

CLINICAL REVIEW

Diagnosis and Treatment of Obesity in Adults: An Applied Evidence-Based Review

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Background: Obesity is epidemic and leads to substantial morbidity/mortality. Effective strategies exist for managing obesity yet are rarely used by physicians. This applied evidence-based review provides a rationale for the diagnosis and treatment of obesity in adults by providing test characteristics for the body mass index (BMI) and number needed to treat (NNT) for relevant treatments.

Methods: We integrated evidence supporting recommendations from scientific bodies addressing obesity in adults, including: the National Heart, Lung, and Blood Institute, the World Health Organization, the Canadian Task Force on Preventive Health Care, and the US Preventive Task Force. In addition, pertinent studies were identified from MEDLINE, Database of Abstracts of Reviews of Effectiveness, and the Cochrane Database.

Results: (1) manage obesity as a chronic relapsing disease; (2) use BMI as a vital sign to screen for overweight/obese patients and to decide treatment (positive predictive value of 97%); (3) modest weight loss (10%) positively affects prevention/treatment of hypertension (NNT = 3), diabetes (NNT = 9), and hyperlipidemia; (4) effective treatments exist for overweight/obese patients and a combination of diet and exercise provides the best results (NNT = 7); (5) counsel patients to achieve a goal of 10% reduction in weight (500 to 800 kcal/day decrease to affect 1- to 2-pound loss/week); (6) counsel patients to exercise to achieve a goal of any increased energy expenditure.

Conclusions: Weight loss has an impact on important disease states and risk factors. Effective strategies exist for the management of obesity when viewed as a chronic relapsing disease. (J Am Board Fam Pract 2004;17:359–69.)

Obesity is epidemic world-wide,¹ and the United States is no exception.^{2–4} Initial results from the 1999 National Health and Nutrition Survey (NHANES) estimate that 61% of US adults are either overweight or obese; adult obesity nearly doubled, increasing from 15% to 27%, during the 14-year reporting period of NHANES III.⁵ Strong evidence links obesity to increased morbidity and mortality.^{1,6} Psychosocial consequences are substantial as well,^{7–10} including a limitation of capacity for physical activity.¹¹ Moreover, in the United States, the economic costs of obesity have been assessed at 6.8% of total health costs.¹² Although prospective studies of weight loss by obese persons have not demonstrated improvements in long-term

morbidity and mortality, reductions have been shown in risk factors for several cardiovascular, pulmonary, and cancer conditions.^{1,6,13–15}

Primary care physicians are in a special position to treat obesity. It is estimated that primary care doctors see 11.3% of the US population every month¹⁶ and that overweight patients are over-represented in this patient population.¹⁷ A number of scientific bodies have published treatment recommendations based on systematic reviews of the literature^{6,15,18–21} that could be used by primary care physicians. Although there are some differences of opinion, there is consensus on the treatment of obese patients with comorbid conditions (diabetes, hypertension, and hyperlipidemia) and partial agreement on treatment of all overweight and obese^{16,17} patients. However, these disparate treatment recommendations currently are not framed in such a way that they can be integrated easily into the other competing demands of primary care practice. Perhaps because of this, only 27% to 42% of obese patients seeking medical help

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are advised by their health professionals to lose weight.^{22,23}

This applied evidence-based review provides a rationale for the diagnosis and treatment of obesity in adults by providing test characteristics for the body mass index (BMI) and number needed to treat (NNT) for relevant treatments, thereby synthesizing the evidence and recommendations in a way that we feel will be useful to practicing clinicians. We believe that obesity should be approached as a chronic disease with genetic, environmental, and behavioral components.^{24,25} We acknowledge the public health aspect of obesity,¹ an epidemic requiring measures aimed at its social determinants,^{1,26,27} that are beyond the direct scope of the primary care encounter. Furthermore, we recognize that because of variation in physician style and patient characteristics, a "one size fits all" cookbook approach to the treatment of obesity will not be helpful. The purpose of this review is to provide a resource for primary care physicians that can be adapted for use in the way most appropriate for any individual practice.

Background

Evidence of Increased Health Risk Associated with Obesity

The health consequences of obesity are many.¹ Obesity is an independent risk factor for increased mortality. Overall mortality begins to increase with BMI levels greater than 25 (relative risk = 1.1) and increases most dramatically as BMI levels surpass 30 (relative risk = 1.5).²⁸ For example, a BMI of 35 increases mortality by a factor of 2.5.²⁸ For comparison, diastolic blood pressure values of 100 and 120 mm Hg increase mortality by factors of 2 and 5, respectively²⁹; cholesterol levels of 236 and 290 mg/dL increase mortality by factors of 1.8 and 4, respectively.³⁰ Finally, studies show that the longer the duration of obesity, the higher the risk.¹

Furthermore, obesity predisposes a person to a number of cardiovascular risk factors, including hypertension, elevated cholesterol and impaired glucose tolerance. Cardiovascular and other obesity-related disease risks, such as sleep apnea, osteoarthritis, increased anesthetic risk, and reproductive abnormalities, increase significantly when BMI exceeds 25.0.¹

Evidence that Reducing Weight Decreases Disease Risk

Unfortunately, most studies investigating weight loss and mortality have not controlled for unintentional weight loss or for smoking. Thus, longer term, well-controlled studies are needed to define accurately the benefits of weight loss on mortality; however, data from a number of studies have shown that modest weight loss (defined as a weight loss of up to 10% of body weight) improves glycemic control, reduces blood pressure, and reduces cholesterol levels.³¹ For example, each 1% reduction in body weight leads, on average, to a fall of 1 mm Hg systolic and 2 mm Hg diastolic pressure.³² This effect on blood pressure is independent of sodium restriction.³³ Low-density lipoprotein cholesterol has been estimated to decrease by 1% for every 2.2 lb lost.³⁴ There seems to be a critical threshold weight loss of $\geq 5\%$ of body weight for improved glycemic control.³⁵

