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AHA Scientific Statement

Clinical Implications of Obesity With Specific Focus on Cardiovascular Disease

A Statement for Professionals From the American Heart Association Council on Nutrition, Physical Activity, and Metabolism

Endorsed by the American College of Cardiology Foundation

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***Abstract*—**Obesity adversely affects cardiac function, increases the risk factors for coronary heart disease, and is an independent risk factor for cardiovascular disease. The risk of developing coronary heart disease is directly related to the concomitant burden of obesity-related risk factors. Modest weight loss can improve diastolic function and affect the entire cluster of coronary heart disease risk factors simultaneously. This statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism reviews the relationship between obesity and the cardiovascular system, evaluates the effect of weight loss on coronary heart disease risk factors and coronary heart disease, and provides practical weight management treatment guidelines for cardiovascular healthcare professionals. The data demonstrate that weight loss and physical activity can prevent and treat obesity-related coronary heart disease risk factors and should be considered a primary therapy for obese patients with cardiovascular disease. **(*Circulation*. 2004; 110:2952-2967.)**

Key Words: AHA Scientific Statements ■ obesity ■ cardiovascular diseases ■ exercise ■ diet

Obesity is an important risk factor for coronary heart disease (CHD), ventricular dysfunction, congestive heart failure, stroke, and cardiac arrhythmias. Weight loss in obese patients can improve or prevent many of the obesity-related risk factors for CHD (ie, insulin resistance and type 2 diabetes mellitus, dyslipidemia, hypertension, and inflammation)[1,2] and can improve diastolic function.[3] Therefore, it is important for cardiovascular healthcare professionals to understand the clinical effects of weight loss and be able to implement appropriate weight-management strategies in obese patients. The purpose of this statement is to review the physiological and cardiovascular effects of weight loss and provide clinicians with appropriate treatment guidelines for weight management in patients with obesity and cardiovascular disease.

Clinical Effects of Weight Loss

Body Composition

The increase in body fat mass in most obese persons represents primarily an increase in the size of fat cells, although the number of fat cells may also be increased, particularly in people with childhood-onset obesity.[4] In addition, the specific distribution of excess fat can influence the relationship between obesity and cardiac disease. Excess abdominal adipose tissue, particularly visceral fat, and excess triglyceride content in liver, skeletal muscle, and heart tissues are associated with hepatic and skeletal muscle insulin resistance, impaired ventricular function, and increased CHD.[5–9]

Although an energy deficit of ≈3500 kcal is needed to oxidize 1 lb of adipose tissue, a 3500-kcal energy deficit will cause a >1-lb loss in body weight because of the oxidation of lean tissue and associated water losses. Approximately 75% of weight lost by dieting is composed of fat and 25% is fat-free mass (FFM).[10] The addition of exercise training to a diet program can decrease the percentage of weight lost as FFM by half.[10,11] Most, if not all, of the loss of fat results from a decrease in the size (triglyceride content) of existing fat cells,[12] not a decrease in the number of fat cells.[13] The distribution of fat loss is heterogeneous, with greater relative

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losses of intraabdominal fat than total body fat mass, particularly in men and women with increased initial intraabdominal fat mass.[14] In addition, diet-induced weight loss decreases intramyocellular[15] and intrahepatic[16] lipids.

Clinical Outcomes

Intentional weight loss can improve or prevent many of the obesity-related risk factors for CHD (ie, insulin resistance and type 2 diabetes mellitus, dyslipidemia, hypertension, and inflammation). Moreover, these metabolic benefits are often found after only modest weight loss (≈5% of initial weight) and continue to improve in a monotonic fashion with increasing weight loss.[17]

Metabolic Syndrome

The metabolic syndrome represents a constellation of physical and metabolic abnormalities that are risk factors for cardiovascular disease. The characteristics of this syndrome, as defined by the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III [ATP III]), include large waist circumference, insulin-resistant glucose metabolism (impaired fasting glucose, impaired glucose tolerance, and type 2 diabetes mellitus), dyslipidemia (high triglyceride and low serum HDL-C [cholesterol] concentrations), and increased blood pressure.[18] Patients who have the metabolic syndrome have a 1.5- to 3-fold increase in the risk of CHD and stroke.[19–21] Weight loss can improve all features of the metabolic syndrome.[17]

Insulin Resistance and Type 2 Diabetes Mellitus

Insulin sensitivity, with regard to glucose metabolism, improves rapidly after beginning an energy-deficit diet before much weight loss occurs and continues to improve with continued weight loss.[22] In patients with obesity and type 2 diabetes mellitus, a 5% weight loss at the end of 1 year of dietary therapy can decrease fasting blood glucose, insulin, hemoglobin A_{1c} concentrations, and the dose of oral hypoglycemic therapy,[23] whereas an average weight loss of ≈30% in extremely obese patients with diabetes after gastric bypass surgery resulted in normalization of blood glucose and glycosylated hemoglobin concentrations in 83% of patients.[24]

Weight loss can also prevent the development of new diabetes in high-risk persons who are overweight or obese.[25–28] Lifestyle dietary and activity modifications, which resulted in modest (≈5%) weight loss, decreased the 4- to 6-year cumulative incidence of diabetes by >50% in men and women who were overweight or obese and had impaired glucose tolerance.[25,26] The Swedish Obese Subjects (SOS) Study demonstrated that greater weight losses (≈16% of body weight) induced by gastric surgery in patients who are extremely obese (initial body mass index [BMI; weight in kilograms divided by height in square meters] of 41 kg/m^2) were associated with a 5-fold decrease in the cumulative incidence of diabetes for 8 years.[27]

Dyslipidemia

Weight loss decreases serum LDL-C and triglyceride concentrations, whereas increases in serum HDL-C typically are seen when weight loss is sustained.[1,29,30] The greatest relative improvements in serum triglyceride and LDL-C usually occur within the first 2 months of weight loss.[31] The beneficial effects on serum lipids are related to the percentage of weight lost, and regaining the lost weight leads to a relapse in serum concentrations. A sustained weight loss of ≥5% is needed to maintain a decrease in serum triglyceride concentrations, whereas serum total and LDL-C revert toward baseline if a ≥10% diet-induced weight loss is not maintained.[31,32] In contrast, data from the SOS study showed that an average weight loss of 33% at 2 years after bariatric surgery decreased serum triglyceride concentrations and increased serum HDL-C concentrations, but it did not affect serum total cholesterol.[2]

