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Clinical Evaluation of the Overweight Patient

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Evaluation of an overweight patient is the first step in any therapeutic program. The syndromes of obesity can be classified in several ways. The first is an anatomic classification based on the size, number, and distribution of fat cells and fat tissue. The second is an etiologic classification based on identification of specific diseases and settings that produce obesity. Hypothalamic injury and endocrine disease such as Cushing's disease and the polycystic ovary syndrome are three identifiable causes of obesity. In this medicated society drugs are always candidates to produce weight gain. The most common causes, however, are stopping smoking, overconsumption of high-fat foods, a decrease in the level of activity, and aging. The natural history of obesity provides a useful framework in which to view both preventive and therapeutic strategies. Some individuals will never become overweight, but of those who do, about one-third will do so during the first two decades, and the remaining two-thirds will become overweight after age 20. A number of epidemiological and metabolic factors can serve as a guide to those individuals who are at high risk. Having overweight parents tops the list, but multiple births, cessation of smoking, and a sedentary lifestyle are additional factors. Therapeutic decisions should be based on risk-benefit decisions. The risk can be assessed from the body mass index, the distribution of fat in upper or lower body obesity, the rate of weight gain, and the degree of physical inactivity. After assessing risk, the therapeutic choices can be selected from the age category of the patient. With any therapeutic activity, involvement of the patient in a realistic approach to the treatment process is essential.

Key Words: Causes of obesity; natural history of obesity; body mass index.

The Realities of Being Overweight

Overweight is a chronic, stigmatized disease that is increasing in prevalence. An estimated 97 million people in the United States are now overweight or obese, representing 54.9% of the adult population. The social disapproval of obesity and the lengths to which people go to prevent or reverse it fuels an annual $50 billion set of industries. Nearly 65% of women in the United States consider themselves overweight and even more (66–75%) wish to weigh less. The figures for men are somewhat less. More than 50% of the women with a body mass index (BMI) <21 kg/m^2 (normal weight) wish to weigh less. It is this individual perception of what is a "desirable" weight that indicates the degree of stigmatization for those who are not "thin" and the drive to lose weight.

The cultural expectations for thinness can be seen in the decreasing weight of the Miss America contestants since 1950 and in the centerfolds of adult magazines. The stigma of obesity is also evident in the general public disapproval of corpulence and in the disapproving moralistic attitudes of many health care professionals. For example, mental health workers are more likely to assign negative psychologic symptoms to the obese than to normal weight people. Nursing, medical, and ancillary health care personnel also carry these negative stereotypes. Sensitivity training for health professionals dealing with overweight patients is an important task in any office or clinic offering treatment for obesity.

Clinical Classification

There are many causes of overweight, and it can be classified in several ways.

Anatomic Characteristics of Adipose Tissue and Fat Distribution

An anatomic classification of obesity can be based on the number of adipocytes, the regional distribution of body fat, or the characteristics of localized fat deposits *(1,2)*. The pathology of obesity is the increase in size and number of fat cells.

Size and Number of Fat Cells

The number of fat cells can be estimated from the total amount of body fat and the average size of a fat cell. Because fat cells differ in size from one region of the body to another, a reliable estimate of the total number of fat cells should be based on the average of fat cell size from more than one location. In adults, the upper limit of normal fat

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cell number ranges from 40 to 60 × 10^9 cells. The number of fat cells increases most rapidly during late childhood and puberty but may increase even in adult life. The number of fat cells can increase by three- to fivefold when obesity occurs in childhood or adolescence.

Hypertrophic obesity, which is obesity with enlarged fat cells but not an increased number of fat cells, tends to correlate with an android or truncal fat distribution, and is often associated with metabolic disorders such as glucose intolerance, dyslipidemia, hypertension, and coronary artery disease. Hypertrophy of the fat cell is the pathologic lesion of obesity. These enlarged fat cells produce increased amounts of several peptides and metabolites that are involved in the pathophysiology of obesity.

