however, each model contributes to a greater understanding of the complex multifactorial contributors to the obesity epidemic. These processes also provide insight into why a single treatment for obesity has failed to materialize

thus far, as differences in diet, environment, and epigenetics among individuals require unique considerations.

OBESITY AS A CHRONIC DISEASE

A vestige of ancient human adaptation to times of food scarcity, genes that promote fat storage and weight gain processes have likely been retained over those that promote weight loss.[19] These protective physiologic mechanisms are hardwired into the brain and body, meaning that even after weight loss the individual who was formerly obese may be physiologically and metabolically different than other individuals.[21] In addition, these physiologic and metabolic processes are outside of conscious control.[27] This hardwiring also contributes to high rates of relapse, with weight loss and the view of obesity as a chronic disease requiring ongoing maintenance.[19]

Obesity was labeled a chronic disease by the World Health Organization in 1997. In 2013, the American Medical Association began describing obesity as a "disease state with multiple pathophysiologic aspects," and in 2021 the European Union issued a brief declaring obesity a "chronic relapsing disease."[16(p418)] Several other medical associations and societies have followed suit.[16,28] Viewing obesity as a chronic illness is not new to health care providers, but society continues to view it as a lifestyle choice rather than a disease process. It will take education and time to shift the social stigma of obesity.[28]

HEALTH RISKS ASSOCIATED WITH OBESITY

Body systems and organs are affected as increased deposition of excessive energy in the form of triglycerides in the adipose tissue occurs. Remodeling and cellular apoptosis within the adipose tissue in response to triglyceride storage leads to increased activity of macrophages and other immune system components.[29] In turn, this immune response releases proinflammatory cytokines, contributing to insulin resistence.[29] Insulin resistance can cause hyperinsulinemia and type 2 diabetes.[30] Chronic stimulation of the sympathetic nervous system can occur in obesity, resulting in elevated blood pressure and hypertension.[31]

The distribution and location of energy deposits in adipose tissue also contributes to the effects of obesity on the body. Increased visceral fat can compress the kidneys, contributing to hypertension.[31] The increase in pharyngeal tissue from increased adiposity can lead to airway compression, resulting in sleep apnea.[32] The oxygen desaturation that occurs during apneic episodes coupled with disturbances in sleep also negatively effects glucose metabolism.[32] Increased weight load on the skeletal system can alter biomechanics of the joints, particularly the knees.[21] In addition, the inflammation associated with adipose tissue may also affect joints, increasing the risk of osteoarthritis in individuals with obesity.[33] Increased intra-abdominal pressure from adipose tissue may cause gastroesophageal reflux disease, which in turn increases the individual's risk of developing Barrett's esophagus and esophageal cancer.[34]

In addition to storage of triglycerides, adipose cells secrete steroid hormones such as estrogen and cell-signaling proteins called adipokines.[35] The interaction of estrogen with adipokines and inflammatory mediators is thought to be responsible for the increased risk of breast cancer in individuals with obesity.[35] The release of free fatty acids from the breakdown of triglycerides in adipose cells can result in dyslipidemia, which in turn contributes to coronary artery disease.[21] Enlargement of liposomes within the liver in response to lipid storage can lead to nonalcoholic fatty liver disease, steatohepatitis, and cirrhosis.[21] The interactions of these obesity-related changes increase risk of congestive heart failure, chronic kidney disease, and stroke.[21]

Mental health is also affected by obesity. Individuals with obesity are 32% more likely than those at normal weight to experience depression.[36] Evidence suggests that the detrimental effect of increased body weight on depression is related to the effect of stigma toward individuals with obesity, rather than pathology of the disease itself.[37] Weight stigma occurs at both the structural and individual levels and is associated with weight gain,[38] social isolation, poor social support, poor academic performance, and discriminatory hiring practices[10] and can be a barrier to health care use.[10]

Obesity and Reproductive Health

Obesity is a common cause of ovulatory disorders in pregnancy-capable individuals.[39] Even in the absence of menstrual dysfunction, individuals with obesity experience a decrease in fertility, which persists in those using assisted reproductive technology.[39] Obesity can decrease the quality of oocytes as well as endometrial decidualization, which can have an impact implantation.[40] Ovulation and menstruation are disrupted by hormonal changes related to obesity. Levels of progesterone, follicle-stimulating hormone, estrogen metabolites, and luteinizing hormone are lower in obese individuals.[41] The amplitude of the luteinizing hormone pulse is also decreased, which also contributes to ovulation disruption.[40] Similarly, disruptions to the hypothalamus-pituitary-gonadal axis occur in individuals assigned male at birth with obesity, resulting in changes in semen parameters and hormone levels.[42]