Hypertension

Weight loss decreases both systolic and diastolic blood pressure in a dose-dependent fashion; therefore, greater weight loss is generally associated with greater improvement in blood pressure.[33,34] Weight regain results in a steady increase in blood pressure toward baseline. The results of retrospective analyses of large surgical group experiences showed that marked weight loss induced by gastric surgery improved or completely resolved hypertension in ≈67% of patients.[35,36] In contrast, data from the SOS study revealed that on average blood pressure began to progressively increase 2 years after surgery.[27] Most subjects enrolled in the SOS study underwent vertical banded gastroplasty or gastric banding procedures and lost less weight than those who underwent gastric bypass. Subjects who had gastric bypass surgery maintained a decrease in both systolic and diastolic blood pressure for 5 years after surgery.[37]

Diet-induced weight loss can prevent the development of hypertension in persons who are obese. The results from large epidemiological studies and intervention trials suggest that the risk of developing hypertension in normotensive women is inversely correlated with changes in body weight.[33,38] Data from the SOS study showed, however, that the beneficial effect of gastric surgery-induced weight loss in preventing new cases of hypertension disappeared 3 years after surgery, despite persistent weight loss.[27]

Pulmonary Disease

Obesity is associated with altered pulmonary function. A marked excess in abdominal fat mass can mechanically interfere with lung function because of the increased weight on the chest wall and thoracic cage. In addition, obesity is associated with serious pulmonary diseases, obstructive sleep apnea (OSA), and obesity hypoventilation syndrome (OHS).

OSA is characterized by multiple episodes of apnea and hypopnea during sleep caused by partial or complete upper airway obstruction. The interruption in nighttime sleep and hypoxemia causes daytime sleepiness and cardiopulmonary dysfunction. Episodes of oxygen desaturation during apnea and hypopnea cause transient increases in pulmonary artery and pulmonary wedge pressures, and myocardial perfusion defects.[39] Over time, electrocardiographic abnormalities and cardiac rhythm alterations, permanent pulmonary hypertension, right ventricle hypertrophy, and bilateral leg edema can develop.[40–42]

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OHS is caused by a decreased ventilatory response to hypercapnia, hypoxia, or hypercapnia and hypoxia and inadequate respiratory muscle strength to meet the increased ventilatory demand caused by the mechanical effects of obesity. Patients with OHS have shallow and inefficient breathing, and a pCO_2 >50 mm Hg. Patients may become more symptomatic when lying down because abdominal pressure pushes up the diaphragm, which increases intrathoracic pressure and reduces respiratory capacity. Pickwickian syndrome is a severe form of OHS and is associated with extreme obesity, irregular breathing, cyanosis, somnolence, and right ventricular dysfunction.

Inflammation

Obesity is associated with an increase in circulating inflammatory markers, including C-reactive protein (CRP)[43–45] and cytokines (ie, interleukin-6 [IL-6], IL-18, and P-selectin).[46–49] Adipose tissue itself is a likely source of these excess cytokines,[46,50] and IL-6 stimulates the production of CRP by the liver.[51] The increase in inflammatory markers is associated with insulin resistance[52–56] and is an important predictor of atherosclerotic events.[57–61]

Data from studies that have ranged in duration from 3 months to 2 years have revealed that weight reduction decreases plasma CRP concentration.[49,52,62–67] The decrease in CRP is directly related to the amount of weight loss, fat mass, and change in waist circumference. In one study, only subjects who were insulin resistant experienced a weight loss–induced decrease in CRP, an effect that paralleled changes in insulin sensitivity.[52] Plasma CRP concentrations did not decrease and insulin sensitivity did not increase in subjects who were insulin sensitive before weight reduction. Decreases in plasma IL-6,[48,49,65,67–69] IL-18,[49,67] P-selectin,[48] and tumor necrosis factor-α[48] concentrations have also been reported[66,68,69] after weight loss in subjects who are obese.

Autonomic Nervous System Dysfunction

Overweight and obesity are associated with cardiac autonomic neuropathy. For example, a 10% increase in body weight is associated with a decline in parasympathetic tone and an increase in heart rate.[70] Alterations in autonomic nervous system function might be an important cause of cardiovascular disease events and mortality, as suggested by the relationship between heart rate and cardiovascular disease mortality.[71,72] Marked weight loss induced by bariatric surgery increases vagal activity.[73] In addition, weight loss achieved by dieting also increases cardiac parasympathetic activity,[74–77] but this increase is not maintained in the absence of sustained weight loss.[77]

Cardiovascular Disease

Although weight loss modifies many cardiovascular disease (CVD) risk factors, it is not known whether weight reduction decreases CVD events or CVD mortality in obese persons.[78–80] This important question has not yet been answered because it is difficult to achieve prolonged periods of sustained weight reduction (eg, >5 years) with nonsurgical therapy[81] and to perform prospective randomized controlled trials (RCTs) involving bariatric surgery. Data from the SOS study showed that despite a greater reduction in weight and CVD risk factors after surgical than medical therapy for obesity, no difference in cardiovascular disease events or mortality was found at 10 years.[82]

Data from large population studies have revealed that obesity is associated with increased CVD mortality.[83–87] Moreover, CVD death rates are directly related to BMI in both men and women. The risk of CVD mortality in obese persons who have a BMI $\geq$35 kg/m^2 was 2 to 3 times the risk among lean persons (BMI 18.5 to 24.9 kg/m^2),[88] and a 30% higher CHD mortality rate occurs for every 5-unit increment of BMI.[89] In addition, overweight in adolescence is associated with a 130% increased risk of CHD mortality in adulthood.[90]

In general, data from large epidemiological studies have shown that weight variability is associated with an increased rate of CVD mortality.[91] The interpretation of the results from these studies is complicated because many studies assessed weight variability rather than weight loss, included large numbers of lean and mildly overweight subjects, and included subjects who experienced "unintentional" weight loss, which may have been caused by diseases that influence mortality. Therefore, the available data are not adequate to reliably determine whether intentional weight loss affects CVD mortality, and carefully designed RCTs are needed to address this issue.

Cardiovascular Structure and Function

Obesity, particularly severe obesity, is associated with abnormalities in cardiac structure and function.[8,92] The severity of these defects is associated with both the degree and duration of obesity.[93] Obesity is associated with an increase in total blood volume and cardiac output and a decrease in peripheral vascular resistance.[8,94] In this setting, ventricular filling pressures are elevated,[95] which eventually results in increased wall stress, diastolic dysfunction, and left ventricular hypertrophy.[93,96,98] Abnormalities of the right heart can also occur and may represent a combination of left heart disease, recurrent pulmonary thromboemboli, and OSA or hypoventilation or both.[99] Finally, lipomatous deposition in the interatrial septum has also been described[100]; however, this anatomic alteration is unlikely to contribute to cardiac dysfunction.