Hypercellular obesity, which is obesity with an increased number of fat cells, shows varying degrees of increase in the number of fat cells. This type of obesity usually begins in early or middle childhood, but may also occur in adult life. An increased total number of fat cells is usually present in individuals who have a BMI <40 kg/m^2.

Fat Distribution

Fat distribution can be estimated by a variety of techniques. The ratio of the waist circumference to the circumference of the hips was the technique used in the pioneering studies that brought scientific recognition to the relationship of central fat to disease in the 1980s, a concept originally suggested by Vague *(44)*. The subscapular skinfold measurement has also served as a valuable tool to estimate central fat in epidemiologic studies. A more sophisticated technique to evaluate skinfolds uses principal components analysis of skinfolds at several sites on the body. The principal components analysis groups together those skinfolds that are best correlated and gives an estimate of total fat, central fat, and peripheral fat. However, accurate measurement of visceral fat can be made only by computed tomography (CT) or magnetic resonance imaging (MRI) scans. Waist circumference alone and waist circumference divided by hip circumference is used later as one criterion for evaluating health risk from obesity.

Localized Deposits of Fat and Lipodystrophy

There are several kinds of localized fat accumulations, including single lipomas, multiple lipomas, liposarcomas, and lipodystrophy *(2)*. Lipomas vary in size from 1 to >15 cm. They can occur in any region of the body and represent encapsulated accumulations of fat. Multiple lipomatosis is an inherited disease transmitted as an autosomal dominant trait. Von Recklinghausen syndrome, Mafucci syndrome, and the Madelung disease are examples of lipomatous syndromes.

Lipodystrophy is a loss of body fat in one or more regions of the body. Total lipodystrophy is a familial disease with absent sc fat. Partial lipodystrophy is usually acquired and can affect the upper or lower part of the body. The total rate of fat metabolism appears to be accelerated in the affected regions of the body in individuals with lipodystrophy.

Table 1
Causes of Hypothtlamic Obesity

Hypothalamic lesion
Tumors
Inflammation
Trauma
Endocrine disturbances
Amenorrhea/impotence
Impaired growth
Diabetes insipidus
Thyroid/adrenal insufficiency
Intracranial pressure
Papilledema
Vomiting
Neurologic disturbances
Thirst
Somnolence

Etiologic Classification

Neuroendocrine Obesity

A variety of neuroendocrine disorders may be associated with the development of obesity.

HYPOTHALAMIC OBESITY

Hypothalamic obesity is a syndrome that occurs rarely in humans but can be regularly produced in animals by injury to the ventromedial or paraventricular region of the hypothalamus or the amygdala *(3)*. These regions of the brain are responsible for integrating metabolic information regarding nutrient stores with afferent sensory information about food availability. When the ventromedial hypothalamus is damaged, hyperphagia develops and obesity follows.

Table 1 outlines the causes of hypothalamic obesity in humans. Hypothalamic obesity may be caused by trauma to the head, hypothalamic tumors, inflammatory disease, surgery in the posterior fossa, or increased intracranial pressure *(4)*. The symptoms usually present in one of three patterns: 1eadache, vomiting, and diminished vision; impaired endocrine function affecting the reproductive system with amenorrhea or impotence, diabetes insipidus, and thyroid or adrenal insufficiency; or neurologic and physiologic derangements including convulsions, coma, somnolence, and hypothermia or hyperthermia. Figure 1 gives the clinical presentation of one patient. Weight gain occurred in the third year following the appearance of several endocrine and hypothalamic changes. The patient died with multiple tuberculomas in her hypothalamus in spite of therapy with triple antibiotics.