Pregnant individuals with obesity are at higher risk of miscarriage and stillbirth than other individuals.[43] Increased risk of gestational diabetes, hypertensive disorders of pregnancy, preterm birth, cesarean birth, and intraoperative morbidity has been observed in individuals with obesity.[43] Many of the complications of pregnancy experienced by individuals with obesity are related to underlying metabolic syndrome.[41] Obesity also effects the fetus, increasing the risk of congenital anomalies, macrosomia, and shoulder dystocia.[43] The maternal effects of obesity persist beyond pregnancy, with a greater likelihood of offspring developing childhood obesity, diabetes, and heart disease later in life.[43]

During the postpartum period, individuals with obesity are at increased risk for hemorrhage, infection, and thrombosis.[44] Lower rates of breastfeeding have been observed in individuals with obesity, and those who do breastfeed tend to do so for shorter durations compared with individuals without obesity.[45] Postpartum depression is also positively correlated with increasing BMI.[46]

With the current rates of obesity in pregnancy-capable individuals, gynecologic and preconception health care

encounters are an opportunity to discuss obesity in relation to reproductive health. In individuals desiring pregnancy, a respectful discussion of weight loss interventions and appropriate referral may allow individuals to reduce their risk of future complications of pregnancy and improve the health of their offspring.

WEIGHT LOSS METHODS

Given the multifactorial nature of the disease, effective intervention to promote weight loss in individuals with obesity should be tailored to fit each person's needs. Cultural norms about weight, individual mobility, access to and affordability of food, presence or history of eating disorder, and other factors may shape the individual approach. Discussion around weight loss goals in both the short term and long term may help guide interventions. Health care providers can reassure individuals with obesity that moderate weight loss of 5% to 10% of body weight can improve mobility, blood pressure, lipid profiles, and general quality of life.[18]

Diet and lifestyle modifications and medical and surgical methods may not be mutually exclusive. Individuals with BMI greater than 50 may benefit from losing weight prior to surgery with the goal of decreasing surgical risks that accompany elevations in BMI.[47] Shared decision-making needs to be used when deciding on the best approach to weight loss. Each treatment option presents risks, benefits, and alternatives. When available, referral to a full-scope weight loss center using a multidisciplinary approach with some combination of counseling, lifestyle and dietary modifications, and weight loss medications is preferable.[18]

Lifestyle Modifications for Obesity

National guidelines for obesity interventions address 3 components of lifestyle modification, including diet, physical activity, and behavioral therapy (BT).[48] The first book about dieting was published in 1863, and countless books continue to be published about weight loss diets every year.[18] Mixed evidence exists on the effectiveness of many weight loss diets marketed toward consumers (eg ketogenic, paleo, whole foods based, and commercial programs) whereas the Dietary Approaches to Stop Hypertension and Mediterranean diets seem to be effective.[49] No diet is universally perfect; dietary counseling needs to be realistic as well as inclusive of individual preferences, access, and affordability.

Although a total of 150 minutes of moderately strenuous physical activity and 2 days of muscle strengthening exercise per week are recommended, evidence suggests that short periods of physical activity to reach the total target are as effective as longer periods.[50] A minimum of 10-minute increments is recommended and can be gradually extended or spread throughout the day. Walking is an effective method to reach activity goals as it requires minimal equipment and can be done anywhere. An inexpensive pedometer is a useful tool to begin counting steps and time spent being active.[50]

BT and cognitive behavioral therapy (CBT) are the most common psychological interventions for obesity and can be used regardless of the presence of a psychological problem.[51] The 2 types of therapies vary slightly in approach, with BT focusing on modifying specific behaviors and developing skills to address weight concerns and CBT focusing on the link between certain behaviors and dysfunctional thoughts.[51] In general, therapy by a trained interventionist is recommended for 6 months for weight loss and one year for maintenance.[48] These counseling sessions may be individual, group, community-based, or digital based on individual preferences and needs.[48]

Medical Management of Obesity

Weight loss medications are a multibillion-dollar industry.[52] Those sold in health food and nutrition supplement stores are generally not regulated or effective and are outside the scope of this article. The medications reviewed here are the most common US Food and Drug Administration (FDA)-approved drugs for the purpose of weight loss. Although these drugs are FDA-approved, many insurance companies do not cover weight loss medications. Out-of-pocket costs can range from about $11 for an older generic medication to $1300 or more for the most recent injectable medications.