Weight loss, particularly in persons who are severely obese, can improve cardiac structure and function.[3,101] Improvements in fractional shortening are associated with decreases in hypertension and left ventricular internal dimension with reduced atrial and left ventricular free and septal wall thickness. Moreover, improvements in left ventricular diastolic filling and ejection fraction also occur.[102] Improvements in left ventricular mass occur in both normotensive and hypertensive patients and are independent of the reduction in blood pressure.[103,104] In addition, adding exercise to a low-calorie diet (LCD) may produce greater benefits in cardiac structure[105,106]; however, these benefits are not consistent across all studies.[107,108] For example, substantial weight loss ($\approx$15% of baseline)[108] and modest weight loss plus physical training[109] did not have beneficial cardiac effects in obese adolescents. At present, the potential benefits of weight loss

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on cardiac function are not completely clear and require further study.

Clinical Efficacy of Obesity Therapies

The goals of obesity therapy include decreasing body fat to improve appearance, physical function, quality of life, and medical health. Although surgical removal of large amounts of subcutaneous adipose tissue (≤20% of total body fat mass) can improve a person's appearance, ability to ambulate, and quality of life, it does not improve the metabolic CHD risk factors associated with obesity[110]; it seems that fat loss induced by negative energy balance is necessary to achieve metabolic benefits. Current therapies available for weight management that cause weight loss by inducing a negative energy balance include dietary intervention, physical activity, pharmacotherapy, and surgery. Behavior modification to enhance dietary and activity compliance is an important component of all of these treatments.

Dietary Intervention

Many different diets have been proposed for the treatment of obesity. These dietary approaches vary in their total energy prescription, macronutrient (fat, carbohydrate, and protein) content, energy density, glycemic index, and portion control. The energy content of a diet is the primary determinant of weight loss. Very-low-calorie diets (VLCDs) provide <800 kcal/d, LCDs usually contain 800 to 1500 kcal/d, and a balanced-deficit diet usually provides ≥1500 kcal/d. An LCD usually causes an ≈8% loss of body weight at ≈6 months of treatment. The results from clinical trials may not reflect the experience in clinical practice because these trials involved subjects who volunteered for a weight loss study and often included formal behavior modification as part of the study protocol. The use of a VLCD usually produces a weight loss of ≈15% to 20% within 4 months[111–113]; however, VLCDs are associated with poorer weight loss maintenance and a greater weight regain than are LCDs, so weight loss at 1 year after treatment with a VLCD does not differ from treatment with an LCD.[113] In addition, treatment with a VLCD may be particularly problematic for patients with CHD because of the risk of diet-induced hypokalemia, dehydration, and gallstones.

The macronutrient composition of a diet does not affect the rate of weight loss unless macronutrient manipulation influences total energy intake or expenditure. The Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults convened by the National Institutes of Health/National Heart, Lung, and Blood Institute recommended a 500- to 1000-kcal/d deficit diet for obese persons, which will initially result in a weekly weight loss of 1 to 2 lb (0.45 to 0.9 kg). It is often difficult, however, to accurately determine a patient's daily energy requirements. Therefore, calorie-intake guidelines for a weight-loss diet have been suggested based on a patient's initial body weight (Table 1).[114] The calorie content of any prescribed diet must be adjusted regularly, based on the patient's weight-loss response and treatment goals.

A low-fat diet is considered the standard approach for the treatment of obesity.[1] Data from diet intervention studies support the notion that decreasing fat intake, even while allowing ad libitum intake of carbohydrates and proteins, causes a spontaneous decrease in total energy intake and weight loss.[115] In addition, a survey of obese persons who were successful at maintaining long-term weight loss found that they consumed <25% of calories from fats.[116] However, a recent systematic review of randomized controlled studies that were specifically conducted to evaluate dietary therapy for obesity found that weight loss induced by low-fat diets and other weight-reducing diets were similar.[117] The composite of these data suggests that low-fat diets can enhance weight loss and may be particularly useful in selected persons, but they are not necessarily more effective than LCDs.

TABLE 1. Suggested Energy and Macronutrient Composition of Initial Reduced-Calorie Diet

Body Weight, lb	Suggested Energy Intake, kcal/d
150–199	1000
200–249	1200
250–299	1500
300–349	1800
≥350	2000

The use of low-carbohydrate diets has become increasingly popular. Several RCTs compared the effect of low-carbohydrate, high-protein, high-fat diets (eg, the Atkins diet) with a conventional low-fat diet (≈30% energy from fats) in adults[118–123] or a very-low-fat diet (≈12% energy from fats) in adolescents.[124] In all studies, weight loss at 3 and 6 months in subjects randomized to the low-carbohydrate diet was ≈2 times as great (≈4- to 5-kg greater weight loss) as those randomized to the low-fat group. In 2 studies that observed patients for 1 year, weight loss at 1 year was not significantly different between groups, however.[121,122] In general, these studies also found the low-carbohydrate diet was more beneficial in serum triglyceride and HDL-C concentrations as compared with the low-fat diet, but the low-fat diet was more beneficial in serum LDL-C concentration. Although these changes in triglycerides and HDL-C after weight reduction on low-carbohydrate diets appear favorable, it is not known whether these alterations are associated with long-term beneficial effects on CHD.[125]

The type of carbohydrate consumed also may be involved in regulating energy intake, and a low glycemic index diet has been proposed as a treatment for obesity. The glycemic index refers to the increase in blood glucose that occurs after consuming a fixed amount (usually 50 g) of available carbohydrate from a test food relative to the increase in blood glucose that occurs after consuming the same amount of available carbohydrate from either glucose or white bread.[126,127] Most refined grain products and potatoes have a high glycemic index, whereas most fruits, legumes, and nonstarchy vegetables have a low glycemic index. The glycemic response to a specific food that is ingested as part of a meal can be altered by many factors, such as the method of preparation and the effect of concomitantly ingested foods on

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intestinal motility. Data from a small (n=14) randomized controlled 1-year trial conducted in overweight adolescents revealed that a reduced glycemic index diet resulted in a greater decrease in body weight and BMI than did a reduced-fat diet.[128] The writing group is unaware of any RCTs evaluating the effect of a low glycemic index diet on body weight in adults.

