Classification of Obesity and Assessment of Obesity-Related Health Risks

Louis J. Aronne

Abstract

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The barriers to the evaluation and treatment of obesity by health-care providers include a lack of awareness of obesity as an independent risk factor for morbidity and mortality and inadequate training in the medical management of obesity. However, the increased risk of medical disorders and emotional consequences associated with obesity make the disorder a priority for physicians to assess and treat. Obesity researchers have published and promoted the use of evidence-based, practical guidelines to educate physicians about how best to approach obesity as a medical disorder. The guidelines support classification and assessment of obesity as an important component of the patient's medical care. Assessment begins with classification by body mass index (BMI), with overweight and obesity defined as a BMI of 25 and 30 kg/m^2, respectively. Patients with high-risk combinations of BMI, waist circumference, and specific cardiovascular risk factors should begin a weight-loss program if no contraindications are present. Proper assessment also includes evaluation of complicating factors for obesity, such as sleep apnea and type 2 diabetes, psychosocial factors, and the use of medications that may contribute to obesity. Special attention should be paid to elements of the physical examination that often are performed incorrectly in obese patients, such as pelvic exams. Gathering this information will allow the clinician to tailor a weight-loss program to each patient individually. Although this represents the most challenging component of obesity care, resources are available to guide the clinician.

Key words: obesity classification, body mass index, waist circumference, risk assessment, obesity-related diseases

Department of Medicine, Weill Medical College of Cornell University, New York, New York.

Address correspondence to Louis J. Aronne, MD, Department of Medicine, Weill Medical College of Cornell University, 1165 York Ave, New York, NY 10021.

E-mail: ljaronne@mail.med.cornell.edu

Introduction

A tremendous need exists today to educate clinicians about obesity. This disorder, which results from a complex interplay of environmental and genetic factors, is associated with significant morbidity and mortality. Those afflicted suffer emotional consequences from social stigmatization in addition to having an increased risk of many medical disorders. The multiple social, economic, and hereditary factors that contribute to obesity make treatment of the condition a potentially daunting prospect, particularly for the primary care physician with limited time available to spend with each patient. Other barriers to evaluating and treating obesity include lack of insurance reimbursement for treatment, lack of time for patient education and counseling, skepticism about efficacy and safety of specific medical therapies for obesity, a negative perception that obesity represents a lack of willpower or self-discipline, and inadequate training and training mechanisms in the medical management of obesity (1).

In response to the growing epidemic of obesity and the need for physicians to recognize and treat obesity as a chronic illness, the National Institutes of Health (NIH) and the North American Association for the Study of Obesity (NAASO) have developed and published guidelines for the assessment and treatment of obesity. These guidelines, set forth by the National Heart, Lung, and Blood Institute of the National Institutes of Health in the *Practical Guide to the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults*, are evidence-based, decision-oriented, and tailored for clinical use (2). More clinicians should be educated about the availability of these guidelines and other resources to guide assessment and treatment and about the importance of treating obesity as well as its consequences.

The *Practical Guide* supports the importance of a proper assessment of obesity in treating the disorder. The recommended evaluation includes specific elements of the physical examination, history, and laboratory tests that will help clinicians identify those at greatest risk for morbidity and mortality from obesity-related disorders. The assessment should include determination of classification, or degree, of obesity because treatment guidelines are based in part on

Height	BMI 19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40
4'10"	91	96	100	105	110	115	119	124	129	134	138	143	148	153	158	162	167	172	177	181	186	191
4'11"	94	99	104	109	114	119	124	128	133	138	143	148	153	158	163	168	173	178	183	188	193	198
5'	97	102	107	112	118	123	128	133	138	143	148	153	158	163	168	174	179	184	189	194	199	204
5'1"	100	106	111	116	122	127	132	137	143	148	153	158	164	169	174	180	185	190	195	201	206	211
5'2"	104	109	115	120	126	131	136	142	147	153	158	164	169	175	180	186	191	196	202	207	213	218
5'3"	107	113	118	124	130	135	141	146	152	158	163	169	175	180	186	191	197	203	208	214	220	225
5'4"	110	116	122	128	134	140	145	151	157	163	169	174	180	186	192	197	204	209	215	221	227	232
5'5"	114	120	126	132	138	144	150	156	162	168	174	180	186	192	198	204	210	216	222	228	234	240
5'6"	118	124	130	136	142	148	155	161	167	173	179	186	192	198	204	210	216	223	229	235	241	247
5'7"	121	127	134	140	146	153	159	166	172	178	185	191	198	204	211	217	223	230	236	242	249	255
5'8"	125	131	138	144	151	158	164	171	177	184	190	197	203	210	216	223	230	236	243	249	256	262
5'9"	128	135	142	149	155	162	169	176	182	189	196	203	209	216	223	230	236	243	250	257	263	270
5'10"	132	139	146	153	160	167	174	181	188	195	202	209	216	222	229	236	243	250	257	264	271	278
5'11"	136	143	150	157	165	172	179	186	193	200	208	215	222	229	236	243	250	257	265	272	279	286
6'"	140	147	154	162	169	177	184	191	199	206	213	221	228	235	242	250	258	265	272	279	287	294
6'1"	144	151	159	166	174	182	189	197	204	212	219	227	235	242	250	257	265	272	280	288	295	302
6'2"	148	155	163	171	179	186	194	202	210	218	225	233	241	249	256	264	272	280	287	295	303	311
6'3"	152	160	168	176	184	192	200	208	216	224	232	240	248	256	264	272	279	287	295	303	311	319
6'4"	156	164	172	180	189	197	205	213	221	230	238	246	254	263	271	279	287	295	304	312	320	328