Individuals prescribed these medications should expect to lose at least 5% of excess weight in the first 12 weeks of titrating dosages.[53] If the 5% threshold is not met by 12 weeks, the medication should be discontinued, as it is unlikely to begin working. If the threshold is met and the medication is well tolerated, it can be continued. Long-term use is often necessary becuase weight regain is common after discontinuation; however, few data are available about the appropriate regimen or safety of long-term use.[54]

Phentermine and Topiramate (Qsymia) and Phentermine (Adipex)

Phentermine is a sympathomimetic amine anorectic that stimulates the release of noradrenaline, dopamine, and serotonin in the brain to reduce hunger and stimulate energy expenditures.[54,55] It can be prescribed alone for short-term use or taken in a combined format with topiramate for long-term use.[56] Topiramate is an anticonvulsant; it is not fully known how it contributes to weight loss, but it has been shown to decrease binge-eating.[54] Therefore, the combination phentermine and topiramate may be particularly useful in individuals with binge-eating disorders and obesity.[18] Labeling for both phentermine and phentermine/topiramate states to discontinue treatment at 12 to 16 weeks if there is not a 4% to 5% weight loss.[18] Phentermine is contraindicated in individuals with uncontrolled or resistant hypertension, glaucoma, and in those taking selective serotonin reuptake inhibitors (SSRIs).[54] Both topiramate and phentermine are contraindicated with monoamine oxidase inhibitors.[54]

Topiramate has multiple drug interactions and requires a careful review of medications prior to prescribing. Topiramate is known to lessen the efficacy of oral contraceptives, making patient education and access to long-acting reversible contraception methods extremely important.[55] Increased risk of oral clefts, small for gestational age newborns, and preterm birth have been associated with the use of topiramate since its introduction in 1996.[55,56] Because of these associations, phentermine/topiramate has a Risk Evaluation

and Mitigation Strategy required by the FDA, which requires that pharmacists be trained to dispense the medication and that they provide patient education materials about pregnancy every time the medication is dispensed.[56] It is recommended that pregnancy-capable individuals use an effective mode of contraception and have a negative pregnancy test prior to initiating treatment.[55,56] After discontinuation, individuals planning pregnancy should consider high-dose folic acid for 3 months prior to conception to reduce the risk of oral clefts. Topiramate is also contraindicated for breastfeeding due to risk of diarrhea and somnolence in the infant.[55]

Naltrexone and Bupropion (Contrave)

Naltrexone is an opioid receptor antagonist that helps decrease cravings, whereas bupropion is a dopamine and noradrenaline reuptake inhibitor that stimulates neurons in the hypothalamus that can reduce appetite.[54,55] Combining naltrexone and bupropion leads to prolonged appetite reduction and may also help decrease reward-based eating behaviors.[54] The bupropion in this combination may help alleviate depressive symptoms in some individuals with obesity.[55] Naltrexone and bupropion can be taken long term but should be discontinued at 12 to 16 weeks if the individual has not lost 4% to 5% of excess body weight.[18] Drug interactions include beta blockers, SSRIs, and antipsychotics. Naltrexone and bupropion should be used in caution in those with a history of bipolar depression and in those with uncontrolled or resistant hypertension.[54] Bupropion is generally considered safe in pregnancy, but consideration of risk and benefit is recommended during lactation due to possible risk of seizures in the infant.[55] Naltrexone was observed to increase rates of early fetal loss in animal studies but is minimally excreted in breastmilk and should not be considered a contraindication during lactation.[55]

GLP-1 Receptor Agonists

GLP-1 is an incretin hormone released by cells in the gut in response to caloric intake that stimulate insulin release from the pancreas.[47,54] GLP-1 receptor agonists act on GLP-1 receptors in several central nervous system areas, especially the hypothalamus and brainstem, to promote reduced food intake.[54] Peripheral actions include slowed gastrointestinal emptying as well as increased insulin secretion under hyperglycemic conditions.[54] GLP-1 agonists are recommended for BMI greater than 30 or BMI greater than 27 with at least one weight-related comorbidity.[54,55]

Semaglutide (Wegovy) and liraglutide (Saxenda) are the GLP-1 agonists approved in the United States for weight loss. Similar medications are FDA-approved for type 2 diabetes management but not weight loss. Semaglutide for weight loss is given once a week via subcutaneous injection.[55] Liraglutide for weight loss is also administered via subcutaneous injection but in a daily format.[55] The most common side effects include mild to moderate nausea and vomiting, which generally improve with time.[57] GLP-1 drugs have been associated with an increased risk of bile duct and gallbladder disease, increasing the risk of the need for cholecystectomy.[57] Patients should be counseled about symptoms of disease accordingly.[57] Both semaglutide and liraglutide are contraindicated in individuals with a personal or family history of medullary thyroid cancer or type 2 multiple endocrine neoplasia.[55] Because GLP-1 receptor agonists lead to delayed gastric emptying, the American Society of Anesthesiologists has recommended that these medications be paused prior to procedures requiring anesthesia or deep sedation due to the theoretical risk of aspiration events.[58] Individuals taking daily GLP-1 doses should hold the medication on the day of the procedure, and those taking it weekly should hold it the week before.[58]