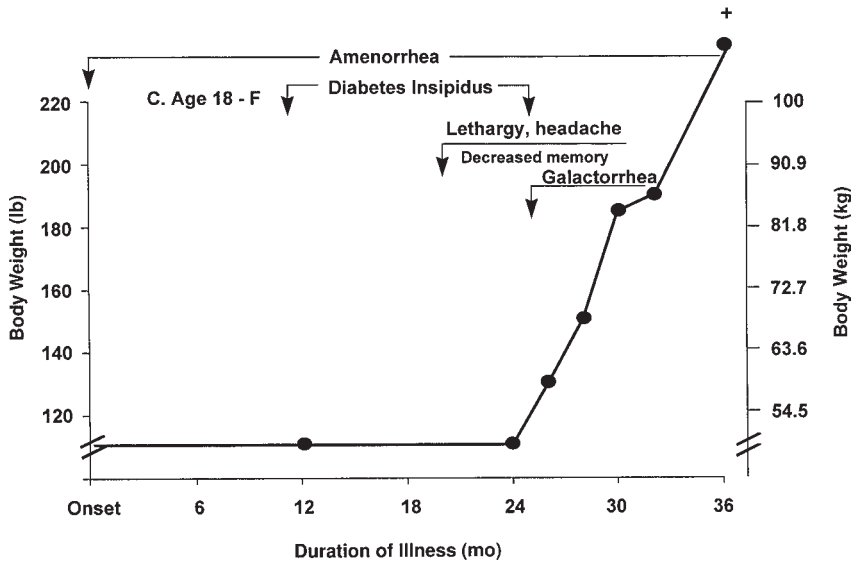

Fig. 1. Symptomatic course of a patient with tuberculomas and hypothalamic obesity. During the first 2 yr numerous symptoms developed, followed in the third year by rapid weight gain.

Table 2
Features of Cushing Syndrome

Central obesity
Hypertension
Plethoric facies
Amenorrhea
Virilism
Edema of lower extremities
Hemorrhagic features

Cushing Syndrome

A common clinical feature in patients with Cushing syndrome is progressive central obesity, usually involving the face, neck (leading to a buffalo hump and obscuring of the clavicles), trunk, abdomen, and, internally, the mesentery and mediastinum *(5)*. The extremities are usually spared and are often wasted (Table 2).

In contrast to adults, nearly all children with Cushing syndrome have generalized obesity, accompanied by a decrease in linear growth. As a result, any child whose weight rises and whose stature remains static when compared with age-matched normal growing children should be evaluated for Cushing syndrome. Identifying Cushing syndrome in obese patients is clinically important for decisions about therapy. Three screening tests can be used to identify patients for a dexamethasone–corticotropin-releasing hormone (CRH) test to confirm Cushing disease. These screening tests are as follows: 24-h urinary-free cortisol (normal: <150 nmol/24h); cortisol production rate (normal: <80 nmol/kg/24 h); and overnight dexamethasone suppression test (0.5 mg of dexamethasone at midnight—AM plasma cortisol of <75 nmol/L). Any patient who is abnormal in one of these tests should receive 0.5 mg of dexamethasone every 6 h for 2 d followed by 100 μg of CRH intravenously. A normal value for this dexamethasone-CRH test is <38 nmol/L of cortisol, 15 min after CRH *(6)*.

Hypothyroidism

Patients with hypothyroidism frequently gain weight owing to a generalized slowing of metabolic activity. Some of this gain is fat. However, the weight gain is usually modest and marked obesity is uncommon. Hypothyroidism is a common diagnosis, particularly in older women. In this group a thyroid-stimulating hormone (TSH) test is a valuable diagnostic test.

Polycystic Ovary Syndrome

Upward of 50% of women with polycystic ovary syndrome are obese *(7)*. The cardinal features of this syndrome are oligomenorrhea, hirsutism, and polycystic ovaries (Table 3). Although obesity is not always present, it is commonly seen. Insulin resistance is present in both the normal and overweight. Luteinizing hormone (LH) is usually increased and ovarian overproduction of testosterone, probably through stimulation of the ovary by insulin-like growth factor-1, is a main source of testosterone. The factors responsible for this association are not understood.