Figure 1: Body mass index chart.

classification. Related health risks should be identified with the initial assessment because they may either interfere with weight loss or require and benefit from specific treatments. Before implementing any treatment plan, it is important to assess the obese patient's readiness to lose weight. To set diet and exercise goals that the patient is most likely to follow, family history, weight history, and current lifestyle and environment should be assessed. The greatest challenge in managing obesity is tailoring a weight-loss program to the obese individual. This objective is achievable, however, for the primary care physician who appreciates the benefits of treating obesity and is aware of the resources available to guide evaluation and treatment.

Classification of Obesity

The initial step in evaluation of obesity is calculation of BMI. To measure BMI, one begins by weighing the patient in underclothes and no shoes. Height is measured without shoes. BMI is calculated by dividing weight (in kilograms) by square height (in meters). When measuring weight in pounds and height in inches, the weight is divided by the square height and the quotient is multiplied by 703, as BMI is always reported and interpreted in kilograms per square meter. Most clinicians have an available BMI table (Figure 1) that easily allows the clinician to correlate weight with BMI for a given height and shows a range of healthy weights for that height.

BMI has replaced percentage ideal body weight as a criterion for assessing obesity for several reasons. BMI correlates significantly with body fat, morbidity, and mortality, and it can be calculated quickly and easily in a busy clinical setting. Furthermore, recommendations for treatment of obesity are based on BMI. A BMI of 25 kg/m^2 is the generally accepted threshold for identifying a patient at higher risk for obesity-related diseases, most notably type 2 diabetes, hypertension, and cardiovascular disease (1). Risk of death begins to increase at a BMI of 23 kg/m^2 when compared with the lowest risk group (BMI, 19.0 to 21.9 kg/m^2) (3). Medical risk rises progressively with increasing degrees of obesity beginning with overweight, defined by BMI between 25.0 and 29.9 kg/m^2, through class I obesity (BMI, 30.0 to 34.9 kg/m^2), class II obesity (BMI, 35.0 to 39.9 kg/m^2), and class III or extreme obesity (BMI $\geq$ 40 kg/m^2) (4). More than 80% of deaths estimated to be caused by comorbidities associated with obesity occur in patients with a BMI of at least 30 kg/m^2 (3). Table 1 lists several obesity-associated disorders and indicates the estimated proportions of disease prevalence attributable to obesity.

Table 1. Proportion of disease prevalence attributable to obesity

Disease	Prevalence (%)
Type 2 diabetes	61
Uterine cancer	34
Gallbladder disease	30
Osteoarthritis	24
Hypertension	17
Coronary heart disease	17
Breast cancer	11
Colon cancer	11

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Table 2. Classification of overweight and obesity by BMI, waist circumference, and associated disease risk

			Disease risk* (relative to normal weight and waist circumference)	
	BMI (kg/m^2)	**Obesity class**	**Men ≤ 40 in (102 cm) Women ≤ 35 in (88 cm)**	**>40 in (102 cm) >35 in (88 cm)**
Underweight	<18.5			
Normal†	18.5 to 24.9			
Overweight	25.0 to 29.9		Increased	High
Obesity	30.0 to 34.9	I	High	Very high
	35.0 to 39.9	II	Very high	Very high
Extreme obesity	≥40	IIII	Extremely high	Extremely high

Adapted from the World Health Organization (5).
BMI, body mass index.
* Disease risk for type 2 diabetes, hypertension, and coronary heart disease.
† Increased weight circumference can also be a marker for increased risk even in persons of normal weight.

Table 2 provides a classification of overweight and obesity by BMI, waist circumference, and associated disease risk. This classification system of obesity by BMI was developed by the World Health Organization Obesity Task Force and has been adopted by the Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults, a group assembled by the National Heart, Lung, and Blood Institute of the National Institutes of Health (5,6).

Waist circumference is an important measure of obesity risk. Waist circumference is measured at the level of the top of the right iliac crest. The measuring tape should be snug but not compressing the skin and held parallel to the floor. The measurement is made at normal respiration (6). A high-risk waist circumference is accepted to be 35 inches or greater for women and 40 inches or greater for men.