There are few data on the use of GLP-1 agonists in human reproduction. Animal studies evaluating the safety of semaglutide showed an increased risk of skeletal abnormalities, reduced growth, and higher rates of pregnancy loss whereas similar studies of liraglutide showed renal, oropharyngeal, osseus, and vascular malformations.[55] It is generally recommended that these medications be stopped at least 2 months before pregnancy.[55]

GLP-1 Receptor Agonists and GLP-1 Receptor Agonists

Tirzepatide (Mounjaro or Zepbound) is a weekly subcutaneous injection that is a dual agonist for both GLP-1 and glucose-dependent insulinotropic polypeptide.[53] It was approved by the FDA in late 2023 for weight loss in addition to its approval for type 2 diabetes management. Tirzepatide may reduce the efficacy of oral contraceptives, so individuals using this medication will need to use an alternative method for the first 4 weeks after starting the injections and for 4 weeks after every dose increase.[55] Side effects of tirzepatide are similar to those of the GLP-1 agonists.

Surgical Interventions For Weight Loss

Bariatric surgery is the most effective treatment for permanent weight loss.[47] It is recommended for individuals with a BMI 35 or higher, for those with a BMI between 30 and 35 with uncontrolled type 2 diabetes, or for those with a BMI 30 to 34.9 with other significant comorbidity.[47,54]

Bariatric surgery has a profound effect on type 2 diabetes, with 78.1% of individuals experiencing complete resolution of the disease and 86.6% experiencing disease improvement or resolution that persists beyond 2 years postoperatively.[59] Symptoms of obstructive sleep apnea are also affected, with improvement or resolution occurring within one year of surgery.[60] Other benefits of bariatric surgery and subsequent weight loss include reduction in blood pressure, improved lipid profile, improvements in polycystic ovary syndrome and infertility, and improvements in skin conditions, arthritis, and overall quality of life.[60]

Individuals planning bariatric surgery are managed by an interprofessional team including a registered dietician, psychologist, bariatric medical specialist, anesthesiology team, and bariatric surgeon.[47,60] They undergo a comprehensive physical and mental health assessment and meet with the dietician to make a long-term plan to facilitate successful weight loss.[47] Although the surgery will lead to reduced appetite, it will not change behaviors such as emotional, comfort, and stress eating or the desire for high-calorie foods, so individuals electing surgery need to commit to permanent lifestyle

changes to include a well-balanced, structured diet.[60] A diet and activity regimen may also be recommended for 2 to 12 weeks prior to surgery with the intent to reduce body fat and liver size.[47]

Gastric Bypass

The gastric bypass, also called a Roux-en-Y or gastric loop, was the first bariatric surgery developed and has been in use for more than 25 years. It is considered the gold standard of weight loss surgeries.[61,62] A smaller pouch is made from the original stomach using staples. The small intestine is divided, and a loop is attached to the new pouch and serves as a new passage for the food to leave the stomach, bypassing the duodenum. The original larger portion of the stomach connects distally to the small intestine, allowing the digestive enzymes and stomach acids to mix with the food. Because the new stomach pouch is smaller than the original stomach, the volume of food that can be eaten at a time is much small.

Individuals that have undergone gastric bypass can expect to lose 60% to 70% of excess weight within 2 years of surgery.[59] Due to the shortened digestive tract, nutrient malabsorption can occur. A multivitamin and vitamin B12 will need to be supplemented permanently.[60] Dumping syndrome can occur, most commonly with high-sugar foods, so individuals need to restrict foods with a high sugar content. The gastric bypass is typically considered the best procedure for obese individuals with type 2 diabetes due to the more rapid weight loss.[59]

Gastric Sleeve

The gastric sleeve procedure is the second most common method for surgical weight loss and is less likely to cause surgical complications.[61] The stomach is divided by stapling, removing approximately 70% of the organ in the process.[62] This procedure works by reducing total stomach volume in addition to altering the hormonal makeup in the stomach. Ghrelin is the hunger-stimulating hormone and is produced by cells located in the fundus of the stomach, which is removed during the sleeve procedure.[54] This may contribute to decreased appetite and increased weight loss.[60]