Growth Hormone

Lean body mass is decreased and fat mass is increased in adults who are deficient in growth hormone (GH) when compared with those who have normal GH secretion. Growth hormone replacement reduces body and visceral fat *(8)*. Acromegaly produces the opposite effects with reduced body fat and particularly visceral fat. Treatment of acromegaly, which lowers GH, increases body fat and vis-

Table 3
Features of Polycystic Ovary Syndrome[a]

Oligomenorrhea/amenorrhea
Hirsutism
Polycystic ovaries
↑ LH/FSH
↑ Testosterone/↓ SHBG
Insulin resistance
Normal IGF-2
↓ IGF-2 binding protein

[a]LH, Luteinizing hormone; FSH, follicle-stimulating hormone; SHBG, sex hormone binding globulin; IGF-2, insulin-like growth factor-2

Table 4
Drugs That Increase Body Weight

Antipsychotics
Phenothiazines and butyrophenones
Chlorpromazine>thioridazine>>trifluoperazine=mesoridazine>promazine>mepazine>perphenazine prochlorperazine>clozapine>olanzepine>quetiapine=haloperidol=loxapine=ziprasidone
Antidepressants
Amitriptyline>imipramine=doxepin=phenelzine amoxapine=desipramine=trazodone=tranylcypromine
Lithium
Antiepileptics
Valproate
Carbamazepine
Steroids
Glucocorticoids
Megestrol acetate
Estrogen
Adrenergic antagonists
α_1-Antagonists
β_2-Antagonists weak
Serotonin antagonists
Cyproheptidine
Antidiabetics
Insulin
Sulfonylureas
Thiazolidinediones

ceral fat. The gradual decline in GH with age may be one reason for the increase in visceral fat with age.

Drug-Induced Weight Gain

Several drugs can cause weight gain, including a variety of psychoactive agents and hormones (*see* Table 4). The degree of weight gain is generally not sufficient to cause true obesity, except for occasional patients treated with high-dose corticosteroids. Antipsychotics (phenothiazines and butyrophenones) often cause weight gain. One study found that men hospitalized for mental illness, many of whom were treated with phenothiazines, gained an average of 3.2 kg during a 35-mo stay. Phenothiazine therapy was thought to play a particularly important role in this weight gain. Among other psychotropic drugs, amitriptyline is a tricyclic antidepressant that is likely to cause weight gain and a carbohydrate preference. Lithium has also been implicated in weight gain. Valproate is an antiepileptic drug that acts on the NMDA (GABA) receptor. It causes weight gain in more than half of the patients who take it. Glucocorticoids cause fat accumulation in particular areas, similar to that seen in Cushing syndrome. These changes occur mostly in patients taking prednisone at doses of 10 mg/d or more. Megestrol acetate (Megace®) is a progestin used in women with breast cancer and in patients with autoimmune deficiency syndrome to increase appetite and induce weight gain *(9)*. The increase in weight is fat. The serotonin antagonist cyproheptidine is associated with weight gain. Insulin stimulates appetite probably through hypoglycemia. Weight gain occurs in diabetic patients treated with insulin or with sulfonylureas, which enhance endogenous insulin release. Weight gain is also a problem with insulin-sensitizing thiazolidinediones, but not with metformin. In one large study, the administration of chlorpropamide, glyburide (glibenclamide), or insulin was associated with a 3.5–4.8 kg weight gain at 6 yr vs no change with metformin *(10)*. The effect of insulin was dose dependent. In the Diabetes Control and Complications Trial, the mean increase in weight in patients with insulin-dependent diabetes was 5.1 kg with intensive insulin therapy and 2.4 kg with conventional insulin therapy *(11)*.

Cessation of Smoking

Weight gain is quite common when people stop smoking. This is thought to be mediated, at least in part, by nicotine withdrawal. Weight gain of 1 to 2 kg in the first few weeks is often followed by an additional weight gain of 2 to 3 kg over the next 4–6 mo. Average weight gain is 4 to 5 kg but can be much greater *(12)*. It has been estimated that smoking cessation increases the odds ratio of obesity compared with nonsmokers by 2.4 times in men and 2.0 times in women.