Waist circumference is a practical indicator of visceral abdominal fat. Evidence suggests that abdominal fat carries a higher health risk than peripheral fat, and that the visceral fat component correlates the most strongly with increased risk (6). Whereas computed tomography and magnetic resonance imaging allow more precise measurement of abdominal fat, they are impractical for routine clinical use (6). Some epidemiological studies have found the waist-to-hip ratio to correlate with increased risk for diabetes, coronary heart disease (CHD), and hypertension (6); however, this measure is not established as an independent risk factor. Waist circumference also has been found to be a superior indicator of abdominal fat distribution (6).

The truncal fat distribution indicated by an increased waist circumference correlates with the hypertrophic form of obesity. Hypercellular obesity, which is characterized by an increased total number of fat cells, typically affects patients with a BMI <40 kg/m^2 but may be a lower risk form of disease. In hypertrophic obesity, existing fat cells enlarge and produce proteins and metabolites involved in the pathophysiology of obesity (7). These proteins include lipoprotein lipase, which contributes to hydrolysis of the triglycerides of very-low-density lipoproteins (VLDL) and chylomicrons, and cytokines (tumor necrotizing factor-α and interleukin-6), as well as angiotensinogen (8). The hypertrophied fat cell also produces leptin, a hormone involved in animal models of obesity. Hypertrophic obesity correlates with metabolic complications of obesity, including impaired glucose tolerance, adverse lipid profile, hypertension, and CHD (7).

Because waist circumference is an independent risk factor for increased risk of complications from obesity, treatment guidelines include this measurement as a parameter in algorithms designed to determine appropriate obesity treatment. Waist circumference may have additional value in the elderly, in whom decreased muscle mass contributes to underestimation of obesity-related risk by BMI alone, and in some ethnic groups genetically predisposed to unfavorable distribution of fat despite normal body weight (6). Once patients begin treatment for obesity, waist circumference can show an improvement in body-fat distribution, implying a lower health risk even when BMI does not change.

In comparison with measurement of the BMI and waist circumference, history and physical examination constitute the more time-consuming component of an obesity evaluation. In an initial and potentially time-limited assessment of obesity, the goal of which is to identify patients who should be treated for obesity, clinicians should ascertain the smoking history and family history of coronary artery disease. These are the two historical factors included in the *Practical*

Guide's algorithm for medical risk assessment that will enable clinicians to identify a patient at increased medical risk, however, a comprehensive history and physical examination are essential. The history should address trends in the patient's weight over his or her lifetime; risk factors for obesity, such as family history; and diet and exercise habits. As appropriate, the patient should be screened for medical conditions established to contribute to obesity, with confirmatory laboratory studies sent if indicated. The current medication list should be reviewed. The patient also should be evaluated for complications of obesity. More specific recommendations for assessment and evaluation are reviewed later.

Specific historical data that may help to elucidate the factors contributing to a patient's obesity include the following: family history of obesity as quantified by number of first-degree relatives with the condition; lifestyle changes that heralded the onset of weight gain, such as beginning or graduating college, childbirth, marriage, divorce, or a job change; and changes in exercise or eating habits (9). Age of onset of obesity should be determined in part because the age potentially can be correlated with lifestyle changes contributing to obesity. Also, age of onset helps to distinguish hypercellular from hypertrophic obesity, because the hypercellular form often begins in childhood, whereas hypertrophic obesity often begins in adulthood. It may be helpful to review alcohol consumption habits, because alcoholism causes hypercortisolism and a central obesity syndrome similar to Cushing's syndrome (10).

The pattern of weight gain and loss since puberty is important to ascertain when developing a patient's treatment plan. Evidence does not support an association between persistent metabolic abnormalities and a history of weight cycling, nor should weight cycling be considered a contraindication to obesity treatment (4,9). Patients with binge-eating disorder, characterized by eating large amounts of food over a short time, should be identified, because a disproportionate percentage of these patients have psychiatric disorders that may respond to treatment (6). In such patients, and in others with suspected eating disorders, normalization of eating patterns should take precedence over weight loss (11). Because obese patients are rarely able to attain a weight lower than their minimum adult weight, this value also should be determined and used to guide treatment goals. Finally, diet history can identify patients who, despite a normal weight, have either recently gained or currently are gaining weight. Weight gain itself carries an increased risk of morbidity and mortality from obesity-related diseases. For example, one study found a doubling of risk of developing type 2 diabetes with weight gain of 5 to 8 kg and a near-quadrupling of risk with a 22-kg weight gain (hazard ratio, 2.11 and 3.85, respectively) (12). Other studies have found increased risks of CHD with weight gain. Weight gain in early adulthood may predict an increased risk of CHD occurring much later in life. For example, among a cohort of Japanese-American men, weight gain of 5.1 to 10 kg after age 25 carried a relative risk of nonfatal myocardial infarction of 1.60 when compared with men who gained 2.5 kg or less. A gain of more than 10 kg was associated with a relative risk of 1.75 (13). For women as well, weight gain at any time in adulthood has been found to be a strong predictor of CHD later in life. Among a large cohort of women, CHD risk was studied in women with weight gain after age 18 compared with those with stable weight ($\pm$5 kg). Relative risk was determined to be 1.64 for weight gain of 8 to 10.9 kg, 1.92 for weight gain of 11 to 19 kg, and 2.65 for weight gain of 20 kg or more (14).