After surgery, portion sizes are reduced due to decreased stomach capacity. The average weight loss is approximately 60% of excess body weight by 2 years postoperatively.[54] Because there are no changes to the digestive tract with the gastric sleeve, there are typically no digestive or malabsorption issues postoperatively. A daily multivitamin, calcium, and vitamin D supplement may be needed due to reduced food intake.[63] The gastric sleeve may worsen gastric reflux or induce symptoms in patients without gastric reflux prior to surgery.[54]

Although weight loss from a gastric sleeve procedure is initially similar to that of the gastric bypass, there is evidence to show that the gastric bypass can result in greater weight loss at the 3- and 5-year marks.[61] One benefit of the gastric sleeve procedure is that it can be converted surgically to a gastric bypass if desired weight loss is not achieved after the initial procedure.[61]

Weight Loss Devices

For individuals who are not candidates for or need to lose weight before bariatric surgery, or desire a nonpermanent procedure, there are 2 available FDA-approved devices. These devices are both less common and less effective than bariatric surgery and less effective in terms of the amount of weight loss and the time required to lose weight.[64] Weight loss devices do not alter hormones or metabolism; their primary method of action is limiting space in the stomach and reducing oral intake. As a result, significant diet and lifestyle changes must occur for the weight loss to be permanent.[54]

The gastric band is an adjustable silicone band that is placed around the top of the stomach right below the esophagus.[54] It leads to weight loss by reducing the size of the stomach and the amount of food the stomach can hold as well as by slowing the passage of food into the stomach.[54] The inflatable band can be accessed via a port in the abdomen, allowing for inflating or deflating the band as needed. Compared with other bariatric surgeries, the gastric band is associated with the lowest rates of surgical complications, as there are no stomach or intestinal dissections.[54] The band is not permanent and can be removed at any time. Weight loss with gastric banding is slower and the total amount smaller than with other weight loss procedures.[62,64] Side effects of gastric banding can include nausea and vomiting as well as heartburn, both of which can be worse in the initial weeks to months after the band is placed and being adjusted. The gastric band is not recommended for individuals who overeat for emotional reasons.[60]

Intragastric balloons were approved by the FDA in 2019 and are placed endoscopically or swallowed by the patient before being inflated with water or air.[54] Removal typically occurs at 4 to 12 months postplacement.[54] The balloon most likely functions by displacing stomach volume and delaying gastric emptying, leading to increased feelings of satiety and decreased oral intake.[54] This device is approved for individuals with a BMI of 30 to 34.9 and at least one comorbid condition after trying diet and lifestyle modifications. Weight loss is modest and improves if combined with lifestyle modifications.[54] Possible discomforts include nausea, vomiting, and abdominal pain. There are not many long-term data, although there are few reported complications thus far.[64] Possible risks include displacement, gastric ulceration, gastric outlet obstruction, cholecystitis, and bacterial overgrowth.[64]

IMPLICATIONS FOR PRACTICE

After surgery for weight loss, it is generally recommended to postpone pregnancy for a period of 12 to 24 months. The most dramatic weight loss occurs in the first 12 to 24 months, placing the individual at increased risk of nutritional deficits that could potentially lead to fetal growth restriction or possible malformations.[40] During this time, fertility is improved due to weight loss and a return to ovulation, so contraception should be encouraged. Many bariatric providers are uncomfortable recommending or prescribing a method but consistently agree that a method is needed.[40] In addition, special consideration for pregnant individuals who have undergone bariatric surgery is needed. Guidelines for weight gain in pregnancy have not been studied in individuals

after weight loss surgery; however, the Institute of Medicine guidelines are generally accepted for use.[65] Consultation with a registered dietician can help individuals ensure that macronutrient and micronutrient needs are being met. Additional folic acid supplementation is recommended during pregnancy for individuals who have had bariatric surgery.[65] Oral glucose testing for gestational diabetes may not be tolerated or may cause reactive hypoglycemia. Alternative screening methods have not been studied.[65]

CONCLUSION

Midwives and perinatal care providers care for individuals with obesity, those who have undergone bariatric surgery, and individuals receiving medications for weight loss. Given the effects of obesity on reproductive health, care providers are also likely to provide counseling regarding interventions for obesity management. A baseline knowledge of the disease etiology, pathophysiology, and available treatment modalities and their risks and benefits is essential for nonjudgmental care of individuals with obesity.

CONFLICT OF INTEREST

The authors have no conflicts of interest to disclose.

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