The effect of smoking and smoking cessation on body weight also has been evaluated by comparing identical twin pairs to eliminate genetic and certain environmental factors. Light, moderate, and heavy smokers were an average of 3.2, 2.4, and 4.0 kg lighter than nonsmokers. On the other hand, past smokers had a significantly higher incidence of obesity than their currently smoking siblings (27 vs 20%). Because of the substantial predictability of weight gain following smoking cessation, it has been suggested that an exercise program and decreased caloric intake be recommended to all patients who are planning to stop smoking.

Sedentary Lifestyle

A sedentary lifestyle lowers energy expenditure and promotes weight gain in both animals and humans. Restric-

tion of physical activity in rats causes weight gain, and animals in zoos tend to be heavier than those in the wild. In an affluent society, energy-sparing devices in the workplace and at home reduce energy expenditure and may enhance the tendency to gain weight.

Several additional observations illustrate the importance of decreased energy expenditure in the pathogenesis of weight gain:

1. The highest frequency of overweight occurs in men who have sedentary occupations.
2. Estimates of energy intake and energy expenditure in Great Britain suggest that reduced energy expenditure is more important than increased food intake in causing obesity *(13)*.
3. A study of middle-aged men in the Netherlands found that the decline in energy expenditure accounted for almost all the weight gain *(14)*.
4. According to the Surgeon General's report on physical activity, the percentage of adults in the United States participating in physical activity decreases steadily with age and reduced energy expenditure in adults and children is predictive of weight gain.

Diet

The amount of energy intake relative to energy expenditure is crucial in the development of obesity, but the composition of the diet may also play a role of variable importance in its pathogenesis. There are a variety of settings in which dietary factors become important.

Overeating

Voluntary overeating (repeated ingestion of energy in excess of daily energy needs) can increase body weight in normal weight men and women. When these individuals stop overeating, they invariably lose the excess weight. The use of overeating to study the consequences of food ingestion has shown the importance of genetic factors in the pattern of weight gain and subsequent loss *(15)*.

Japanese sumo wrestlers, who eat large quantities of food twice a day for many years along with maintaining a very active training schedule, have low visceral fat relative to their total weight. When wrestlers' active careers end, however, they tend to remain overweight and have a high probability of developing diabetes mellitus.

Restrained Eating

A pattern of conscious limitation of food intake is termed *restrained eating (16)*. It is a common pattern in many, if not most, middle-aged women who are of "normal weight." It may also account for the inverse relationship of body weight with social class; women of upper socioeconomic status (SES) often use restrained eating to maintain their weight. In a weight loss clinic, higher restraint scores were associated with lower body weights. Weight loss was associated with a significant increase in restraint, indicating that higher levels of conscious control are at work to maintain lower weight. The greater the increase in restraint the greater the weight loss, but the higher the risk of a lapse or loss of control and overeating.

Frequency of Eating

The relationship between the frequency of meals and the development of obesity is unsettled. There are many anecdotal reports that overweight persons eat less often than normal weight persons, but documentation is difficult. However, the frequency of eating does change lipid and glucose metabolism. When normal individuals eat several small meals a day, their serum cholesterol concentrations are lower than when they eat a few large meals each day. Similarly, mean blood glucose concentrations are lower when meals are frequent *(17)*. One explanation for the effects of frequent small meals vs a few large meals could be the greater insulin secretion associated with larger meal sizes.