Weight loss has beneficial effects on blood pressure, glucose, lipids, and psychological status.³⁶ In addition, other benefits include (1) improvement in quality of life,³⁷ (2) relief in symptoms of dyspnea and chest pain,³⁸ and (3) reduction in number of days of sick leave.³⁸

Although the above changes in physiologic and biochemical parameters are important for risk reduction, the results for diabetes and hypertension have practical outcomes that matter to patients. For example, it takes helping 9 patients with type 2 diabetes to lose at least 5% of their body weight to get 1 patient off oral hypoglycemic agents.³⁹ For hypertension, it takes helping 3 patients to lose 10 pounds of weight and maintain that weight loss for 4 years to discontinue antihypertensive medication for 1 patient.⁴⁰

Evidence that Reducing Weight Increases Disease Risk

Only 2 hazards have been documented in a variety of prospective studies involved in intentional and controlled weight loss. In the Nurses Health Study, the BMI-adjusted relative risk for cholecystectomy or unremoved gallstones was 1.97 (on average) for those losing 22 lb or more in the previous 2 years.²⁸ Bone density is typically increased in obese patients and reduced after weight loss. In white women, weight loss beginning at age 50 was found to increase the risk of hip fracture.⁴¹

It has been suggested that weight cycling is associated with negative health outcomes and makes future weight loss more difficult. However, the National Task Force on the Prevention and Treatment of Obesity concluded that the evidence available was not sufficiently compelling to override the potential benefits of moderate weight loss in obese patients.⁴²

Methods

This review is based on a synthesis of the evidence focused on the role of the primary care clinician in the diagnosis and treatment of adult overweight and obese patients. It integrates the evidence supporting the recommendations from a number of systematic reviews.^{6,18,19,21,43} Among these reviews are the more conservative Canadian Task Force recommendations,¹⁸ and the more aggressive National Heart, Lung, and Blood Institute (NHLBI) recommendations,⁶ which prepared the clinical guidelines in 1998 based on a systematic review of the literature from the years 1980 to 1997. In addition, this review incorporates pertinent additions based on 2 recent reports from the US Preventive Services Task Force^{15,44} and results from clinical trials, in particular those related to the drug treatment of obesity, that were completed after the above published recommendations.^{39,45–52}

The literature search and selection approach involved the following steps. The authors identified the major scientific bodies* that have made recommendations regarding the diagnosis and treatment of obesity by searching MEDLINE, Database of Abstracts of Reviews of Effectiveness, Cochrane Database of Systematic Reviews, and HealthSTAR. The search term obesity.mp [mp = title, original title, abstract, mesh headings, heading words, keyword] was used. In addition, the web sites of the Agency for Healthcare Research and Quality (AHRQ)⁵⁵ and the National Guideline Clearinghouse⁵⁶ were reviewed for obesity-related guidelines. The most conservative¹⁸ and most aggressive⁶ recommendations were identified. Primary articles referenced to support these 2 guidelines were selected, based on 2 criteria: (1) clinical trials whose results allowed for the calculation of NNT and number needed to harm (NNH), and (2) when possible, patient populations and practice settings

representative of primary care. In addition, papers reporting on the diagnostic test characteristics of BMI were selected. Finally, a MEDLINE search was performed for studies reported after those reported in systematic reviews used by the major scientific bodies. This search used the term obesity.mp and went up to December 2003.

Results

Diagnosis of Obesity

Because normal weight in adults varies with sex, height, and age, the criteria for obesity differ with respect to these variables. Instead of using complicated sex-, height-, and age-specific tables of criteria for obesity, BMI (kg/m^2) is an easily obtained and relatively reliable measurement for overweight and obesity. If weight is measured in pounds and height in inches, the BMI is calculated as $[\text{weight}/\text{height}^2] \times 703$. This index gives body mass corrected for height for a wide range of heights and is a good approximate estimate of the fat content of the body. The issue of whether indices of overweight predict body composition is important because body composition (ie, adiposity) rather than excess body mass is the important health risk.⁵⁷

The current diagnostic criteria of obesity for adults are based on epidemiologic data and are set at a round number of $\text{BMI} = 30 \text{ kg}/\text{m}^2$.¹ Because morbidity and mortality increase gradually with excess of BMI, it is a common practice to set not only diagnostic criteria for obesity but also for 'overweight,' ie, 25 to $29.9 \text{ kg}/\text{m}^2$.

Despite variations in criteria used to define obesity and the age, body constitution, gender, weight, and race/ethnic characteristics of the test subjects, the findings from studies looking at test characteristics of the BMI have several implications relevant to the clinician.^{58–60} First, compared against a number of standard tests for estimating fat content of the body, BMI has extremely high specificity (98% to 99%). This high specificity, coupled with 43% to 67% prevalence of patients with obesity in primary care clinicians' offices,^{17,61} yields a positive predictive value of 97%. Therefore, a patient with a positive test ($\text{BMI} \geq 30$) effectively rules in the diagnosis. On the other hand, studies have shown low sensitivities for BMI, ranging from 13% to 55%.^{58–60} Therefore, a patient with a negative test ($\text{BMI} < 30$) has a negative predictive value of only 68% and will tend to misclassify overweight pa-

*Refs. 1, 6, 15, 18, 19, 21, 43, 44, 53, and 54.

