

Obesity Part I: Epidemiology, Etiology and Pathophysiology, and Nonpharmacotherapeutic Treatments

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Abstract

The economist John Kenneth Galbraith wrote in the 1958 edition of *The Affluent Society* that “More people die in the United States of too much food than of too little” - an observation that is even more fitting today than it was in the 1950's. Nearly one third of adults in the United States are obese and the prevalence is increasing according to a recent report by the Institute of Medicine.¹ The prevalence of obesity increases with age for both men and women, but to a greater extent in women. The prevalence also is higher amongst Black and Hispanic persons, especially among women.² Findings from the CDC's Third National Health and Nutrition Examination Survey (NHANES III) revealed that 14% of children and 12% of adolescents were overweight.³

DEFINITIONS OF OBESITY

Obese: From Latin *obesus*, past participle of *obedere* (to devour) which derives from *ob-* (meaning to) and *edere* (meaning eat)

Websters New World Dictionary of the American Language, Second College Edition

Although any one definition of obesity is apt to be incorrect, obesity is a chronic disease characterized by an excess of adipose tissue. It should be considered a serious medical condition that can lead to significant morbidity and mortality rather than a character flaw or personal weakness. In terms of size and girth, obesity is defined as a body mass index (BMI) of 27.8 or more for men and of 27.3 or more for women¹ which corresponds to about 20% above desirable weight according to the 1983 Metropolitan Life Insurance Company tables. For a man of average height (5'9", 1.75 m) this translates to a weight greater than 85 kg (187 pounds)

and for a woman of average height (5'4", 1.63 m) a weight greater than 72 kg (158 pounds). The body mass index (BMI) is calculated by dividing the body weight (in kilograms) by the height (in meters) squared.¹ Obesity is probably best defined as any degree of excess weight that imparts a health risk. Accordingly, the National Institute of Health issued new guidelines in 1995 that define healthy weight as a BMI less than 25. At this threshold an alarming 59% of Americans are obese.¹

CONSEQUENCES OF OBESITY

Obesity, in combination with other risk factors, kills an estimated 300,000 Americans each year and it is the second most common cause of preventable deaths.⁴ Similar trends have been noted worldwide.⁵ Epidemiologic studies have revealed a clear association between obesity and the risks for cardiovascular disease, non-insulin dependent diabetes mellitus, certain forms of cancer, gallstones, some respiratory disorders, osteoarthritis, and an increase in overall mortality.⁶

In addition to the amount of excess weight, the regional distribution of body fat contributes significantly to the risk for metabolic and cardiovascular disease. Upper body obesity (the “apple” shape, android, or central fat distribution) is characterized by a waist-to-hip girth ratio of greater than 0.85 and a subscapular skinfold thickness of greater than 25 mm. In contrast, lower body obesity (the “pear” shape, peripheral fat distribution) is characterized by a waist-to-hip girth ratio of less than 0.85 and a subscapular skinfold thickness of less than 25 mm.⁷ The quartet of upper body obesity, hypertension, hypertriglyceridemia, and glucose intolerance is associated with an increased risk for

coronary heart disease, hypertension, and diabetes mellitus in both men and women. ⁷ These health risks appear to be mostly mediated by increased visceral intraabdominal fat, rather than subcutaneously distributed abdominal fat. ⁶

The impact of obesity on social functioning and economic well-being can be as devastating as the medical consequences. Men who are obese are less likely to be married. Obese women not only marry less, but also complete fewer years of schooling and have lower incomes (and more have incomes below the poverty level).⁸ Much of this is attributed to discrimination against overweight persons.⁸⁻¹⁰ The economic costs of obesity are equally shocking. Epidemiologists at Harvard University estimated that the cost of treating obesity and the consequent diabetes, heart disease, hypertension, and gallstones exceeded an appalling \$45.8 billion in 1990 accounting for 6.8% of total health care expenditures. ¹¹

REGULATION OF BODY WEIGHT

For land mammals, the ability to efficiently store food energy as fat provides a survival value when the food supply is scarce or sporadic.¹² To maintain such food stores without undergoing continual alterations in size and shape, a system to maintain the balance between caloric intake and energy expenditure is necessary. It is increasingly apparent that the body has a highly complex and sophisticated system for regulating energy balance and fat stores. ¹³ The biology of this system is only beginning to be understood but pieces of the puzzle are falling into place. Some of these pieces are outlined below.