Dietary Fat Intake

Epidemiologic data suggest that a diet high in fat is associated with obesity. The relative weight in several populations, for example, is directly related to the percentage of fat in the diet *(18)*. A high-fat diet introduces palatable, often high-fat foods into the diet and a corresponding increase in energy density of the diet, i.e., lesser weight of food for the same number of calories. This makes overconsumption more likely. Differences in the storage capacity in the body for various macronutrients may also play a role. Carbohydrate stores are limited by the capacity to store glycogen in liver and muscle and they need to be replenished frequently in contrast to fat stores, which are more than 100 times the daily intake of fat. This difference in storage capacity makes eating carbohydrates a more important physiologic need that may lead to overeating when carbohydrates are limited in the diet and carbohydrate oxidation cannot be reduced sufficiently.

Night-Eating Syndrome

The night-eating syndrome is defined as the consumption of at least 25% (and usually more than 50%) of energy between the evening meal and the next morning. It is a common pattern of disturbed eating in the obese. It is related to sleep disturbances and may be a component of sleep apnea, in which daytime somnolence and nocturnal wakefulness are common.

Binge-Eating Disorder

Binge-eating disorder is a psychiatric illness characterized by uncontrolled episodes of eating that usually occur in the evening *(19)*. The patient may respond to treatment with drugs that modulate serotonin or its reuptake.

Infant-Feeding Practices

Infant feeding practices have been related to obesity. Infants fed an artificial formula tend to gain more weight

Table 5
Genetic Syndromesof Obesity with Hypogonadism and Mental Retardation

	Syndrome				
Feature	Prader-Willi	Bardet-Biedl	Ahlstrom	Cohen	Carpenter
Reproductive status	1° Hypogonadism	1° Hypogonadism	Hypogonadism in males but not in females	Normalgonadal function or hypogonadotropic hypogonadism	2° Hypogonadism
Other features	Enamel hypoplasia Hyperphagia Temper tantrums Nasal speech			Dysplastic ears Delayed puberty	
Mental retardation	Mild to moderate		Normal I.Q.	Mild	Slight

per unit time than breast-fed infants. However, there does not appear to be a correlation between the type of infant feeding (breast or bottle) and the rate of weight gain in later childhood, because weight in the first year has weak predictive value for weight in adolescents unless the child had an overweight parent (*see* next section).

PROGRESSIVE HYPERPHAGIC OBESITY

A small number of people begin to be overweight in childhood and then have unrelenting weight gain, usually surpassing 140 kg (300 lb) by age 30 yr. A recent death of a 13-yr-old weighing 310 kg (680 lb) illustrates the maximal rate of weight gain of nearly 25 kg/yr. These individuals gain about the same amount of weight year after year. Because approx 22 kcal/kg is required to maintain an extra kilogram of body weight in an obese individual, the energy requirements in these patients must increase year by year, with the weight gain being driven by excess dietary caloric intake *(2)*.

Psychologic and Social Factors

Psychologic factors in the development of obesity are widely recognized, although attempts to define a specific personality type that causes obesity has been unsuccessful. One condition that has been linked to weight gain is seasonal affective disorder, which refers to the depression observed during the winter season in people living in the north, where days are short. These patients tend to have an increase in body weight in the winter that can be effectively treated by providing artificial lighting of higher intensity during the winter months.

Socioeconomic and Ethnic Factors

Obesity is more prevalent in lower socioeconomic groups in the United States and elsewhere. The inverse relation of SES and overweight is found in both adults and children. In the Minnesota Heart Study, SES and BMI were inversely related. People in the higher SES groups were more concerned with healthy weight-control practices, including exercise, and tended to eat less fat. In the Growth and Health Study of the National Heart, Lung and Blood Institute, there was a strong association between SES and overweight in Caucasian girls ages 9 to 10 and their mothers, but no association between SES and overweight in African-American girls. Among adult women, the association of SES and overweight is much stronger in Caucasian women than in African-American women. African-American women of all ages are more obese than are Caucasian women. African-American men are less obese than Caucasian men and socioeconomic factors are much less evident in men. The prevalence of obesity in Hispanic men and women is higher than in Caucasian men and women. The basis for these ethnic differences is unclear. In men the socioeconomic effects of obesity are weak or absent. This gender difference and the higher prevalence rates for overweight in women suggest important interactions of gender with many factors that influence body fat and fat distribution. The reason for this association is not known.