Patients with a sedentary lifestyle should be identified, because this lifestyle contributes to obesity and because the sedentary lifestyle itself is an independent risk factor for all-cause and cardiovascular mortality (6,7). Conversely, in some obese patients with underlying coronary disease, a sudden increase in physical activity could be dangerous, but more importantly, a baseline level of activity needs to be established to make exercise recommendations (4).

Special attention should be paid to specific conditions that not only predispose patients to obesity but also influence health in other ways and may respond to treatment. These conditions include polycystic ovarian syndrome (PCOS), hypothyroidism, and obstructive sleep apnea. PCOS usually presents with hirsutism and infertility in women of reproductive age. Patients are often obese, with peripheral insulin resistance and hyperinsulinemia believed to be key factors in the development of associated abnormalities. These abnormalities include elevated luteinizing hormone, depressed follicle stimulating hormone, elevated plasma androgens, elevated plasma estrogens derived from increased peripheral aromatization of the androgens, and anovulation. Persistent anovulation, which is present in $\sim$20% of patients with PCOS, results in infertility, menstrual abnormalities, hirsutism, and acne. It also places patients at increased risk of endometrial cancer, breast cancer, and cardiovascular disease (15). PCOS may respond to diet therapy and/or drug therapy with metformin. One study found that metformin treatment in PCOS led to normalization of endocrine function, including menstrual irregularities and anovulation, with minimal adverse effects (16).

Hyperinsulinemia and peripheral insulin resistance, although usually believed to be consequences of rather than causes of obesity, are predictors of future weight gain (8). Patients with type 2 diabetes treated with insulin tend to gain more weight, especially when on an intensive rather than standard insulin regimen (17). This may result from hypoglycemia, which can act as a strong appetite stimulant. Hyperinsulinemia and peripheral insulin resistance respond well to exercise (through increased number and sensitivity of insulin receptors) and weight loss. They also may improve with metformin treatment.

Hypothyroidism contributes modestly to obesity by slowing the metabolic rate (7). Some weight gain may be because of development of edema (10). Because the disorder is common, is easily tested for using the serum thyroid-stimulating hormone, and can be treated with thyroid hormone replacement therapy, a thyroid-stimulating hormone test should be included in the initial laboratory evaluation of an overweight or obese patient. Hyperthyroidism, which is also a common and treatable disorder, most commonly causes weight loss but can cause mild weight gain because of increased appetite (10).

Rare endocrine causes of obesity in humans include Cushing's syndrome, pancreatic insulinoma, growth hormone deficiency, and hypothalamic insufficiency. These disorders may be evaluated for when suspected on the basis of history and physical examination. Cushing syndrome may be suspected when patients have central obesity, diabetes, hypertension, proximal muscle weakness, and a history of easy or spontaneous bruising, and it can be confirmed by 24-hour urinary free cortisol or overnight dexamethasone suppression test (18). Obesity with headache and signs of hypopituitarism, with or without history of head trauma or brain surgery, may signal the need to image the hypothalamus and pituitary by computed tomography scan or magnetic resonance imaging (18). Hypothalamic insufficiency contributes to obesity by hyperphagia and by increased parasympathetic and decreased sympathetic nervous system activity (8). Hypothalamic lesions, although rare, can cause massive obesity (10).

Obstructive sleep apnea (OSA) is an under-recognized and often misdiagnosed disorder. Although managing sleep apnea has not been found to spontaneously cause weight loss, it seems difficult for the patient with untreated sleep apnea to lose weight because of daytime sleepiness resulting in hunger and difficulty focusing on the problem. This may be related to impairments of performance in divided attention tasks (19) and in cognitive function, particularly when OSA is complicated by nocturnal hypoxemia (20). Although OSA may interfere with weight loss through cognitive function impairment, it also carries serious health risks, including increased risk of cardiac arrhythmia, severe nocturnal hypoxemia, congestive heart failure, and pulmonary hypertension.

OSA should be suspected when the patient or the patient's partner reports a history of loud snoring or cessation of breathing during sleep, which is often followed by a loud clearing breath with brief awakening. Patients may admit to excessive daytime sleepiness at inappropriate times, such as while driving. One study found the question, "Do you fall asleep during the day, particularly when not busy?" to be a superior predictor of increased oxygen desaturation episodes overnight (21). Studies have found the biggest risk factor for sleep apnea to be neck circumference adjusted for height (21). Individuals with suspected OSA can be screened with overnight home oximetry, which has a high negative predictive value (22), or a formal sleep study. Patients with OSA should avoid alcohol and hypnotic medications because these contribute to relaxation of upper airway musculature with resulting airway compression—the pathophysiologic process at issue in OSA. The disorder often is treated with continuous positive airway pressure (CPAP) overnight. Compliance is a problem, however, with 25% of patients discontinuing use within the first year of treatment (22). Referral to an ear, nose, and throat physician and treatment with oral appliances or surgery may be appropriate in selected patients (22).