The use of low-energy-density foods may be another effective approach for treating obesity. The energy density of a diet is defined as the calories present in a given weight of food. A food's energy density is directly correlated with its fat content and inversely correlated with its water content. Energy intake during a meal is partially regulated by the weight of ingested food and is inversely correlated with energy density.[129] Moreover, the results of a 6-month RCT demonstrated that providing subjects with ad libitum low-fat and low-energy-density foods causes modest (1% to 2%) weight loss.[130]

Portion control is an important aspect of reducing energy intake. During ad libitum feeding, a direct relationship is found between portion size served and intake; therefore, increasing the size of the portion served increases the amount of food consumed.[131]

Providing prepackaged prepared meals, either as frozen entrees of mixed foods or liquid-formula meal replacements improves portion control and can enhance weight loss. Data from RCTs have shown that obese persons who were given prepackaged prepared meals or liquid-formula meal replacements lost several kilograms more weight than did those who were randomized to a standard diet.[132–134] Educating patients about food labels, recipe modification, restaurant ordering, social eating, and healthy cooking methods are also important to help patients understand portion size and energy intake during meals and snacks.

In summary, the data from RCTs demonstrate that different dietary interventions can cause short-term weight loss. At the present time, we suggest that patients who are overweight or obese and trying to lose weight consume a diet that induces an energy deficit of 500 to 1000 kcal/d and has a macronutrient composition that is known to reduce the risk of CVD. This diet involves (1) consuming a variety of fruits, vegetables, grains, low-fat or nonfat dairy products, fish, legumes, poultry, and lean meats; (2) limiting intake of foods that are high in saturated fat, *trans*-fatty acids, and cholesterol; and (3) following the current dietary guidelines of the American Heart Association[135] and the NCEP ATP III[18] (Table 2). These recommendations may require modification, based on the results of ongoing and future dietary therapy studies. The key to successful weight management is to provide patients with a dietary regimen that results in long-term compliance. The available data suggest that it is unlikely that one approach is appropriate for all patients.

TABLE 2. Suggested Dietary Nutrient Composition for Patients Who Are Overweight or Obese

Nutrient	Recommended Intake
Saturated fat[1,2]	<7% of total calories
Monounsaturated fat	≤20% of total calories
Polyunsaturated fat	≤10% of total calories
Total fat	25% to 35% or less of total calories
Carbohydrate[3]	50% to 60% or more of total calories (complex carbohydrates from a variety of vegetables, fruits, whole grains)
Fiber	20–30 g/d
Protein	≈15% of total calories
Cholesterol	<200 mg/d

Physical Activity

Regular physical activity has important health benefits. A consensus public health recommendation for physical activity developed in the mid-1990s proposed that sedentary adults should accumulate ≥30 minutes of at least moderate-intensity physical activity (eg, brisk walking) on most but preferably all days of the week.[136–138] The health benefits of 30 minutes of daily moderate-intensity physical activity apply to all persons. Data from several studies show that persons who are overweight or obese and physically active (ie, participate in ≥30 minutes of moderate-intensity physical activity most days of the week) or who have moderate to high levels of cardiorespiratory fitness (ie, in the upper four fifths of the age and sex fitness distributions) have much lower death rates from cardiovascular disease and all-cause mortality than people who are sedentary and unfit.[87,139–143] Therefore, regular physical activity may improve survival in persons who are overweight or obese, independent of weight loss.

Weight loss results from a negative energy balance, which can be achieved by decreasing energy intake, increasing energy expenditure, or both. It is usually much easier to induce a daily energy deficit by restricting energy intake than by increasing energy expenditure. The calories consumed during physical activity can be estimated as a function of a metabolic equivalent task (MET) score. One MET is the energy consumed during resting conditions, such as television viewing, and is equal to ≈1 kcal/kg of body weight per hour. Other activities such as carrying packages, doing housework or gardening (2 to 5 METs), walking at a pace of 3 to 4 mph (3 to 4 METs), and jogging (8 to 10 METs) consume greater amounts of energy. A person weighing 90 kg would need to walk briskly for 4 to 5 h/d to increase his or her energy expenditure above resting metabolic rate by an amount that is equivalent to reducing energy intake by 750 to 1000 kcal/d. Therefore, it is difficult to lose a substantial amount of weight through physical activity. A review of 19 studies with randomized designs showed that exercise plus diet caused a 0.1-kg/wk greater weight loss than did diet alone.[144] Weight loss induced by combining physical activity with diet decreases the loss of FFM that occurs when weight loss is induced by diet alone.[145]

Data from observational studies strongly support the notion that physical activity is critical for preventing weight regain.[145,146] Moreover, the available evidence suggests that a high volume of physical activity, 80 to 90 minutes of moderate-intensity activity such as walking or 35 minutes of vigorous activity such as jogging, is necessary to maintain weight loss.[145] The interpretation of the results from these

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studies is complicated because subjects who achieved successful long-term weight loss had chosen to be physically active and had not been randomized a priori to a high-volume physical activity program. Data from a recent prospective RCT revealed that high-volume physical activity did not completely prevent weight regain.[147] Nonetheless, weight regain after 6 months was smaller and total weight loss was greater at 12 and 18 months in obese subjects who were randomized to dietary and behavior therapy plus high-volume physical activity (2500 kcal of energy expenditure per week) than they were in persons randomized to dietary and behavior therapy plus conventional physical activity (1000 kcal of energy expenditure per week). Although it is in general difficult to achieve long-term adherence to an exercise program, several approaches have been used to enhance adoption and maintenance of physical activity. Behavior-intervention strategies originally developed for smoking cessation or dietary programs have been used to increase physical activity. One study showed comparable improvements over 24 months in activity, fitness, and CHD risk factors for participants who were randomly assigned to a traditionally structured gymnasium-based program or to a behaviorally based intervention.[148] Increased contact by mail or telephone also helps maintain long-term adherence to exercise.[149] Total exercise time during the course of a study is greater when daily exercise is divided into multiple short bouts (eg, 10-minute bouts 3 to 4 times per day) than one long bout (eg, 30- to 40-minute bout once per day)[150]; ie, multiple short bouts of exercise result in greater adherence to an exercise program. In addition, many patients may be more compliant with an exercise program conducted at home than at a health club because fewer barriers are found with home-based exercise, including costs and travel time. Developing a home-based walking program and using home exercise equipment such as a treadmill has been shown to improve exercise adherence and long-term weight loss.[151,152] Finally, exercise does not need to be a structured activity. Altering daily lifestyle activities (eg, walking instead of riding, using stairs instead of escalators/elevators) may make it easier to increase overall physical activity than would participation in programmed exercise. In one study, weight loss was similar after dietary therapy plus either lifestyle activity or programmed exercise, but a trend toward better maintenance of weight loss 1 year after treatment was observed in individuals randomized to lifestyle activity than to programmed exercise.[153] Although these strategies are a welcome improvement, all studies still report a decline in exercise adherence over time.[148,149,151,154]

In summary, physical activity is not an effective approach for achieving initial weight loss, but it does have beneficial effects on fitness and obesity-related complications such as CHD and diabetes. In addition, a high level of regular physical activity is important for preventing and attenuating weight regain after diet-induced weight loss. Most data suggest that it is the total volume of physical activity that is important to weight management and that it does not matter whether the activity is of moderate or vigorous intensity, a lifestyle or structured program, or taken in a single bout each day or in several intermittent bouts.