Table 1. Effectiveness of Weight Loss Treatments

Strength of Recommendation	Treatment	Comment
A	Dietary therapy	LCDs (1000 to 1200 kcal/day) can reduce total body weight by an average of 8% over 3 to 12 months Very-low-calorie diets produce greater initial weight loss than LCDs. However, the long term (>1 year) weight loss is not different from that of LCD
A	Aerobic physical activity	Exercise at 60% to 85% of estimated maximum heart rate over 3 to 7 30- to 60-min sessions per week produce a modest change (3 to 6 lb) at 1 year
A	Diet and physical activity	Combination results 3.3 to 6.6 lb greater weight loss over diet alone up to 2 years
B	Behavior therapy	9 lb over 4 years when used in combination with other weight loss approaches No one behavior therapy seemed superior
B	Pharmacotherapy	Part of comprehensive program of diet/exercise for BMI ≥ 30 or BMI ≥ 27 with obesity-related risk factors or disease. Produce modest change in weight (3 lb) at 6 months to 1 year
B	Surgery	BMI ≥ 40 or BMI ≥ 35 with obesity-related risk factors or disease and after less invasive methods have failed can produce weight loss of 90 lb at 1 year

LCD, low-calorie diet; BMI, body mass index.
Adapted from ref. 6.

tients as normal. However, as the BMI surpasses 30, the negative predictive value improves up to 84%.⁵⁸ Because the studies demonstrating increased risk associated with obesity use BMI as the “exposure” measurement,^{1,6,28} and studies show the major ill effects of weight appear in patients in the obese or BMI ≥ 30 ,⁶ the BMI, on balance, presents a good screening test⁶² in clinical practice.

Finally, because several studies have shown that not only the mass but also distribution of the body fat affects the magnitude of health hazards, some expert panels such as the NHLBI have recommended complementing the BMI with estimates of abdominal fat through measurement of waist circumference. However, the evidence for the existence of the increased risk and more importantly the evidence for impact on reducing the hazard with treatment focused on abdominal fat loss are consistently lower (grade C)⁶ than the evidence associated with BMI (grade A).⁶ Furthermore, because evidence exists that clinicians rarely address overweight and obese patients already,^{22,61,63} and given the competing demands of primary care practice, promoting measurement of the BMI alone seems the most practical strategy for diagnosis of obesity in primary care.

Evidence that Effective Treatments Exist for Obesity

Information on the effectiveness of different interventions for patients has become available only

relatively recently with the publication of systematic reviews.^{6,15,18,19,44,53,64} Although there are gaps in the evidence, the report by the NHLBI⁷ identified a number of potentially effective weight loss interventions: (1) diet; (2) exercise; (3) behavioral strategies; (4) the preceding 3 in combination where possible; (5) limited use of pharmaceutical interventions in conjunction with strategies to change lifestyle; and (6) surgery for selected morbidly obese patients. Recommendations also include the use of maintenance strategies such as continued therapist contact and prevention strategies such as screening and counseling. The NHLBI guidelines suggest that weight loss programs should aim initially to reduce body weight by 10% from baseline, at a rate of 1 to 2 lb a week, for 6 months. Subsequent strategies should be based on how much weight has been lost initially.

Table 1, adapted from the NHLBI,⁷ presents the various weight loss treatments organized in order of their strength of recommendation. A brief summary of the treatment is also provided to give some context. Category A requires substantial numbers of randomized controlled trials involving substantial numbers of participants. Category B is used when few RCTs exist, they are small in size, and the trial results are somewhat inconsistent or trials were undertaken in a population that differs from the target population of the recommendation.

Table 2 presents representative clinical trials of recommended weight loss treatments with the results expressed as NNT when available for calculation. Because the adequacy of data required to calculate the NNH was rarely available, this was included in the text below rather than in the table. The treatment outcome as an expression of weight loss varies because of the way the trials were designed. Below, we discuss in detail the 6 treatment recommendations found to be effective.

Diet Therapy

Current dietary recommendations continue to focus on the low-calorie, low-fat diet, with intake of 800 to 1500 kcal of energy per day. Caloric reduction in the range of 500 to 1000 kcal less than the usual intake is appropriate.⁶ This will allow for approximately 1 to 2 pounds of weight loss per week. Very-low-calorie diets, <800 kcal/day, have not been shown to be any more effective after 1 year, require close monitoring, and are not recommended.⁶ One problem with reviewing studies is the great overlap between pure dieting as diet instruction versus behavior therapy and dieting. The other problem is the intensity, frequency, and format of diet therapy. A recent review emphasizes counseling was most effective when intensive and combined with behavioral therapy.⁴⁴ Intensity related not only to the frequency of sessions (1 to 2/month) but also to the number of components (eg, diary, family involvement, social support, group counseling, goal setting, taste, cooking time) covered in the sessions.⁴⁴ Regardless, the literature is consistent that the goal of therapy does not have to be greater than a 10% loss of body weight to have measurable effects on outcomes. To successfully achieve a sustained decrease of 10 lb of body weight in 1 patient, 9 patients require dietary treatment (Table 2).

Exercise

Obesity is frequently associated with low physical fitness as a result of inactivity, and increases in energy expenditure can create a negative energy balance that leads to a reduction in body fat. Compared with diet alone, exercise alone would require treating 17 patients to successfully have 1 patient lose 10 lb (Table 2). One important point is that the goal of exercise need not be cardiovascular fitness, an outcome that often requires a level of intensity

Table 2. Clinical Trials of Effective Treatments for Weight Loss

Treatment	Reference Citation	Selection Criteria/ Sample Size	Duration	Design	Outcome	NNT
Dietary (nutritionists LCD)	34	155 men, age 30 to 59 120% to 160% of IBW	1 year	RCT	Decrease of 8 lb of fat weight (~10 lb total weight)	9
Exercise (treadmill 30 minutes ~3 to 5 days/week)	34	155 men, age 30 to 59 120% to 160% of IBW	1 year	RCT	Decrease of 8 lb of fat weight (~10 lb total weight)	17
Combination of Diet and Exercise	65	165 M/F, ≥ 31 lb overweight, ages 25 to 45	2 years	RCT	Decrease 10 lb of total weight	7
Behavior (psychologists)	78	59 M/F, community volunteers >55 lb overweight	1 year	RCT	Mean weight loss 21 lb at 1 year	Unable to Calculate
Drug sibutramine (and diet) vs diet only 1 year 10 and 15 mg	45	485 M/F, mean BMI 32.7, ages 27 to 40	1 year	RCT double-blind/placebo	Decrease 10% or more of body weight	8, 4
Drug orlistat 120 mg	39	399 M/F, age ≥ 18 , BMI 28 to 40, with DM	1 year	RCT double-blind/placebo	Decrease $\geq 5\%$ of body weight	4
Surgery gastroplasty	71	57 M/F ages, BMI versus VLCD	5 year	RCT	Maintained weight loss of ≥ 22 lb	8
Surgery gastric bypass/gastroplasty	72	310 M/F, ages 18 to 62, weight 160% to 318% $>$ IBW, gastric bypass/gastroplasty	3 years	RCT	Loss of $>50\%$ excess weight	2, 3