A variety of psychosocial factors contribute to the development of obesity and to difficulty losing weight. Identifying which, if any, factors are at issue in a given patient will help to guide the behavioral component of obesity therapy. This approach is supported by evidence from more than 100 controlled clinical trials documenting the effectiveness of behavioral therapy in treating obesity (23). In addition to facilitating weight loss, addressing these factors may improve the patient's quality of life and self-esteem. Patients who eat as a coping mechanism in response to stress may benefit from education about stress management strategies (23). Those caught in a cycle of negative thinking because of repeated failed weight-loss attempts may benefit from coaching in problem-solving skills (23). The process of identifying problem areas and generating solutions helps the patient replace self-punishment with a positive attitude toward doing better the next time (23). Some patients may benefit from referral to a professional with training in psychology or social work (23).

Of particular importance in the assessment of the obese patient is a thorough review of present and previous medications. Comorbid diabetes, mental and nervous disorders, seizure disorders, and immune disorders should alert the physician to the possibility that the patient's medications might be contributing to his or her obesity. Medications documented to increase weight gain include antipsychotics (phenothiazines, butyrophenones); antidepressants and antiepileptics, some of which are used as mood stabilizers as well (tricyclic antidepressants, lithium, valproate, carbamazepine); and insulin and some oral hypoglycemics. Whereas most of these medications contribute modestly to obesity, the large doses of steroids sometimes used to treat autoimmune diseases can cause true obesity. In patients taking prednisone at doses of 10 mg or more per day, weight gain may occur with a predominantly truncal distribution (7). For many of these and other medications documented to cause weight gain, alternative drugs can be substituted. Table 3 lists some representative categories of drugs that may cause weight gain, with alternatives listed. The antipsychotics, in particular, are well studied with regard to differential weight gain associated with specific drugs. Nonsteroidal anti-inflammatory drugs and disease-modifying agents, such as etanercept for rheumatoid arthritis, may be

Table 3. Medications that may promote weight gain and suggested treatment alternatives

Drugs that may promote weight gain	Alternative drugs that may promote weight loss or be weight neutral
Psychiatric/neurologic treatments	
• Antipsychotics: olanzapine, clozapine, risperidone	• Ziprasidone, quetlapine
• Antidepressants: selective serotonin reuptake inhibitors, tricyclic antidepressants	• Bupropion, nefazodone
• Lithium	
• Antiepileptic drugs: valproate, gabapentin, carbamazepine	• Topiramate, lamotrigine, zonisamide
Diabetes treatments	
• Insulin	• Metformin
• Sulfonylureas	• Acarbose, miglitol
• Thiazolidinediones	• Orlistat, sibutramine
Steroid hormones and miscellaneous agents	
• Hormonal contraceptives	• Barrier methods
• Corticosteroids	• Nonsteroidal anti-inflammatory drugs
• Progestational steroids	
• Antihistamines	• Decongestants, inhalers
• α-Blockers, β-blockers	• Angiotensin-converting enzyme inhibitors, Ca^{2+} channel blockers

Adapted from Aronne (23).

initiated as steroid-sparing agents in treatment of some inflammatory disorders. When treating patients with diabetes, clinicians should be aware of the potentially beneficial effects of metformin, alpha glucose inhibitors, and weight loss, in comparison with exogenous insulin and many other oral hypoglycemics. For example, in addition to improving insulin sensitivity, metformin is not associated with weight gain (24). Weight gain or loss associated with different antidepressants also has been well studied.

As part of the medication history, clinicians must inquire about past or present use of weight-loss medications. This includes over-the-counter and herbal supplements patients may be using as weight-loss aids. A wide variety of these are available to the public. Some have insufficient documented efficacy to recommend their use, such as chromium picolinate, chitosan, L-carnitine, and hydroxycitric acid (23). Others have sympathomimetic effects that predispose patients to arrhythmia and hypertension. These include the over-the-counter medication phenylpropanolamine and herbal supplements, such as ephedra, ma huang, "herbal fen/phen," guarana, and gotu kola (23). The lack of ingredient standardization and testing in these agents, combined with the prevalence of CHD among the obese, makes them generally unsafe, and their use should be discouraged, with alternative management strategies substituted. Patients who have been treated previously with dexfenfluramine or fenfluramine should be evaluated for signs and symptoms of valvular heart disease. If these are present, or if a previously treated patient is about to undergo a dental procedure, an echocardiogram should be performed in accordance with Centers for Disease Control and Prevention guidelines (25).