TABLE 3. Behavioral Strategies to Improve Weight Management

Strategy	Description
Self-monitoring	Record "what, where, and when" of eating and physical activity to increase patients' awareness of their own behavior.
Goal setting	Set specific short-term targets in eating and exercise habits to achieve incremental improvements.
Stimulus control	Identify triggers associated with poor eating and physical activity behaviors, and design strategies to break link.
Cognitive restructuring	Change perceptions, thoughts, or beliefs undermining weight control efforts, and help patients develop realistic expectations about weight loss.
Problem solving	Analyze situations preventing maintenance of a healthier lifestyle and identify possible solutions to problems; maintain philosophy that planning, not willpower, is key to weight management.
Relapse prevention	Develop skills based on premise that lapses in weight control behavior can be anticipated in certain situations (eg, travel, celebrations, bad mood).
Stress management	Decrease psychological stress to prevent dysfunctional eating.
Contingency management	Use rewards (tangible or verbal) to increase performance of specific behaviors or when specified goals reached.
Social support	Use assistance from family members and friends in modifying lifestyle behaviors.
Ongoing contact	Maintain visits, telephone calls, or Internet communication with physician and office staff or other healthcare professionals to promote adherence with recommended lifestyle changes.

Behavior Modification

Behavior therapy focuses on analyzing and modifying eating and activity behaviors that increase body weight and provides techniques to help patients change their lifestyle habits and overcome barriers to compliance. A summary of behavioral strategies for treating obesity is shown in Table 3. The most important principles of behavioral treatment are that it (1) is goal-oriented and specifies goals that can be easily attained and measured, (2) is process-oriented and helps patients develop realistic goals and a reasonable plan for reaching those goals, and (3) involves making small rather than large changes so that incremental steps are taken to achieve larger and more distant goals.[155,156]

Self-monitoring, the systematic observation and recording of target behaviors, is the cornerstone of behavioral treatment.[156] Self-monitoring tools include (1) food diaries in which to record food intake, including types, amounts, energy contents, and times, places, and feelings associated with eating (usually in paper-and-pencil format but also available on the Internet or in commercially available programs for use on a personal digital assistant), (2) physical activity logs in which to record the frequency, duration, and intensity of exercise or step counters on which to monitor the daily steps

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taken, and (3) weight scales on which to measure changes in body weight. Self-monitoring increases patients' awareness of their behaviors, generates records that can be reviewed by healthcare professionals, and provides targets for intervention.

In clinical practice, formal behavior therapy can be provided through group sessions or individual meetings with a healthcare professional who is skilled in the delivery of behavioral techniques used to modify lifestyle habits.[155,157] If possible, contact should be regular, preferably once every 1 to 2 weeks, during the initial 6-month phase of a treatment program.[155] Comprehensive group behavior therapy, in conjunction with diet and physical activity, usually results in an ≈9% body weight loss within 26 weeks of treatment (≈0.5 kg/week).[157] Patients usually regain ≈33% of their lost weight in the year after ending behavior therapy, but most still maintain a weight loss of ≥5% at the end of 1 year. Providing ongoing contact by scheduled visits, telephone calls, food evaluation and exercise diaries, and Internet communication can enhance long-term adherence and helps prevent weight regain.[158,159] In addition, Internet-based treatment programs for weight loss[160,161] and structured commercial programs such as Weight Watchers[162] can augment the professional guidance provided by the physician.

Pharmacotherapy

Pharmacotherapy can help selected patients lose weight. The approved indications for drug therapy for obesity are a BMI ≥30 kg/m^2 or a BMI between 27 and 29.9 kg/m^2 in conjunction with an obesity-related medical complication in patients with no contraindications for therapy. Effective pharmacotherapy for obesity is likely to require long-term, if not lifelong, treatment because patients who respond to drug therapy usually regain weight when the therapy is stopped. The expected length of drug treatment of obese patients who respond to therapy makes it important to carefully consider the long-term risks of being obese, the beneficial effects of pharmacotherapy on body weight and obesity-associated diseases, and the side effects and costs of treatment before beginning therapy. In addition, pharmacotherapy alone is not as effective as pharmacotherapy given in conjunction with a comprehensive weight-management program.[163] Therefore, patients given drug treatment without the other standard approaches to weight management, including behavior modification, diet education, and activity counseling, are exposed to all of the risks of drug treatment without all of the medical benefits.

Drug therapy adds a level of complexity to the treatment of obesity. The patient with medication prescribed for obesity may have comorbidities that already require pharmacotherapy, thereby increasing the likelihood of nonadherence.[164] Strategies to enhance medication compliance include regularly assessing adherence and response to therapy, counseling about and reinforcing the importance of adherence, simplifying the treatment regimen, assisting the patient in reducing barriers to adherence, providing reminders and cues to facilitate improved adherence, and enlisting support when needed.[159,164–166] In addition, weight loss drugs usually are not covered by health insurance or health care plans, so a considerable economic incentive exists for the obese patient to discontinue taking these medications.

TABLE 4. Drugs Approved by FDA for Treating Obesity

Generic Name	DEA Schedule
Orlistat	None
Sibutramine hydrochloride	IV
Phentermine	IV
Diethylpropion hydrochloride	IV
Benzphetamine hydrochloride	III
Phendimetrazine tartrate	III

DEA indicates Drug Enforcement Agency.

Medications for the treatment of obesity available in the United States are listed in Table 4. Effective therapy for obesity usually requires chronic intervention; however, only 2 drugs, sibutramine and orlistat, are approved for long-term use.