LCD, low-calorie diet; IBW, ideal body weight; BMI, body mass index; M/F, men and women; DM, diabetes mellitus; RCT, randomized controlled trial; VLCD, very-low-calorie diet; NNT, number needed to treat.

that overweight or obese patients may not be able to achieve. Any exercise will increase energy expenditure and consequently create some negative balance.

Diet and Physical Activity

The combination of diet and physical activity results in a greater weight loss compared with diet or exercise alone (NNT = 7; Table 2). There also seems to be some evidence that although diet alone will achieve greater weight loss, that physical activity alone is associated with maintaining weight loss better than diet alone.⁶⁵

Behavior Therapy

Behavior therapy usually consists of behavior modification directed toward diet and exercise. Behavior modification involves counseling the patient regarding stimulus control, goal setting, cognitive restructuring, self-monitoring, and contracts to reward behavior. As mentioned, it is difficult to separate out studies that are diet only or exercise only versus behavior modification associated with diet and exercise. The studies reporting the use of behavioral therapy usually involved regular ongoing contact with a professional other than a physician. The level of recommendation was grade B (Table 1). The lower grade of evidence was the result of fewer available RCTs. In addition, in those studies that were available, the comparison groups chosen did not allow a determination of the unique effect of behavioral therapy. Therefore, we were unable to determine the NNT. However, summary of evidence from more recent studies suggests that brief infrequent counseling by physicians (1 to 3 times/year) may be less effective than physician counseling plus weekly or bimonthly counseling from another para/professional (dietician, nurse counselor, commercial weight loss program).^{15,44}

Drug Therapy

Current accepted pharmacotherapy treatments include 2 main classes of drugs: (1) drugs to suppress appetite (eg, serotonin-norepinephrine re-uptake inhibitors, such as sibutramine hydrochloride) and (2) drugs to change metabolism (eg, gastrointestinal lipase inhibitor that prevents absorption from the gut, such as Orlistat). The evidence from the systematic reviews for both is level B. This is mainly because of the lack of long-term studies

(greater than 1 year), especially weighing benefits versus risks; however, additional studies not available at the time of the NHLBI report demonstrate greater weight loss effects^{39,45} and some observed the patients for up to 2 years.^{66,67}

Whereas older-generation noradrenergic appetite suppressants (eg, mazindol, diethylpropion, and phentermine) are approved for obesity treatment, obesity experts agree⁴⁶ that these schedule II and III drugs have no current appropriate role in obesity treatment given the availability of newer schedule IV anorectics (eg, sibutramine) with negligible addiction and abuse liabilities. Finally, herbal treatments such as garcinia cambogia, a common component of commercial weight-loss products, have not been found useful in the treatment of obesity.⁶⁸

Sibutramine. Because sibutramine's appetite suppression is completely reversed by adrenergic blockade, and pure serotonin reuptake inhibitors have been shown not to produce long-term weight loss, sibutramine's weight loss effects are primarily mediated by its noradrenergic action. Sibutramine is dispensed as Meridia; doses of 10 or 15 mg/day have been shown to optimize weight loss versus adverse effects. The study in Table 2 is a more recent one and shows NNT of 8 and 4 for 10- and 15-mg doses, respectively. Although the NNH attributable to dry mouth was found to be no different in either treatment dose (NNH = 7),⁴⁵ pulse rates were higher in the 15-mg group.

Despite a pending review by the FDA of a petition⁶⁹ to recall sibutramine from a consumer advocacy group because of deaths of patients on sibutramine, the FDA has only reiterated its initial labeling instructions. These instructions include monitoring patients closely for elevated blood pressure and strongly advising against the use of sibutramine in patients with hypertension, heart disease, arrhythmia, and in stroke survivors. In Europe, Italy temporarily suspended use of sibutramine because of 2 patient deaths. Both patients had other complicating conditions and had been taking other medications.

Orlistat. Orlistat's inhibition of pancreatic lipase is responsible for its therapeutic action of blocking the absorption of approximately 30% of ingested fat calories.⁷⁰ Orlistat is dispensed as Xenical and is given in a 120-mg dose thrice daily. Overall, in 2 of the largest and most well-designed trials of obesity pharmacotherapy so far, orlistat has been shown to

induce 1-year weight loss of 6.6 to 8.8 pounds in excess of placebo.^{48,66} Adverse effects (NNH = 5)³⁹ are limited to the gastrointestinal tract (flatus, oily spotting, fecal urgency and incontinence) because of low fat absorption. Adverse gastrointestinal events led to withdrawal of 9.0% and 3.5% of patients from the European and US studies, respectively.⁴⁶ Although 15 mg/day sibutramine and orlistat both had an NNT = 4, orlistat achieved only a 5% reduction in body weight versus 10% for sibutramine (Table 2).