By definition, patients with major depression may experience significant changes in appetite and body weight. These include a weight gain of more than 5% of body weight within a month and a self-reported increased in appetite nearly every day (26). Major depressive episodes also may be characterized by feelings of worthlessness or inappropriate guilt (26), which can contribute to patterns of negative thinking with resulting failure to lose weight. Cognitive dysfunction, specifically, diminished ability to think or concentrate (26), can figure prominently in the symptomatology of a depressed patient. Up to 91% of depressed patients may suffer from poor concentration (26). Such a patient may have difficulty focusing on his or her weight loss. Impairment of problem-solving ability and insight may cripple weight-loss efforts and potentiate the cycle of negative thinking. Depression may be complicated by alcoholism or substance abuse that further interfere with weight loss. Depression responds to some lifestyle therapies, such as exercise, and to an array of medications appropriate for prescription by the primary care physician. These factors, as well as the considerable morbidity from depression itself,

Table 4. Symptoms, diseases, and special problems associated with obesity

Cardiovascular system
Coronary heart disease
Hypertension
Pulmonary embolism
Varicose veins

Gastrointestinal system
Cholelithiasis
Gastroesophageal reflux disease (GERD)
Colon cancer
Hepatic steatosis
Hernias
Nonalcoholic steatohepatitis (NASH)

Integumental system
Cellulitis
Carbuncles
Hygiene problems
Intertrigo
Venous stasis of legs

Musculoskeletal system
Immobility
Low back pain
Osteoarthritis

Genitourinary system
Hypogonadism
Prostate cancer
Urinary stress incontinence

Neurologic system
Idiopathic intracranial hypertension
Meralgia paresthetica
Stroke

Psychosocial
Depression
Social/employment discrimination
Work disability

Reproductive/endocrine systems
Amenorrhea
Breast cancer
Cushing syndrome
Type 2 diabetes
Dyslipidemia
Glucose intolerance
Hypothyroidism
Infertility
Insulin resistance
Uterine cancer
Polycystic ovary syndrome (PCOS)

Respiratory system
Dyspnea and fatigue
Obesity-hypoventilation syndrome (Pickwickian syndrome)
Obstructive sleep apneas (OSA)

Reprinted with permission from Weinsier and Kushner (9).

mandate the screening for depression as part of the medical history in overweight and obese patients.

Physical examination should target signs or conditions that predispose to or are complications of obesity. In looking for possible secondary causes of obesity, clinicians should be alerted by mild hirsutism in women (PCOS), large neck size (sleep apnea), thyroid tenderness or goiter (hypothyroidism), slowed reflexes (hypothyroidism), and proximal muscle weakness (Cushing's syndrome, hypothyroidism) (9). A number of skin findings also may signal secondary causes of obesity, including striae (Cushing syndrome's, steroid use), hirsutism, acne (Cushing syndrome's, PCOS), and dry or coarse skin and hair (hypothyroidism) (9).

An extensive evidence review published in 1998 by the National Heart, Blood, and Lung Institute of the NIH found ample evidence for morbidity and mortality from CHD, hypertension, and type 2 diabetes increasing with BMI (6). Other conditions increasing with BMI in the overweight and obese ranges include stroke, gallbladder disease, osteoarthritis, and sleep apnea. Additionally, morbidity and mortality from endometrial, breast, prostate, and colon cancers are increased among the obese (6). Table 4 presents a comprehensive list of medical disorders for physicians to keep in mind while they perform the physical examination.

Care should be taken to perform elements of the physical examination that often are not performed, or are incorrectly performed, in obese patients. For example, gynecologic examinations often are not performed in obese women. This may be in part because obese women go to the gynecologist less often and because of clinician reluctance to perform the

examination because it may be more difficult to conduct and less sensitive to detect abnormalities. Given the increased risks of PCOS and endometrial cancer in these women, the examination should be performed at intervals appropriate for the patient's age. Similarly, rectal examinations sometimes are neglected in obese patients but should be performed at the same intervals as in the non-obese population. Importance of using a blood pressure cuff of the correct size cannot be overemphasized. Specifically, the width of the cuff's inflatable bladder should be at least 40% of the arm's circumference, and the length of the bladder should be at least 80% of the circumference (27). Guidelines for cuff selection size usually are printed on the cuff. Use of a cuff that is too small will overestimate blood pressure.