Genetic and Congenital Disorders

Genetic factors influence obesity in two ways. First, there are genes and chromosomal abnormalities that are primary factors in the development of obesity. Second, there are genes on which environmental factors act to cause obesity. Obesity is also a characteristic feature of several congenital disorders which are described in Table 5 *(20)*. All of these syndromes display some degree of reproductive impairment and mental retardation. The Prader-Willi syndrome is the most common syndrome in this group. This abnormality on chromosome 15q11.2 is transmitted paternally and produces an infant that is described as a "floppy baby" who usually has trouble feeding. The obesity begins at about age 2 and is associated with overeating. Hypogonadism and mental retardation round out the clinical features of this syndrome.

Natural History of Obesity

Individuals can become overweight at any age, but there are certain times when this is more common. At birth there is little if any distinction in weight between those who will and will not become obese later in life, except for the in-

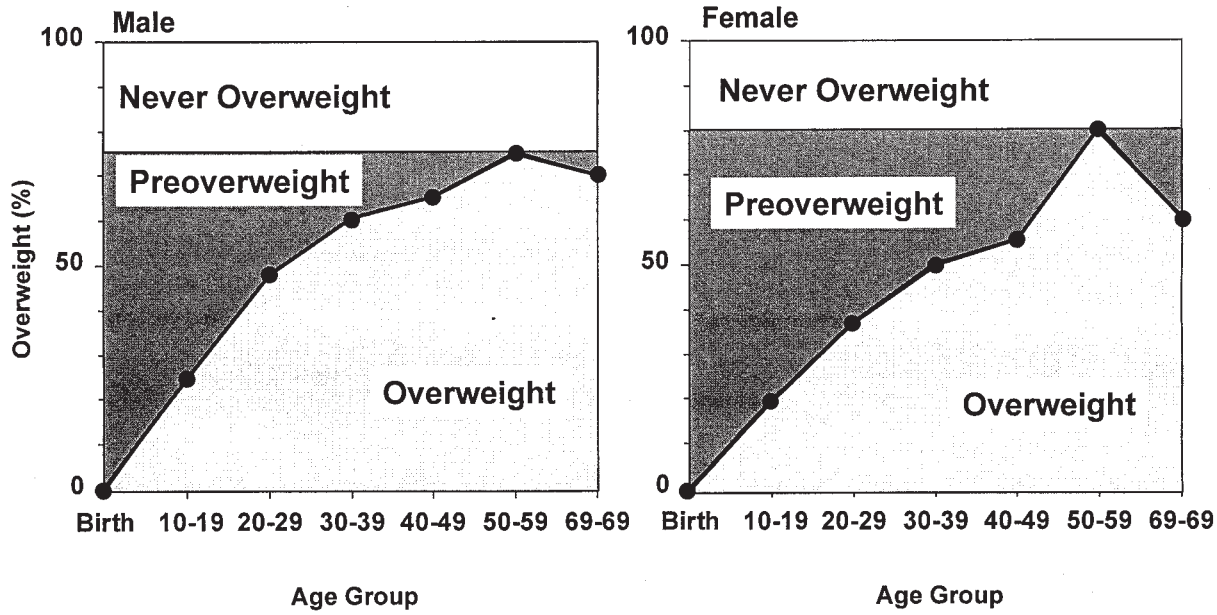

Fig. 2. Natural history of overweight. Because many nonoverweight babies will become overweight during their life, this group is labeled preoverweight. About one-third of those who become overweight do so before age 20 yr and two-thirds after. The remainder are never overweight.