These recent RCTs, coupled with the recent US Preventive Services Task Force report,¹⁵ therefore, strengthen the NHLBI guideline recommendation that drug therapy can be useful as part of a comprehensive weight loss program in addition to diet and exercise. It should be considered for those with

BMI ≥ 30 or BMI ≥ 27 with obesity-related risk factors or disease.⁶

Surgery

Bariatric or weight reduction surgery is usually considered for people with morbid obesity who are refractory to other weight-reduction interventions. Surgery should be confined to those with BMI ≥ 40 or BMI ≥ 35 with obesity-related risk factors or disease and when less invasive measures have failed. Bariatric surgery includes several techniques, each of which may vary depending on the surgeon or clinical center with this expertise. In general, currently acceptable surgical procedures fall into 2 categories: (1) gastric bypass, which involves complete partitioning with anastomosis of the proximal gastric segment to a jejunal loop; and, (2) gastro-

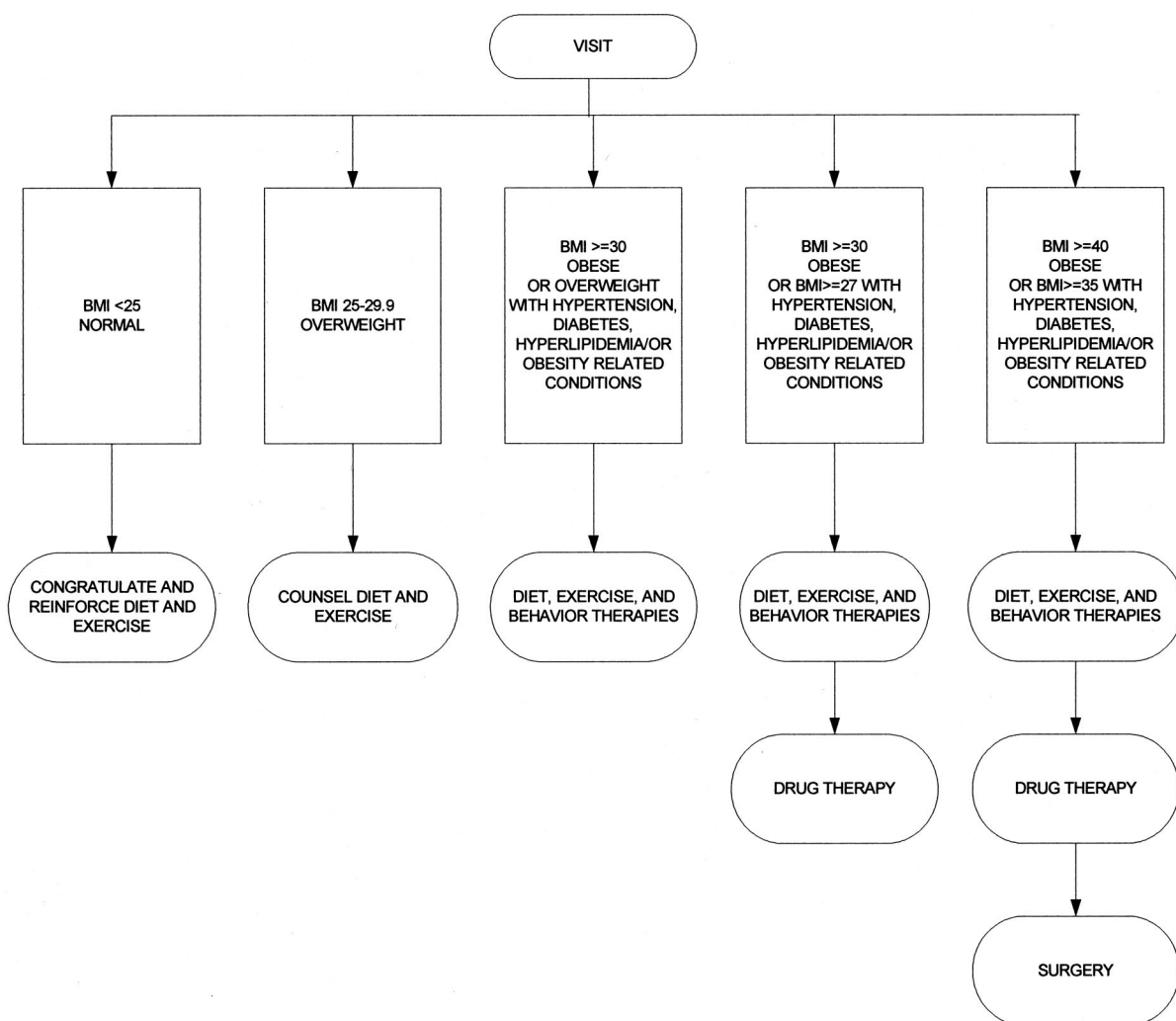


Figure 1. Treatment flow diagram for obese and overweight patients.

plasty, which involves partial partitioning at the proximal gastric segment with placement of a gastric outlet stoma of fixed diameter. Both methods are intended to create an upper gastric pouch that reduces gastric luminal capacity and causes early satiety. The newer laparoscopic procedures will not be discussed because of insufficient evidence by which to evaluate them.

In general, with a mean weight loss of 61 to 100 lb,⁶ the magnitude of weight loss with surgical therapy was greater than other interventions. Postoperative mortality was low at 1 surgery-related death in 5 studies (~650 patients).⁷¹⁻⁷⁵ Postoperative morbidity was usually a result of infection (wound-related, subphrenic abscess, pneumonia) or pulmonary complications (atelectasis, pulmonary edema) and occurred in less than 5% of patients across the studies. The need for reoperation because of either surgery-related complications (revision of procedure) or a complication related to weight loss (acute cholecystitis) varied from 1.7% to 33.3%.

Gastric bypass seemed to show greater weight loss than other treatments with NNT values of 2 and 3 for gastric bypass and gastroplasty, respectively (Table 2). The potential for adverse effects was substantial, however (NNH = 3 and 9 for gastric bypass and gastroplasty, respectively).^{71,72} A grade B recommendation was given because of the paucity of good surgical studies, many of which reported on surgical procedures that are no longer used (Table 1). Furthermore, although for surgery the NNH approaches that in drug therapy, the nature of the complications are far more serious.

Figure 1 provides a flow diagram for clinicians that outlines management of overweight and obese

patients based on the synthesis of the evidence presented in this review.