As with physical examination, laboratory and radiologic testing should include studies recommended for all patients regardless of BMI as appropriate for age and risk factors other than obesity. These include mammography, fecal occult blood testing, flexible sigmoidoscopy, and Papanicolaou stain testing. Of note, the most recently published guidelines for hyperlipidemia diagnosis and treatment advocate checking a fasting lipid profile every 5 years beginning at age 20; this represents a change from the previous recommendations, which recommended nonfasting total cholesterol as the screening test for men ages 35 and older and women ages 45 and older (28). Laboratory studies may be useful in identifying secondary causes of obesity. These include 24-hour urine collection for free cortisol (Cushing's syndrome) and serum lutenizing hormone and follicle-stimulating hormone (PCOS) (9). Other studies may be indicated to confirm the diagnosis of complications of obesity that are suggested by the history and physical examination for a specific patient. Finally, as part of the algorithm for risk stratification as presented in the *Practical Guide*, all patients with a BMI of 25 kg/m^2 or greater and/or waist circumference $>$35 inches for women or $>$40 inches for men should have a fasting lipid profile and fasting serum glucose performed to identify risk- and treatment-modifying risk factors. Because hypothyroidism sometimes is associated with weight gain, can be diagnosed accurately using the serum thyroid-stimulating hormone, and can be treated with thyroid hormone replacement, a thyroid-stimulating hormone test should be sent for all obese patients.

Assessment of the Obese Patient

One of the goals of assessment in an obese patient is to decide whom to treat. Three main issues must be considered: 1) whether treatment is indicated, 2) whether treatment is safe for the patient, and 3) whether the patient is ready and motivated to lose weight. Figure 2 shows a suggested algorithm for stratifying risk in patients. This algorithm is a simplified version of the algorithm presented in the *Practical Guide*. The algorithm takes into account the BMI; waist circumference; and a finite group of risk factors, including cigarette smoking, hypertension, elevated LDL-cholesterol, low levels of HDL-cholesterol, impaired fasting glucose, family history of coronary heart disease, and age. Of note, patients with a BMI of 30 kg/m^2 or greater should be considered for treatment regardless of waist circumference or risk factors. Treatment may be indicated for patients of normal weight if they have a waist circumference $>$35 inches for women or $>$40 inches for men, as well as two or more of the listed risk factors.

The recommendations should not be implemented without first considering their applicability to an individual patient. In muscular patients and in edematous patients, for example, BMI can overstate medical risk by overestimating body fat. Because muscle mass declines with age, BMI can understate risk in the elderly. The relationship between BMI and body fat can vary with ethnicity and gender (2), although including waist circumference as a parameter in risk assessment may help compensate for associated differences in fat distribution.

Weight-loss therapy is contraindicated in some patients. In most obese pregnant or lactating women, weight maintenance, but not weight loss, may be recommended. Treatment of active psychiatric disorders, including most eating disorders and forms of substance abuse, takes precedence over weight loss. Weight loss should not be recommended for patients with acute illnesses, or in the terminal stages of illness, such as cancer or in serious medical conditions, which might be exacerbated by caloric restriction (6,29).

Before beginning a weight-loss program, patients should be evaluated for number and severity of cardiovascular risk factors. These conditions may require that treatment be initiated along with weight-loss strategies. However, they also may alert the clinician to a need for a cardiac evaluation before starting the patient on an exercise program. Patients with major risk factors for coronary artery disease, including obesity, may need to undergo an exercise stress test before starting a conditioning program (30). In a full-intensity program, the patient exercises to 80% of the maximal predicted heart rate for at least 20 minutes at least three times per week (30). Some studies have found health benefits associated with low-intensity exercise even when cardiovascular fitness has not increased. These benefits include an increase in HDL (31) and a decrease in all-cause mortality (32).

Patients who are unready or unwilling to lose weight rarely succeed in a weight-loss program. As part of the *LEARN Program for Weight Control*, Brownell and Wadden (33) recommend exploring the following issues to help evaluate a patient's readiness to make the lifestyle changes necessary to achieve and maintain a lower weight: the patient's reasons for wanting to lose weight; what support the patient expects from family and friends; the possible risks and benefits the patient associates with weight loss; the patient's attitude toward physical activity; the patient's past