Sibutramine

Pharmacology

Sibutramine is a β-phenethylamine derivative that blocks the reuptake of norepinephrine, sibutramine, and, to a lesser degree, dopamine. Sibutramine decreases food intake by producing early satiety during feeding and by delaying initiation of the next meal. Although sibutramine has no potential for abuse, it is classified as a Schedule IV drug. Sibutramine is available in 5-, 10-, and 15-mg doses; 10 mg/d as a single daily dose is the recommended starting level, with titration up or down based on response. Doses >15 mg/d are not recommended.

Clinical Efficacy

In a 1-year RCT, subjects treated with sibutramine lost 7% of their initial body weight and those treated with placebo lost 2%. Of the subjects treated with sibutramine or placebo, 57% and 20%, respectively, lost ≥5% of their initial body weight; 34% and 7%, respectively, lost ≥10% of their initial body weight.[167] Weight loss with intermittent sibutramine therapy (15 mg/d given during weeks 1 through 12, 19 through 30, and 37 through 48, and placebo given during the two 6-week periods when sibutramine was withdrawn) was equivalent to weight loss with continuous sibutramine therapy (15 mg/d).[168] Sibutramine therapy also has been shown to maintain weight loss for 12 to 18 months in subjects who initially lost weight by eating a VLCD[163] or who successfully lost weight after 6 months of sibutramine treatment.[170] The use of sibutramine in obese patients with either medication-controlled hypertension[171] or type 2 diabetes mellitus[172] causes greater weight loss than with placebo therapy, but the overall weight loss is less than that observed in studies conducted in subjects who do not have comorbid disease.

Weight loss with sibutramine therapy is more effective when combined with behavior and dietary therapies. In a 1-year RCT, weight loss with sibutramine therapy alone was ≈5 kg, with sibutramine therapy plus behavior modification was ≈10 kg, and with sibutramine therapy plus behavior modification and a structured meal plan was ≈15 kg.[173]

Side Effects and Safety

The most common side effects of sibutramine are dry mouth, constipation, and insomnia. Sibutramine increases

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heart rate (a dose of 10 to 15 mg/d causes an increase in heart rate of 4 to 6 bpm) usually in the first few weeks of treatment and lasts as long as the drug is taken. Sibutramine also causes a dose-related increase in blood pressure (a dose of 10 to 15 mg/d causes an average increase in systolic and diastolic blood pressure of 2 to 4 mm Hg) and can prevent weight loss–induced decrease in blood pressure.[155] Therefore, careful monitoring is needed when combining sibutramine with other drugs that can increase blood pressure. Sibutramine should not be used in patients who have uncontrolled hypertension, a history of coronary artery disease, congestive heart failure, cardiac arrhythmias, or stroke, or who are being treated with monoamine oxidase inhibitors or selective serotonin reuptake inhibitors.

CVD Risk Factors

The composite data from RCTs demonstrate that sibutramine causes improvements in serum triglyceride, total cholesterol, LDL-C, and HDL-C concentrations that are directly related to the magnitude of the weight loss. However, sibutramine therapy decreases or eliminates weight loss–induced benefits on blood pressure.

Orlistat

Pharmacology

Orlistat blocks the digestion and absorption of dietary fat by binding to intestinal lipases.[174] The percentage of fat that is malabsorbed is related to drug dose in a curvilinear fashion.[175] Near-maximal fat malabsorption occurs at a dose of 120 mg when given with a meal, which causes malabsorption of ≈30% of fat ingested from a meal that contains ≈30% of energy as fat. Less than 1% of ingested orlistat is absorbed; therefore, it has no effect on systemic lipases.[176]

Clinical Efficacy

The effects of orlistat on body weight and CHD risk factors have been evaluated in a large number of RCTs. The data from most studies demonstrate that at 1 year, subjects who were randomized to orlistat therapy (120 mg tid) lost ≈8% to 10% of their initial body weight and those randomized to placebo therapy lost ≈4% to 6%.[177–181] Approximately 33% more patients treated with orlistat lost ≥5% of their body weight than did those treated with placebo; ≈2 times as many patients treated with orlistat lost ≥10% of their body weight as did those treated with placebo. Ending orlistat therapy results in weight regain,[177,180] and starting orlistat therapy after successful diet-induced weight loss helps maintain body weight.[182] In subjects with obesity and type 2 diabetes mellitus who are treated with sulfonylureas,[183] metformin,[184] or insulin,[185] the percentage who achieve a ≥5% or ≥10% reduction in body weight is 2 to 3 times higher in those receiving orlistat plus dietary therapy than it is in those receiving dietary therapy alone. The overall weight loss effect of orlistat therapy in patients with diabetes is less than that reported in previous studies of obese patients who did not have diabetes, however.

Recently, the results of a 4-year RCT were reported.[28] The lowest body weight was achieved during the first year and was greater in the orlistat-treated group (11% weight loss) than in the placebo-treated group (6% weight loss). Subjects regained weight during the remainder of the trial; orlistat-treated subjects had lost 6.9% of their initial body weight and placebo-treated subjects had lost 4.1% at the end of 4 years. Orlistat therapy also decreased the cumulative 4-year incidence of type 2 diabetes mellitus by 37%.

Side Effects and Safety

About 70% to 80% of subjects treated with orlistat experienced ≥1 gastrointestinal event as compared with ≈50% to 60% of those treated with placebo. Gastrointestinal events usually occurred early (within the first 4 weeks), were of mild or moderate intensity, were usually limited to 1 or 2 episodes, and resolved despite continued orlistat treatment. Approximately 4% of subjects treated with orlistat and 1% of subjects treated with placebo withdrew from the studies because of gastrointestinal complaints. During treatment, small decreases in plasma fat-soluble vitamins, particularly vitamins A, D, and E, can occur, although plasma concentrations almost always remain within the reference range. A few patients, however, may experience decreases in plasma vitamin concentrations to below the reference range. Because it is impossible to determine a priori which patients will need vitamin supplements, it is recommended that all patients who are treated with orlistat be given a daily multivitamin supplement that is taken at a time when orlistat is not being ingested.