Discussion

It is useful to consider obesity as a chronic disease of multifactorial etiology that is a lifelong condition for most persons. In many, it is characterized by slow progression throughout adult life, whereas in others, it is characterized by periods of weight stability or short-term weight loss followed by relapse. There is a dose-response relationship between the degree of obesity and the risk of morbidity and mortality from cardiovascular disease and diabetes, among other conditions. Modest weight loss results in improvement in the prevention of hypertension, diabetes, and hyperlipidemia.

This chronic disease "biological" model has important implications for the treatment used. First, the condition must be brought to the clinician's and patient's attention. The BMI is a useful tool to establish a diagnosis in obese patients and to decide on treatment options. Just as in other chronic diseases, treatment must be maintained for life with lifestyle interventions combined, when necessary, with pharmaceutical therapy and, in selected patients, surgery. Finally, when the system resources are available, a chronic disease "care" model⁷⁶ that uses a collaborative team approach might provide opportunities to provide more intensive treatment and consistent reinforcement and follow-up.

Effective treatments exist for overweight and obese patients. Diet and exercise combined provide the best results. Patients should be encouraged to set a goal of 10% reduction in total body weight rather than struggle to attain ideal body weight. This can be accomplished by having the patient

Table 3. Key Points For Clinicians Managing Obese Patients

Manage obesity as a chronic relapsing disease.
Use BMI as a tool to establish a diagnosis in obese patients and to decide on treatment.
Modest weight loss (10% reduction in total body weight) results in improvement or prevention of hypertension, diabetes, and hyperlipidemia.
Effective treatments exist for overweight and obese patients. Diet and exercise combined provide the best results. Use sibutramine with caution pending FDA review.
Counsel patients to achieve a goal of 10% reduction in total body weight (500 to 800 kcal/day decrease to affect 1- to 2-lb weight loss/week) rather than attain an ideal body weight
Counsel patients to exercise to achieve increased energy expenditure rather than to attain aerobic fitness.
Consider referral to a behavioral program to leverage your office counseling.
Serve as advocates for social policies that promote good nutrition and increased physical activity.

BMI, body mass index.

sustain a 1- to 2-lb weight loss per week through a decrease of 500 to 800 kcal/day. Exercise should be encouraged for all overweight patients, including those physically challenged, by prescribing exercise designed to achieve increased energy expenditure rather than aerobic fitness. Sibutramine should be used with caution pending continuing FDA review.

Obesity provides both challenges and opportunities to the primary care clinician. Armed with a practical understanding of recent evidence, clinicians can implement an effective strategy in the management of their overweight and obese patients. The 8 key points in Table 3 provide a simple synopsis of the strategy.

Although we have concentrated on the role of the primary care clinician in the context of the clinical encounter, this is a reactive rather than a proactive strategy. Once patients are already overweight, sustained weight loss is difficult to achieve. Convincing epidemiologic data suggest that the increasing incidence of obesity is related to the availability and promotion of high calorie density foods, and decreasing physical activity as a result of the sedentary nature of work and recreation in Western industrialized societies.^{1,77} It is incumbent on primary care clinicians not only to treat obesity during office visits but also to serve as advocates for social policies that promote good nutrition and increased physical activity.

References

1. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organ Tech Rep Ser 2000;894:i-xii, 1-253.
2. Mokdad AH, Serdula MK, Dietz WH, Bowman BA, Marks JS, Koplan JP. The spread of the obesity epidemic in the United States, 1991-1998. *JAMA* 1999;282:1519-22.
3. Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL. Overweight and obesity in the United States: prevalence and trends, 1960-1994. *Int J Obes Relat Metab Disord* 1998;22:39-47.
4. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999-2000. *JAMA* 2002;288:1723-7.
5. National Center for Health Statistics. NHANES 1999 Prevalence of overweight and obesity among adults: US, 1999. Hyattsville (MD): US Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention; 1999.
6. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults—The Evidence Report. National Institutes of Health [published erratum appears in *Obes Res* 1998;6:464]. *Obes Res* 1998;6 Suppl 2:51S-209S.
7. Palinkas LA, Wingard DL, Barrett-Connor E. Depressive symptoms in overweight and obese older adults: a test of the "jolly fat" hypothesis. *J Psychosom Res* 1996;40:59-66.
8. Desmond SM, Price JH. Self-efficacy and weight control. *Health Educ* 1988;19:12-8.
9. Friedman MA, Brownell KD. Psychological correlates of obesity: moving to the next research generation. *Psychol Bull* 1995;117:3-20.
10. Hill AJ, Williams J. Psychological health in a non-clinical sample of obese women. *Int J Obes Relat Metab Disord* 1998;22:578-83.
11. Fontaine KR, Cheskin LJ, Barofsky I. Health-related quality of life in obese persons seeking treatment. *J Fam Pract* 1996;43:265-70.
12. Wolf AM, Colditz GA. The cost of obesity: the US perspective. *Pharmacoeconomics* 1994;5(Suppl): 34-7.
13. US Preventive Services Task Force. Guide to clinical preventive services: report of the US Preventive Services Task Force, 2nd ed. Baltimore: Williams & Wilkins; 1996.
14. Blackburn G. Effect of degree of weight loss on health benefits. *Obes Res* 1995;3 Suppl 2:211S-6S.
15. McTigue KM, Harris R, Hemphill B, et al. Screening and interventions for obesity in adults: summary of the evidence for the US Preventive Services Task Force. *Ann Intern Med* 2003;139:933-49.
16. Green LA, Fryer GE Jr, Yawn BP, Lanier D, Dovey SM. The ecology of medical care revisited. *N Engl J Med* 2001;344:2021-5.
17. Noel M, Hickner J, Ettenhofer T, Gauthier B. The high prevalence of obesity in Michigan primary care practices. An UPRNet study. Upper Peninsula Research Network. *J Fam Pract* 1998;47:39-43.
18. Douketis JD, Feightner JW, Attia J, Feldman WF. Periodic health examination, 1999 update: 1. Detection, prevention and treatment of obesity. Canadian Task Force on Preventive Health Care. *CMAJ* 1999; 160:513-25.
19. Glenny AM, O'Meara S, Melville A, Sheldon TA, Wilson C. The treatment and prevention of obesity: a systematic review of the literature. *Int J Obes Relat Metab Disord* 1997;21:715-37.
20. Harvey EL, Glenny AM, Kirk SF, Summerbell CD. An updated systematic review of interventions to improve health professionals' management of obesity. *Obes Rev* 2002;3:45-55.
21. Harvey EL, Glenny A, Kirk SF, Summerbell CD. Improving health professionals' management and the organisation of care for overweight and obese people. *Cochrane Database Syst Rev* 2001;(2): CD000984.
22. Heath C, Grant W, Marchetti P, Kamps C. Do