The two major hypotheses that have been proposed as the explanation for how this system maintains a steady body weight are the set point hypothesis and settling point hypothesis.

According to the set-point hypothesis, a change in energy balance sufficient to alter body fat stores elicits a compensatory change in food intake and energy expenditure that eventually returns fat stores to their pre-set level.¹⁴ The mechanism by which the body measures its own fat stores has long been a mystery. Early evidence from parabiosis experiments (technique whereby two animals are physically joined to one another such that they share some circulation) suggested the presence of a circulating factor that appeared to signal the amount of stored fat.^{15 16} The discovery of the obese (ob) gene and its protein product in 1994 was a major breakthrough in the understanding of the systems regulating energy balance and clarified the nature of the circulating

factor. ¹⁷ The ob gene is located in adipose tissue and encodes for a hormone called leptin (from the Greek leptos, meaning thin) which is secreted by adipocytes in proportion to the level of body adipose mass. In animals, the inability to make leptin or respond to it results in excessive food intake and inappropriately decreased energy expenditure - inducing profound obesity and insulin resistance. Administration of recombinant leptin to these animals reverses these changes and induces weight loss. ¹⁸ Unlike mice with mutated ob genes, mutations in this gene have not been reported in humans. In humans, ob gene expression is prevalent, serum leptin concentrations are elevated, and the elevation of leptin correlates with the percentage of body fat. ^{19 20}

The mechanisms by which circulating levels of leptin signal the brain and trigger changes in food intake and energy expenditure recently have been the subject of considerable scientific and commercial interest. This interest has established the importance of neuropeptide Y (NP-Y) and a melanocortin receptor as key components of the system that regulate energy balance and body weight. ^{21 22 23}

Neuropeptide-Y, a 36 amino acid peptide first isolated 15 years ago from pig brains and one of the most abundant neuropeptides in mammalian brains, is a potent appetite stimulant. In the hypothalamus, NP-Y profoundly affects energy balance by stimulating appetite and increasing energy expenditure. ²⁴ NP-Y is a central effector of leptin deficiency in mice and therefore appears to function as an important mediator in the response to the low levels of leptin which occur during starvation.²¹

To round out the set point story, Friedman proposed that a loss of body fat leads to a decrease in leptin production and low circulating leptin levels which, in turn, stimulates neuropeptide Y in the hypothalamus. Neuropeptide Y interacts with its hypothalamic receptor and induces a cascade of events that includes increased food intake, decreased energy expenditure and reproductive function, decreased body temperature, and increased parasympathomimetic activity. The net result is a state of positive energy balance in which food intake exceeds energy expenditure. Conversely, an increase in body fat results in an increase in the levels of circulating leptin which induces melanocyte-stimulating hormone to interact with its receptor. This interaction leads to decreased food intake, increased energy expenditure, and increased sympathetic activity. The net result is a state of negative energy balance in which energy expenditure exceeds food intake. ²⁵

The set point hypothesis has been criticized because, if body fat stores are centrally controlled, the amount of fat in the diet should have little effect on body weight. To the contrary, epidemiologic studies document that obesity is up to three times more common in populations that consume fat compared to those who rely on sugars for most of their energy.¹ Proponents of the newer “settling point” hypothesis propose that, like a thermostat, the adipostat can be reset by factors in the environment.²⁶ This hypothesis asserts that we maintain weight when our various metabolic feedback loops, tuned by whatever susceptibility genes we carry, settle into an equilibrium with our environment. Factors that appear capable of resetting the adipostat include drugs, the composition and sensory properties of the usual diet, and the habitual level of exercise. High fat diets liberally add calories to the body and exercise subtracts them. But the ultimate influence of diet and exercise on the defended level of body fat appears not to result from this simple arithmetic. Rather, sustained consumption of a diet high in fat, for example, or regular exercise, to take another, has a tonic effect on the set-point mechanism, shifting the defended level of body fat higher or lower, respectively.