Orlistat can have medically significant effects on the absorption of lipophilic medications if both drugs are taken simultaneously. Subtherapeutic plasma cyclosporin levels that occurred in organ transplant recipients after they began orlistat therapy for obesity have been reported.[186–189] Therefore, orlistat should not be taken for ≥2 hours before or after the ingestion of lipophilic drugs, and plasma drug concentrations should be followed to ensure appropriate dosing. Orlistat does not affect the absorption of selected drugs with a narrow therapeutic index (warfarin, digoxin, phenytoin) and selected drugs that are likely to be taken concomitantly with orlistat (glyburide, oral contraceptives, furosemide, captopril, nifedipine, and atenolol).[189]

CVD Risk Factors

Because of its weight loss effects, orlistat therapy improves all major cardiovascular disease risk factors such as blood pressure and insulin sensitivity. Moreover, data from several RCTs suggest that orlistat has a beneficial effect on serum cholesterol concentrations that is independent of weight loss alone. Subjects given orlistat had a greater reduction in serum LDL-C concentrations than those given placebo, even after adjusting for percentage of weight loss.[178,179] The mechanism responsible for this additional lipid-lowering effect may be related to the effect of orlistat in blocking both dietary cholesterol and triglyceride absorption.[190] In contrast, orlistat is not as effective in lowering serum triglyceride concentrations, presumably because it increases the proportion of absorbed energy derived from carbohydrate, which tends to increase serum triglycerides.[191]

Phentermine

Phentermine is a β-phenethylamine derivative that stimulates the release of norepinephrine and dopamine from nerve terminals. Although phentermine is not approved by the Food and Drug Administration (FDA) for long-term use, it is the most commonly prescribed anorexiant medication in the United States,[192] presumably because it is less expensive than sibutramine. Phentermine was approved by the FDA >30 years ago, when the criteria needed for approval were less rigorous than they are currently. Therefore, fewer studies have evaluated the efficacy and safety

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of phentermine therapy than have evaluated sibutramine and orlistat. Only one long-term (36 weeks) RCT evaluated the effect of phentermine therapy on body weight.[193] In that study, obese women were randomized to dietary therapy and treatment with daily phentermine, daily phentermine every other month alternating with daily placebo every other month, or daily placebo. Of the 108 enrolled subjects, approximately two thirds completed the study; among those who completed the study, the groups that received either continuous or intermittent phentermine therapy lost ≈13% of their initial weight as compared with a 5% weight loss in the placebo group.

Side Effects and Safety

The most common side effects of phentermine are dry mouth, insomnia, and constipation. Although all sympathomimetic agents can increase blood pressure and heart rate, these side effects are uncommon when weight loss is adequate.

Herbal Products

Several different dietary supplements and herbal preparations have been used to treat obesity, including chromium picolinate, garcinia cambogia as a source of hydroxycitrate, chitosan that is claimed to reduce fat absorption, phenylephrine from *Citrus aurantium* (bitter orange), and ma huang as a source of ephedra alkaloids with or without guarana as a source of caffeine. In general, few RCTs have evaluated the clinical efficacy of these agents, and most of the RCTs that have been done were of substandard quality[194–196]; however, data from several RCTs demonstrated greater weight loss in subjects given herbal products that contain ephedra than in those given placebo.[197,198] Nonetheless, the sale of ephedra in over-the-counter products was recently banned by the FDA because of concerns about serious adverse cardiovascular effects.

Bariatric Surgery

Bariatric surgery is the most effective therapy available for people who are extremely obese. The current indications for surgical therapy were established at a consensus conference held at the National Institutes of Health in 1991.[199] The panel recommended that bariatric surgery be considered for obese persons who have a BMI of 35.0 to 39.9 kg/m^2 plus ≥1 severe obesity-related medical complication such as hypertension, type 2 diabetes mellitus, heart failure, or OSA and persons with a BMI ≥40 kg/m^2. At present, ≈109 000 bariatric surgery procedures are performed each year in the United States.

Five surgical procedures are most commonly used to treat obesity: (1) gastric bypass (Roux-en-Y anastomosis), (2) gastroplasty (gastric stapling, vertical banded gastroplasty, silastic ring gastroplasty), (3) gastric banding (LAP-BAND), (4) biliopancreatic diversion (partial biliopancreatic bypass), and (5) biliopancreatic diversion with duodenal switch (partial biliopancreatic bypass with duodenal switch). Gastric bypass accounts for ≈70% of the bariatric operations performed in the United States. This procedure involves the construction of a small (≈20 mL) proximal gastric pouch, which empties into a segment of jejunum that is anastomosed to the pouch as a Roux-en-Y limb. Gastroplasty involves the formation of a small pouch along the lesser curvature near the gastroesophageal junction, which empties into the rest of the stomach through a 1-cm outlet stoma. Gastric banding involves the placement of a band around the upper stomach. The band circumference size can be changed by percutaneously inflating or deflating a balloon in the band that is connected to a subcutaneous port and is commonly adjusted after surgery based on weight loss response and gastrointestinal symptoms. Biliopancreatic diversion involves the creation of a 200- to 500-mL proximal gastric pouch and transsection of the small intestine 250 cm from the ileocecal valve; the distal end of the small intestine is anastomosed to the gastric pouch and the proximal limb anastomosed to the ileum 50 cm from the ileocecal valve. These anastomoses create a 200-cm "alimentary tract," a variable length (300 to 500 cm) "biliary tract," and a 50-cm "common tract" in which digestion and absorption of ingested food occur. The biliopancreatic diversion with duodenal switch procedure involves the removal of ≈60% of the greater curvature of the stomach and transection of the proximal duodenum. The proximal portion of the duodenum is anastomosed end-to-end to the distal small intestine 250 cm proximal to the ileocecal valve. The distal end of the resected proximal intestine, which receives secreted pancreatic enzymes, is anastomosed to the ileum 100 cm proximal to the ileocecal valve. All bariatric surgical procedures have been performed as open and laparoscopic procedures.

TABLE 5. Effect of Different Bariatric Surgical Procedures on Long-Term (≥2 y) Body Weight

Procedure	Approximate Loss of Initial Weight, %	Approximate Loss of Excess Weight, %
Gastric banding	20–35	35–70
Gastroplasty	20–25	40–50
Gastric bypass	25–30	50–65
Biliopancreatic diversion ± duodenal switch	35–40	70–80

The approximate weight loss reported after each procedure is shown in Table 5.[114] It is difficult to determine the relative weight loss effectiveness of each procedure because only vertical banded gastroplasty and gastric bypass have been compared directly in RCTs.[200–203] The data from these RCTs consistently revealed that weight loss was greater with the gastric bypass procedure than with vertical banded gastroplasty. Fewer studies have evaluated the long-term effects of gastric banding, biliopancreatic diversion, and biliopancreatic diversion with duodenal switch than gastric bypass or gastroplasty because the procedure has been more recently developed or has been performed less often.