- family physicians treat obese patients? *Fam Med* 1993;25:401–2.
23. Galuska DA, Will JC, Serdula MK, Ford ES. Are health care professionals advising obese patients to lose weight? *JAMA* 1999;282:1576–8.
24. Hill JO. Dealing with obesity as a chronic disease. *Obes Res* 1998;6 Suppl 1:34S–8S.
25. Rippe JM, Crossley S, Ringer R. Obesity as a chronic disease: modern medical and lifestyle management. *J Am Diet Assoc* 1998;98(10 Suppl 2):S9–S15.
26. Ebrahim S, Smith GD. Systematic review of randomised controlled trials of multiple risk factor interventions for preventing coronary heart disease. *BMJ* 1997;314:1666–74.
27. Hill JO, Peters JC. Environmental contributions to the obesity epidemic. *Science* 1998;280:1371–4.
28. Manson JE, Willett WC, Stampfer MJ, et al. Body weight and mortality among women. *N Engl J Med* 1995;333:677–85.
29. Stamler J, Neaton JD, Wentworth DN. Blood pressure (systolic and diastolic) and risk of fatal coronary heart disease. *Hypertension* 1989;13(5 Suppl):I2–I12.
30. Stamler J, Wentworth D, Neaton JD. Is relationship between serum cholesterol and risk of premature death from coronary heart disease continuous and graded? Findings in 356,222 primary screenees of the Multiple Risk Factor Intervention Trial (MRFIT). *JAMA* 1986;256:2823–8.
31. Goldstein DJ. Beneficial health effects of modest weight loss. *Int J Obes Relat Metab Disord* 1992;16:397–415.
32. Reisin E, Abel R, Modan M, Silverberg DS, Eliahou HE, Modan B. Effect of weight loss without salt restriction on the reduction of blood pressure in overweight hypertensive patients. *N Engl J Med* 1978;298:1–6.
33. Pi-Sunyer FX. Short-term medical benefits and adverse effects of weight loss. *Ann Intern Med* 1993;119:722–6.
34. Wood PD, Stefanick ML, Dreon DM, et al. Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. *N Engl J Med* 1988;319:1173–9.
35. Wing RR, Koeske R, Epstein LH, Nowalk MP, Gooding W, Becker D. Long-term effects of modest weight loss in type II diabetic patients. *Arch Intern Med* 1987;147:1749–53.
36. Garrow J. Christmas factor and snacking. *Lancet* 2000;355:8.
37. Karlsson J, Sjostrom L, Sullivan M. Swedish obese subjects (SOS)—an intervention study of obesity. Two-year follow-up of health-related quality of life (HRQL) and eating behavior after gastric surgery for severe obesity. *Int J Obes Relat Metab Disord* 1998;22:113–26.
38. Karason K, Lindroos AK, Stenlof K, Sjostrom L. Relief of cardiorespiratory symptoms and increased physical activity after surgically induced weight loss: results from the Swedish Obese Subjects study. *Arch Intern Med* 2000;160:1797–802.
39. Hollander PA, Elbein SC, Hirsch IB, et al. Role of orlistat in the treatment of obese patients with type 2 diabetes. A 1-year randomized double-blind study. *Diabetes Care* 1998;21:1288–94.
40. Stamler R, Stamler J, Grimm R, et al. Nutritional therapy for high blood pressure. Final report of a four-year randomized controlled trial—the Hypertension Control Program. *JAMA* 1987;257:1484–91.
41. Langlois JA, Harris T, Looker AC, Madans J. Weight change between age 50 years and old age is associated with risk of hip fracture in white women aged 67 years and older. *Arch Intern Med* 1996;156:989–94.
42. Weight cycling. National Task Force on the Prevention and Treatment of Obesity. *JAMA* 1994;272:1196–202.
43. Harvey EL, Glenny AM, Kirk SF, Summerbell CD. A systematic review of interventions to improve health professionals' management of obesity. *Int J Obes Relat Metab Disord* 1999;23:1213–22.
44. Pignone MP, Ammerman A, Fernandez L, et al. Counseling to promote a healthy diet in adults: a summary of the evidence for the U.S. Preventive Services Task Force. *Am J Prev Med* 2003;24:75–92.
45. Smith IG, Goulder MA. Randomized placebo-controlled trial of long-term treatment with sibutramine in mild to moderate obesity. *J Fam Pract* 2001;50:505–12.
46. Glazer G. Long-term pharmacotherapy of obesity 2000: a review of efficacy and safety. *Arch Intern Med* 2001;161:1814–24.
47. Hill JO, Hauptman J, Anderson JW, et al. Orlistat, a lipase inhibitor, for weight maintenance after conventional dieting: a 1-y study. *Am J Clin Nutr* 1999;69:1108–16.
48. Sjostrom L, Rissanen A, Andersen T, et al. Randomised placebo-controlled trial of orlistat for weight loss and prevention of weight regain in obese patients. European Multicentre Orlistat Study Group. *Lancet* 1998;352:167–72.
49. Hauptman J, Lucas C, Boldrin MN, Collins H, Segal KR. Orlistat in the long-term treatment of obesity in primary care settings. *Arch Fam Med* 2000;9:160–7.
50. McMahon FG, Fujioka K, Singh BN, et al. Efficacy and safety of sibutramine in obese white and African American patients with hypertension: a 1-year, double-blind, placebo-controlled, multicenter trial. *Arch Intern Med* 2000;160:2185–91.
51. James WP, Astrup A, Finer N, et al. Effect of sibutramine on weight maintenance after weight loss: a randomised trial. STORM Study Group. Sibutramine Trial of Obesity Reduction and Maintenance. *Lancet* 2000;356:2119–25.
52. Finer N, Bloom SR, Frost GS, Banks LM, Griffiths