The precise areas within the brain and hypothalamus where these mediators interact to regulate eating behavior is incompletely understood. Appetite appears to be controlled by several areas in the hypothalamus; in part by a feeding center in the ventrolateral nucleus of the hypothalamus (VLH) and a satiety center in the ventromedial hypothalamus (VMH). The feeding center signals the cerebral cortex which stimulates eating. The satiety center modulates this process by sending inhibitory signals to the feeding center. Several factors may be involved in the activation of the satiety center. Since the VMH possesses insulin receptors and is insulin sensitive, elevated blood glucose or insulin levels may activate the satiety center. Distention of the stomach following a meal may serve as another inhibitory factor and this may be mediated by the peptide cholecystokinin. In addition to NP-Y, the feeding and satiety centers are sensitive to monoamines such as catecholamines and serotonin which modulate satiety, appetite, and energy expenditure.^{27 28}

The three main components of energy expenditure are the resting metabolic rate (RMR), exercise-induced thermogenesis, and food-induced thermogenesis. Thermogenesis refers to the physiologic generation of heat and therefore the loss of energy. The RMR reflects the energy

expended at rest by normal metabolic and organ functions and accounts for 60 to 75% of daily energy expenditure. The average RMR for a 70 kg adult male is about 1500 kcal daily.²⁷

The adrenergic system plays a major role in regulating energy expenditure. Beta-3 adrenoceptors are found in brown and white adipose tissue and appear to induce lipolysis and thermogenesis when activated by catecholamines.²⁹ Brown adipose tissue is distributed around the great vessels in the thorax and abdomen and serves to oxidize lipids for heat production and to rid the body of excess fat. White adipose tissue, which includes the subcutaneous and visceral fat, is more abundant. White adipose tissue serves to store energy as fat and can be transformed by lipolysis to free fatty acids.

ETIOLOGY OF OBESITY

When caloric intake exceeds energy expenditure the excess calories are stored in adipose tissue. Obesity results if this net positive balance is prolonged. Although it is often assumed that obesity results simply from overeating or a sedentary lifestyle, it is quite a bit more complex than this. Obesity is regarded as a “complex disease” because it arises from multifaceted interactions of genetic and environmental factors.³⁰ The ultimate cause of obesity is therefore an imbalance between caloric intake and energy expenditure resulting from a complex interaction of genetic, physiologic, behavioral, and environmental factors.

Evolutionary biology may explain some of the story. Over a third of a century ago Neel at the University of Michigan proposed that the pressure of natural selection endowed our ancestors with a “thrifty” genotype, which boosted the ability to store fat from each feast in order to sustain people through the next famine.¹² Unlike our hunter-gatherer ancestors whose food supply was scarce and sporadic, our food supply in western societies is abundant and constant. As a consequence, the ability to store fat efficiently has actually become a liability with obesity as the result.

It has been long known that the tendency to gain weight runs in families. Although early studies estimated that hereditary influence accounted for up to 80% of the tendency to gain weight, more recent data indicate that 33% of the BMI is attributable to genetics.¹⁰ In rare cases, human obesity results from a single gene disorder such as in the Bardet

Biedl, Prader Willi, Ahlstrom, and Cohen syndromes.¹⁸ Although mutations in the ob gene have not been found in humans, there is evidence suggesting a linkage between the ob gene and some obese populations.³¹⁻³² Persons with mutations in the gene that encodes for adipose beta-3 receptors appear prone to weight gain.³³ Two other genes that have been implicated in the development of human obesity are the genes that encode for the glucocorticoid receptor and Na-K-ATPase.³⁴⁻³⁵