The perioperative mortality rate within 30 days after open bariatric surgery is ≈1%[23,200,204,205] but can vary depending on the experience of the surgeon.[206] Approximately 75% of deaths are caused by anastomotic leaks and peritonitis and 25% by pulmonary embolism. Laparoscopic gastric bypass is associated with fewer wound complica-

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TABLE 6. Weight Classification by BMI*

	Obesity Class	BMI, kg/m^2	Disease Risk
Underweight		<18.5	Increased
Normal		18.5–24.9	Normal
Overweight		25.0–29.9	Increased
Obesity	I	30.0–34.9	High
	II	35.0–39.9	Very high
Extreme Obesity	III	≥40.0	Extremely high

*Data from *Obes Res*.[1]

Additional adiposity-related risk factors: waist circumference >40 (in men) and >35 (in women); weight gain of ≥5 kg since age 18–20 y.

tions, less postoperative pain, less blood loss, and shorter hospital stays and convalescence periods than does the open procedure[207]; however, late anastomotic strictures occur more frequently after the laparoscopic than after the open procedure.

Treatment Guidelines

The goal of weight loss therapy for patients with CVD is to reduce or eliminate CHD risk factors and improve cardiac function. Aggressive weight loss therapy could be harmful in selected patients, such as those who have had a recent myocardial infarction or stroke or who have unstable angina, and attempts at weight loss should be delayed until these patients are medically stable.

Clinical Evaluation

The physician's office should be an environment that is sensitive to the needs of obese patients. The waiting room should contain chairs without arms, large gowns and large blood pressure cuffs should be available, and a scale that can weigh patients who weigh >300 lb should be available and located in a private area. The initial assessment should include an appropriate history, physical examination, and laboratory tests.

History

In addition to a standard medical interview, a patient's history should include an assessment of (1) weight history (highest and lowest adult body weight, previous weight loss attempts, weight pattern, and potential triggers and social and environmental factors that contributed to weight gain), (2) dietary history, including an assessment of types and timing of meals and snacks and an attempt to identify possible triggers that result in excessive energy intake, (3) physical activity and function (daily and exercise activities, physical limitations, effect of obesity on physical lifestyle), (4) obesity-related health risk (age of onset and duration of obesity, family history of obesity and obesity-related medical complications, current obesity-related disease), (5) possible psychiatric illnesses, such as binge eating disorder and depression, that may require therapy before a weight loss program is initiated, and (6) ability to lose weight (desire to lose weight, weight loss goals and expectations, limitations for achieving weight loss, including medications and illnesses, lifestyle and work patterns, financial resources, and special needs).

Physical Examination

The patient's BMI and waist circumference should be determined. BMI is generally correlated with percentage of body fat in a curvilinear fashion.[208] Some people with an "obese" BMI, who have a normal amount of body fat and a large muscle mass, are not at increased risk for CHD, whereas people with a "normal" BMI, who have excessive body fat and small muscle mass, are at increased risk. Waist circumference, measured halfway between the last rib and the iliac crest, correlates with abdominal fat mass.[5] Table 6 provides a classification of risk based on BMI. A waist circumference of ≥88 cm (35 in) for women and ≥102 cm (40 in) for men is associated with an increased risk of metabolic diseases and CHD.[1] Additional assessments should include measuring blood pressure with a large cuff and searching for physical signs of right or left ventricular dysfunction, congestive heart failure, and pulmonary disease. An electronic stethoscope can increase a physician's ability to detect cardiac abnormalities in patients who are extremely obese.

Laboratory Tests

An ECG is needed to check for evidence of CHD and to obtain a baseline tracing for future comparisons. Standard blood tests should be performed to search for CHD risk factors, including prediabetes (impaired fasting blood glucose or impaired glucose tolerance), dyslipidemia (increased triglycerides, low HDL-C, and increased LDL-C), and the metabolic syndrome. Additional studies may be needed to further evaluate specific clinical suspicions based on the history and physical examination, such as

TABLE 7. Weight Loss Treatment Guidelines*

	BMI Category, kg/m^2				
Treatment	25.0–26.9	27.0–29.9	30.0–34.9	35.0–39.9	≥40.0
Diet, physical activity, behavior therapy, or all 3	Yes	Yes	Yes	Yes	Yes
Pharmacotherapy†		With obesity-related disease	Yes	Yes	Yes
Surgery‡				With obesity-related disease	Yes

*Data from *Obes Res*.[1]

†Pharmacotherapy should be considered only in patients who are not able to achieve adequate weight loss by available conventional lifestyle modifications and who have no absolute contraindications for drug therapy.

‡Bariatric surgery should be considered only in patients who are unable to lose weight with available conventional therapy and who have no absolute contraindications for surgery.

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sleep studies to diagnose OHS or OSA and an exercise treadmill test or electron beam computerized tomography scanning or both to evaluate CHD risk. The comparative value of exercise tolerance testing and electron beam computerized tomography in obese subjects has not been determined. Exercise treadmill testing is not recommended for patients without cardiac symptoms, and neither exercise treadmill testing nor electron beam computerized tomography scanning should be performed in patients who are at low risk for CHD, based on clinical judgment or Framingham risk score.[209–211]

Therapeutic Options

Appropriate management requires identifying patients who need treatment, developing a realistic treatment plan, and implementing a defined treatment strategy that can be modified as needed during long-term surveillance. *The Practical Guide to the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults* was developed by the North American Association for the Study of Obesity in conjunction with the National Heart, Lung, and Blood Institute.[212] Suggested guidelines from the guide for selecting among different weight loss treatment options, based on disease risk, are shown in Table 7. A typical clinical consultation involves a physician's giving advice without adequate consideration of the patient's priorities, motivation, or confidence in undertaking change.[213] In contrast, obesity therapy should involve "patient-centered counseling," which encourages patients to set goals and express their own ideas for therapy, with input from the healthcare professional. The treatment plan also must take into account the patient's readiness for therapy and the patient's ability to comply with the proposed treatment plan. Realistic goals should be established and frequent follow-up visits should be scheduled to monitor progress, modify the treatment plan as needed, and provide encouragement. Effective therapy requires a long-term structured approach with continued support from the physician and other caregivers, particularly during periods of patient recidivism and weight regain.

Reducing energy intake is the cornerstone of weight management therapy. Providing appropriate nutrition counseling and the behavior modification therapy needed to implement dietary changes within the setting of a busy outpatient practice is difficult if not impossible for most physicians because they do not have the time or expertise to provide this kind of care. Therefore, referral to a reputable weight loss program or experienced dietitian should be considered, if these resources are available. Additional therapy with weight loss medications or bariatric surgery can be useful in properly selected patients.

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