- J. Sibutramine is effective for weight loss and diabetic control in obesity with type 2 diabetes: a randomised, double-blind, placebo-controlled study. *Diabetes Obes Metab* 2000;2:105-12.
53. NHS Centre for Reviews and Dissemination. The prevention and treatment of obesity. *Effective Health Care* 1997;3.
54. O'Meara S, Glenny AM, Wilson C, Melville A, Sheldon TA. Effective management of obesity. *Qual Health Care* 1997;6:170-5.
55. National Guideline Clearinghouse [database on the Internet]. Rockville (MD): Agency for Healthcare Research and Quality; 2004 [cited 2004 Jul 28]. Available from <http://www.guideline.gov/search/searchresults.aspx?Type=3&txtsearch=obesity&num=20>.
56. National Guideline Clearinghouse [homepage on the Internet]. Washington DC: US Department of Health and Human Services, Agency for Healthcare Quality and Research; 2004 [updated 2004 Jul 12; cited 2004 July 19]. Available from: <http://www.guideline.gov>.
57. Segal KR, Dunaif A, Gutin B, Albu J, Nyman A, Pi-Sunyer FX. Body composition, not body weight, is related to cardiovascular disease risk factors and sex hormone levels in men. *J Clin Invest* 1987;80:1050-5.
58. Curtin F, Morabia A, Pichard C, Slosman DO. Body mass index compared to dual-energy x-ray absorptiometry: evidence for a spectrum bias. *J Clin Epidemiol* 1997;50:837-43.
59. Smalley KJ, Knerr AN, Kendrick ZV, Colliver JA, Owen OE. Reassessment of body mass indices. *Am J Clin Nutr* 1990;52:405-8.
60. Frankenfield DC, Rowe WA, Cooney RN, Smith JS, Becker D. Limits of body mass index to detect obesity and predict body composition. *Nutrition* 2001;17:26-30.
61. Logue E, Gilchrist V, Bourguet C, Bartos P. Recognition and management of obesity in a family practice setting. *J Am Board Fam Pract* 1993;6:457-63.
62. Taylor RW, Keil D, Gold EJ, Williams SM, Goulding A. Body mass index, waist girth, and waist-to-hip ratio as indexes of total and regional adiposity in women: evaluation using receiver operating characteristic curves. *Am J Clin Nutr* 1998;67:44-9.
63. Himmel W, Stolpe C, Kochen M. Information and communication about overweight in family practice. *Fam Pract Res J* 1994;14:339-51.
64. O'Meara S, Riemsma R, Shirran L, Mather L, ter Riet G. A rapid and systematic review of the clinical effectiveness and cost-effectiveness of orlistat in the management of obesity. *Health Technol Assess* 2001;5(18):1-81.
65. Skender ML, Goodrick GK, Del Junco DJ, et al. Comparison of 2-year weight loss trends in behavioral treatments of obesity: diet, exercise, and combination interventions. *J Am Diet Assoc* 1996;96:342-6.
66. Davidson MH, Hauptman J, DiGirolamo M, et al. Weight control and risk factor reduction in obese subjects treated for 2 years with orlistat: a randomized controlled trial. *JAMA* 1999;281:235-42.
67. Yanovski SZ, Yanovski JA. Obesity. *N Engl J Med* 2002;346:591-602.
68. Heymsfield SB, Allison DB, Vasselli JR, Pietrobelli A, Greenfield D, Nunez C. Garcinia cambogia (hydroxycitric acid) as a potential antiobesity agent: a randomized controlled trial. *JAMA* 1998;280:1596-600.
69. Request for the immediate ban of Meridia (sibutramine). Washington DC: US Food and Drug Administration. Available from: http://www.fda.gov/ohrms/dockets/dailys/02/Mar02/032202/02p-0120_cp00001_vol1.pdf.
70. Hauptman JB, Jeunet FS, Hartmann D. Initial studies in humans with the novel gastrointestinal lipase inhibitor Ro 18-0647 (tetrahydrolipstatin). *Am J Clin Nutr* 1992;55(1 Suppl):309S-13S.
71. Andersen T, Stokholm KH, Backer OG, Quaade F. Long-term (5-year) results after either horizontal gastropasty or very- low-calorie diet for morbid obesity. *Int J Obes* 1988;12:277-84.
72. Hall JC, Watts JM, O'Brien PE, et al. Gastric surgery for morbid obesity. The Adelaide Study. *Ann Surg* 1990;211:419-27.
73. Naslund I, Wickbom G, Christoffersson E, Agren G. A prospective randomized comparison of gastric bypass and gastropasty. Complications and early results. *Acta Chir Scand* 1986;152:681-9.
74. Lechner GW, Elliott DW. Comparison of weight loss after gastric exclusion and partitioning. *Arch Surg* 1983;118:685-92.
75. Ashley S, Bird DL, Sugden G, Royston CM. Vertical banded gastropasty for the treatment of morbid obesity. *Br J Surg* 1993;80:1421-3.
76. Wagner EH, Groves T. Care for chronic diseases. *BMJ* 2002;325:913-4.
77. French SA, Story M, Jeffery RW. Environmental influences on eating and physical activity. *Annu Rev Public Health* 2001;22:309-35.
78. Wadden TA, Stunkard AJ. Controlled trial of very low calorie diet, behavior therapy, and their combination in the treatment of obesity. *J Consult Clin Psychol* 1986;54:482-8.