Since genetic factors account for only a third of the variance in body weight, environmental influences must therefore account for the balance. Several environmental factors, involving both energy intake and energy output, contribute to obesity. For years, doubt has persisted about the contribution of excessive food intake to obesity. However, a recent study using double-labeled water to measure energy expenditure (and thus energy consumption) have erased this doubt and made it clear that obesity is associated with increased food consumption.³⁶ The high prevalence of a sedentary lifestyle, resulting from the proliferation of labor-saving machinery and contrivances, is a major environmental factor contributing to the development and maintenance of obesity in western societies. As recently noted by Stunkard “it is the rare person who works the long hours at heavy labor, and even the home presents fewer opportunities for physical activity”.¹⁰

The impact of environmental influences and a thrifty genotype is best exemplified by the plight of the Pima Indians in Arizona, a tribe whose ancestors split into two groups centuries ago. One group settled in what is now southern Arizona and the other group settled into the Sierra Madre Mountains of Mexico. Most of the Pimas in Arizona have been forced out of farming and now consume a fat-laden American diet. Despite common genetic ancestry, American Pimas have the highest reported incidence of obesity in the world and are, on average, 26 kg heavier than their Mexican Pimas relatives. This is attributed to the fact that Mexican Pimas subsist mostly on grains and vegetables and consume about half as much fat as their Arizona counterparts. They also engage in more than 40 hours of physical work a week.³⁷

Socioeconomic status is inversely correlated with the prevalence of obesity, especially among women, with lower socioeconomic status favoring the development of obesity.³⁸ Another environmental influence that has recently been recognized is smoking cessation. Although the health

benefits of smoking cessation are enormous, smoking cessation nevertheless appears to be associated with a 4 to 5 kg weight gain and therefore a small increase in the prevalence of obesity.³⁹ Incidentally, this association has not gone unnoticed by cigarette companies who, as Califano observes, “play shrewdly on this fear of fat and understand how smoking and weight are joined at the hip for young women”. The link between smoking and weight is undoubtedly the basis of advertisements for products such as Virginia Slims and Capri Superslims - with their names, slim cigarette profiles, and extremely thin models - designed to target teenage girls.⁴⁰

TREATMENT OF OBESITY

INTRODUCTION AND OVERVIEW: The basic principle is simple: If food intake is less than energy expenditure, stored calories, mostly in the form of fat, will be consumed and weight will be lost. Therefore, treatment is directed at either reducing caloric intake, increasing energy expenditure, or both. Although the principle is simple, achieving and maintaining meaningful weight loss is, more often than not, a frustrating and discouraging endeavor for obese individuals.

Weight loss is indicated for persons with health problems that will resolve with attainment of a healthier weight. Weight control is also appropriate for persons near the upper limit of the healthy weight range in order to prevent further increases.⁴¹ Treatment is important because studies suggest that even a modest degree of weight loss, if maintained, may result in health benefits for some chronically obese persons who may also be at risk for other diseases.⁴² Benefits include the amelioration of hyperinsulinism, insulin resistance, diabetes, hypertension, and hyperlipidemia. Experience with long term weight loss following surgery for obesity has demonstrated that these benefits can be significant and enduring.⁴³

Diet and exercise are the most frequently cited methods for both men and women attempting to lose weight.⁴¹ However, many forms of therapy are used and promoted including countless fad diets, herbal remedies, acupuncture, accupressure, appetite-suppressing “aroma sticks”, drugs, surgery and more. The treatment of obesity is a multibillion dollar industry and Americans spend over \$30 billion yearly in weight loss

efforts.⁴¹ Quick weight loss schemes abound, fad diets come and go, and unscrupulous promoters prey upon vulnerable persons desperate to lose a few pounds. The results of longer term drug studies coupled with the recent approval of dexfenfluramine have spawned a myriad of for-profit prescription weight loss clinics. A newspaper advertisement for one local “medical facility”, depicting feminine buttocks in tight-fitting jeans, promises an “easy and hip weigh” to lose weight and offers a prescription for the “Wonder Pills” Phen-fen or Redux. When evaluating a weight loss method or program, persons desiring to lose weight should not be distracted by anecdotal “success” stories and unscrupulous advertising claims. As recommended by the 1993 NIH Technology Assessment Panel, evaluation of a weight loss program should be based on the following information ⁴¹:

- The percentage of all beginning participants who complete the program.
- The percentage of those completing the program who achieve various degrees of weight loss.
- The proportion of weight loss that is maintained at 1, 3, and even 5 years.
- The percentage of participants who experienced adverse medical or psychological events and the kind and severity of adverse events.

Additional information regarding program characteristics that should be obtained include:

- The relative mix of diet, exercise, and behavior modification.
- The amount and type of counseling. Individual and closed groups (membership does not change except by attrition) are more effective methods of counseling than open groups in which members may come and go.
- The nature and availability of medical, nutritional, psychological, physiological, and exercise expertise.
- The training provided for relapse prevention to

manage high-risk emotional and social situations.

- The nature and duration of the maintenance phase.
- The flexibility of food choices and suitability of food types.
- Whether weight goals are set unilaterally or cooperatively with the program director.

The ultimate measure of success of a weight-loss program is the ability of the program to help the individual maintain a stable weight or a reduced weight and, ultimately, to improve health. Even in highly structured, medically supervised programs, the drop-out rate is high, and maximum weight loss rarely exceeds 10% of the initial body weight for those who complete the program. The rate of weight loss is typically less than 1.5 pounds per week. Furthermore, if the pattern of eating and activity is not permanently altered, most people regain the lost weight over the next 1 to 5 years. Methods where the objective is short-term, rapid or unsupervised weight loss, or that rely on diet aids such as drinks, prepackaged foods, or diet pills, but do not include education and guidance in the transition to a permanent pattern of healthy eating and activity have never been shown to lead to long-term success. Regardless of how much weight a person would like to lose, modest goals and a slow course will maximize the probability of losing and maintaining weight. It should be recognized that for most people, achieving a body weight or figure like those often depicted by the media is not a reasonable, appropriate, or achievable goal. Therefore, failure to achieve this “look” does not imply a weakness of will power or character. ⁴¹

As with the treatment of any chronic disease, therapy for obesity may lead to adverse effects. Adverse effects associated with weight loss treatment include poor nutrition, possible development of eating disorders, weight cycling, and psychological consequences of repeated failings to lose weight. Medical supervision of weight loss is strongly recommended for severely obese persons, pregnant or lactating women, children, persons over the age of 65 years, and those with serious medical conditions.

The five medically-accepted treatment modalities currently available are diet modification, exercise, behavior modification, drug therapy, and surgery. All of these modalities, alone or in combination, are capable of inducing weight loss sufficient to produce significant health benefits in many obese persons. Unfortunately, health benefits are not maintained if weight is regained and, with the exception of surgery, it is difficult to adhere to these modalities in a manner sufficient to maintain long term weight loss.

DIET MODIFICATION: According to Bray, the medical history of obesity can be traced to the stone age.⁴⁴ It appears that obesity was recognized early on as an imbalance between eating and energy expenditure. From the time of Hippocrates until the twentieth century, diet and exercise served as the primary modes of therapy.⁴⁴ The following advice, attributed to Hippocrates, appears to reflect this philosophy: Obese people and those desiring to lose weight should perform hard work before food. Meals should be taken after exertion and while still panting from fatigue and with no other refreshment before meals except only wine, diluted and slightly cold. Their meals should be prepared with sesame or seasoning and other similar substances and be of a fatty nature as people get thus satiated with little food. They should, moreover, eat only once a day and take no baths and sleep on a hard bed and walk naked as long as possible.⁴⁵