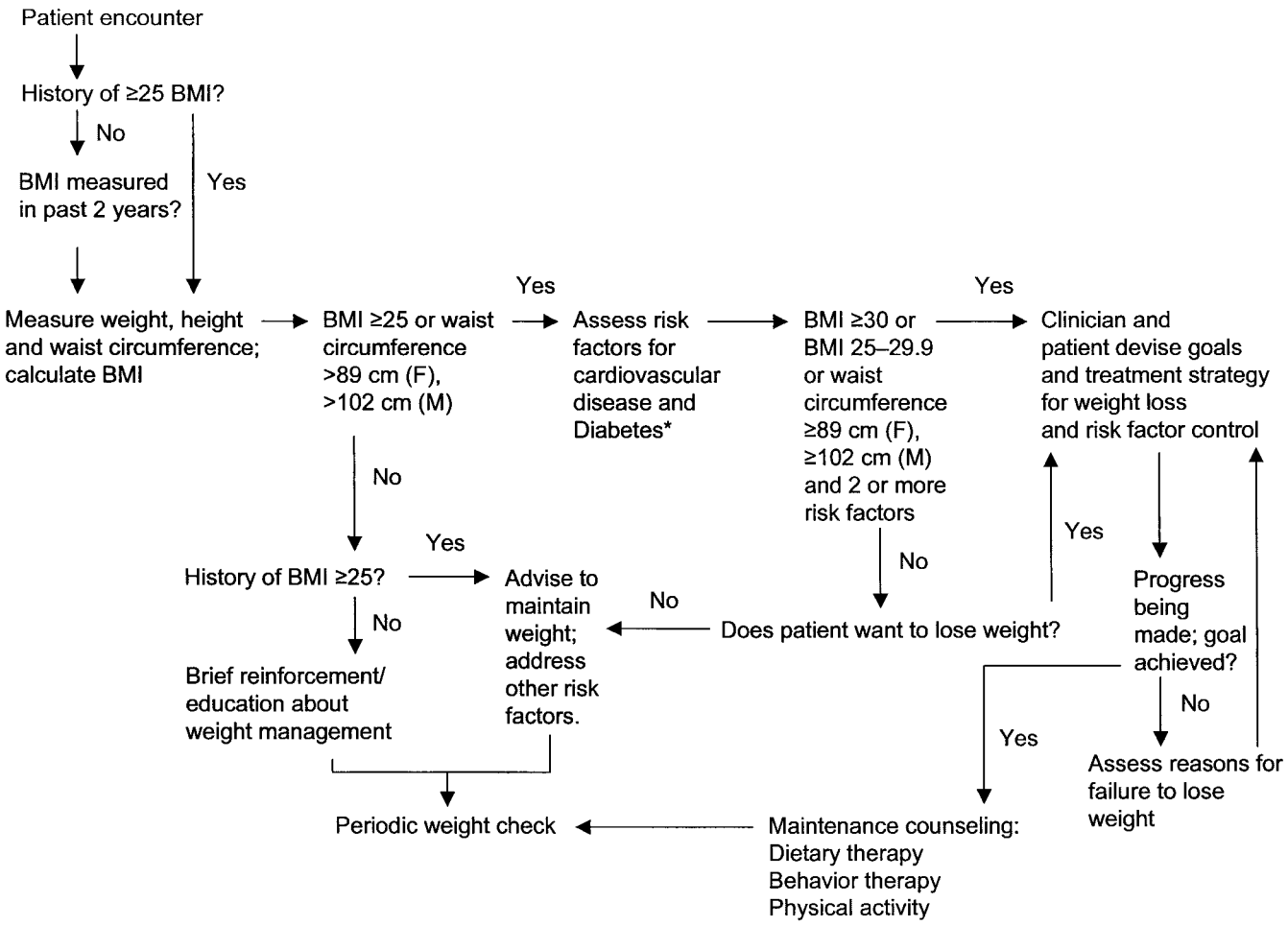

Figure 2: Treatment algorithm for assessment of overweight and obesity. BMI, body mass index; F, female; M, male. Adapted from NIH, NHLBI, and NAASO (6).

experiences with weight loss attempts; and what barriers, including time and financial limitations, could frustrate planned weight-loss efforts (29,33).

Patients at low to moderate risk who are not ready to lose weight should be urged to maintain their current weight. A healthy lifestyle should be encouraged, and any complications of obesity should be managed appropriately. Patients at high risk should be educated about the benefits of weight loss, with the goal of motivating them while maintaining a therapeutic alliance.

For patients who are ready to lose weight, reasonable goals for diet and physical activity should be set. For most patients, weight-loss goals should initially be 5% to 10% of current weight, because this degree of weight loss is reasonable and results in health benefits. Planned diets should provide 1000 to 1200 kcal/d. With very-low-calorie diets, muscle mass is lost as well as fat and body water. The clinician should draw on obesity-related resources, such as the *Practical Guide* and the *LEARN Program*, in evaluating and treating patients. Other professionals, such as dietitians and mental health practitioners, may need to be involved to help the patient lose weight safely and successfully. Finally, a supportive, empathetic approach should be taken to maintain an optimal therapeutic alliance.

Summary

Proper identification and classification of obesity through a determination of BMI and waist circumference and identification of specific clinical risk factors are the most important steps to initiate before beginning weight-loss treatment. These measures are evidence-validated predictors of morbidity and mortality, and treatment recommendations are guided by overall risk stratification, which takes these three factors into account.

Identification of medical conditions and drugs that may be contributing to a patient's obesity may help facilitate the

weight-loss process and improve quality of life once appropriate interventions or modifications are implemented. Similarly, treatment of comorbidities resulting from a patient's obesity should occur in conjunction with weight-loss efforts. The benefits of weight loss in treating many obesity-related conditions are well established and should not be underestimated or ignored.

To effectively manage obesity, physicians must explore with a patient his or her goals, level of readiness, possible obstacles to weight loss, and motivation to lose weight. The greatest challenge to physicians in this process is the reconciliation of these patient-specific factors with the patient's overall medical risk from obesity to develop an individually tailored treatment plan.

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