Calorie restriction has remained as the mainstay for the treatment of obesity. Diets range from mild caloric deprivation to total starvation. One of the best ways to achieve a healthier weight is to adopt a healthful diet with an energy intake that does not exceed expenditure, is low in fat, and provides adequate amounts of all food groups, including whole grains and cereals, fruits, and vegetables.⁴⁶ The more restrictive the diet, the more rapid the weight loss, but the greater the risk of noncompliance. In general, a caloric deficit of about 7,700 kcal leads to a loss of about one kilogram (2.2 pounds) of fat. Estimation of total daily caloric needs (about 25-30 kcal per kilogram of body weight) allows one to calculate the daily calorie deficit required to achieve a given rate of weight loss.²⁷

Studies employing moderate caloric restriction (defined as 1,200 Kcal/day for obese women or 1,500 Kcal/day for obese men), in combination with behavior modification, show that 80% of obese persons will remain in treatment for

20 weeks and that about half will achieve a weight loss of 9.1 kg (20 lbs) or more. About 90% of patients on a very low calorie diet (less than 800 Kcal/day) and behavior modification will attain a weight loss of similar magnitude and that 50% will attain a loss of 18.2 kg (40 lbs) or more. Unfortunately, most of these persons regain their lost weight.⁴⁷ In 1958, Stunkard observed that “most obese persons will not stay in treatment for obesity. Of those who stay in treatment, most will not lose weight, and of those who do lose weight, most will regain it”.⁴⁸ The more restrictive the diet the worse the compliance and the greater the lean body mass loss. Very-low-calories diets are associated with transient fatigue, hair loss, dizziness, and other symptoms. More serious adverse events associated with periods of severe caloric restriction include the development of gallstones and acute gallbladder disease. The risk for cardiac dysrhythmias and death, which occurred in early studies, has been eliminated by supplementing diets with high quality protein, minerals, and electrolytes.⁴¹

PHYSICAL ACTIVITY AND EXERCISE: Like caloric restriction, exercise is a fundamental, albeit underutilized modality for weight control. Although many overweight persons are trying to lose weight, only a small fraction of these are engaging in the recommended amount of physical activity. The addition of an exercise program to diet modification results in more weight loss than dieting alone and seems to be especially helpful in maintaining weight loss and preserving lean body mass. Most controlled studies show that exercise training induces modest weight loss in the range of 2 to 3 kg. Obese persons who exercise do not tend to compensate for their increased energy expenditure by increasing their food intake. Thus they lose weight at a rate that is directly proportional to the amount of energy expended during exercise. Most importantly, overweight persons who are active and fit have a lower rate of morbidity and mortality compared to overweight persons who are sedentary and unfit.⁴⁹

Individuals should seek to obtain about 1,000 kcal (4,200 kJ) per week of moderate physical activity above and beyond the activities of daily living. This amount of physical activity corresponds to about 30 to 40 minutes (150-200 kcal per exercise period) 5 to 7 days per week.^{46,50} A wide variety of physical activities count as exercise including yard work and many recreational activities (eg. dancing, tennis, soccer, basketball, golfing without riding a cart) in addition to the traditionally recognized aerobic exercises (eg. walking, running, bicycling, swimming). In addition to aerobic

physical activity, stretching and resistance training (eg. weight training, rubberized Therabands) to improve flexibility and muscular strength is desirable. People who maintain or improve their strength and flexibility appear better able to participate in aerobic exercises and perform daily activities.⁵⁰ There is evidence that the benefits of physical activity can be obtained through brief exercise periods that do not necessarily need to be continuous. For example, three 10-minute periods of moderate-intensity (intensity eliciting a heart rate of 65-75% of peak heart rate attained on a treadmill) aerobic exercise are equivalent to a single 30-minute period of exercise. For many individuals these short bouts of exercise training may fit better into a busy schedule than a longer exercise session.⁵¹

BEHAVIOR MODIFICATION: Behavior modification for obesity refers to a set of principles and techniques designed to modify eating habits and physical activity and appears to be most helpful for mildly to moderately obese persons. Many of the recent and better-designed studies evaluating the efficacy of appetite-suppressants have combined the drug therapy with behavioral approaches to improved diet and exercise. In most programs, therapy typically consists of weekly, hour-long sessions in a group format. The average length of treatment is 18 weeks although long-term programs lasting for 6 months or more are increasingly common. Behavior modification, in conjunction with diet and exercise, induces an average weight loss of about 10 kg and about 68% of this weight loss is maintained at 52 weeks.⁵²

Examples of principles and techniques used include self-observation and self-recording which are the mainstays of behavioral modification programs. The patient records the situational factors, behaviors, thoughts, moods and feelings that occur before, during, and after attempts at prudent eating and exercising. Stimulus control involves modifying environmental factors that lead to inappropriate eating or exercise. Examples of this modality include keeping away from high-fat foods, eating at specific times and places, and setting aside a time and place to exercise. Contingency management bestows rewards or prizes for appropriate eating and exercise behavior that leads to weight loss or maintenance of weight loss. Cognitive-behavioral modification focuses on strategies to counter the thoughts, moods, diets, and social pressures to be thin.⁵²

SURGERY: Surgery is considered the treatment of choice for well-informed and motivated severely obese (are more than 100% overweight or whose BMI is greater than 40) adults who fail to respond to medical weight control. Surgery may also be considered for those with less severe obesity (BMI between 35 and 40) afflicted with disabling joint disease, pulmonary insufficiency, hypertension, or diabetes mellitus. However, surgery is not yet recommended for severely obese children or adolescents because this population has not been adequately studied and experience is therefore limited.^{43,53}

The operations recognized by the 1991 NIH Consensus Conference are the vertical banded gastroplasty and the Roux-en-Y gastric bypass. ⁴³ The vertical banded gastroplasty restricts food intake by limiting gastric volume whereas the gastric bypass operation not only limits gastric volume but also results in stomach contents bypassing the distal stomach, duodenum, and proximal jejunum. Although some malabsorption of nutrients occurs, most of the weight loss is attributed to delayed gastric emptying and a feeling of fullness that causes patients to limit food intake. Both of these procedures can be reversed at a later date if required.

Weight loss following either of these operations is initially rapid and reaches a plateau at 18 to 24 months. Following the vertical banded gastroplasty, average postoperative weight loss levels out at one year at an average of 60% to 70% above ideal body weight. Postoperative weight loss from the more radical Roux-en-Y gastric bypass tends to be greater than that induced by the vertical banded gastroplasty and levels out at an average of 30% to 40% above ideal body weight. A slight degree of weight regain is common by 2 to 5 years after surgery.

The effect of surgery on comorbid conditions is impressive. There is marked improvement in glucose tolerance often with amelioration of Type II diabetes, allowing patients to reduce or discontinue insulin. Weight-reduction surgery ameliorates hypertension as well as sleep apnea and obesity-related hyperventilation. Lipid abnormalities improve with a decline in serum cholesterol and triglycerides and an increase in high-density lipoprotein (HDL) cholesterol. Improvement in musculoskeletal disability, which nearly always accompanies surgical weight control, results in less arthritic pain and improved mobility. Surgery is also

associated with improvements in psychosocial functioning, employment rate, annual income, and some quality-of-life measures.⁵⁴ Most importantly, there is data to suggest that life expectancy may be increased.⁵⁵

Mortality associated with surgery is low and is reported to be in the range of 0.3% to 1.6% in experienced centers. Major postoperative complications are not unlike those associated with other major abdominal operations which include wound infection and thromboembolism. Long term complications after gastric bypass include micronutrient deficiencies (particularly of iron, vitamin B12 and folate) that require meticulous nutritional and metabolic monitoring to avoid hematologic and neurologic sequelae. A so-called “dumping syndrome” can occur after gastric bypass. The syndrome is attributed to rapid absorption of carbohydrate into the jejunum and is characterized by gastrointestinal distress (abdominal cramping, diarrhea) and other symptoms (sweating, chills, flushing). 27,53

FURTHER READING

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SUGGESTED LINKS

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