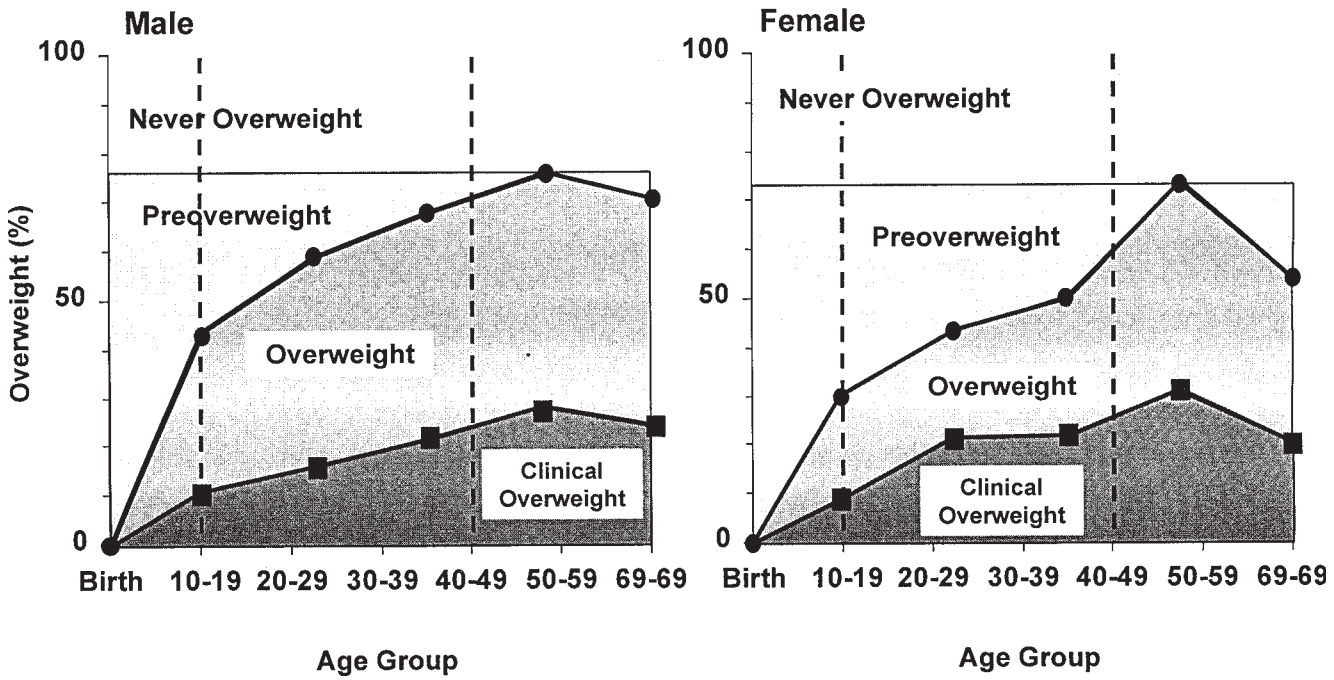

Fig. 3. Comparison of preoverweight, preclinical overweight, and clinical overweight as a percentage of the respective age groups.

fants of diabetic mothers, in whom there is an increased likelihood of obesity later in life *(1,2)*. Thus, at birth there is a large pool of individuals who will eventually become overweight and a smaller group who will never become overweight. In Fig. 2, I have labeled these pools "preoverweight" and "never overweight," using the National Center for Health Statistics data for prevalence of BMI >25 kg/m^2 as the solid line. Several surveys suggest that one-third of overweight adults become overweight before age 20 and two-thirds do so after that age. Thus, if 75–80% of adults will become overweight at some time in their life, 20–25% of the population will display their overweight before age 20 and 50% after age 20.

Of overweight individuals, some will develop illnesses such as diabetes, hypertension, gallbladder disease, or a metabolic syndrome. It is thus possible to divide the popula-

Table 6
Predictors of Weight Gain

1. Parental overweight
2. Lower SES
3. Smoking cessation
4. Low level of physical activity
5. Low metabolic rate
6. Childhood overweight
7. Heavy babies
8. Lack of maternal knowledge of child's habits of eating sweets
9. Recent marriage
10. Multiple births

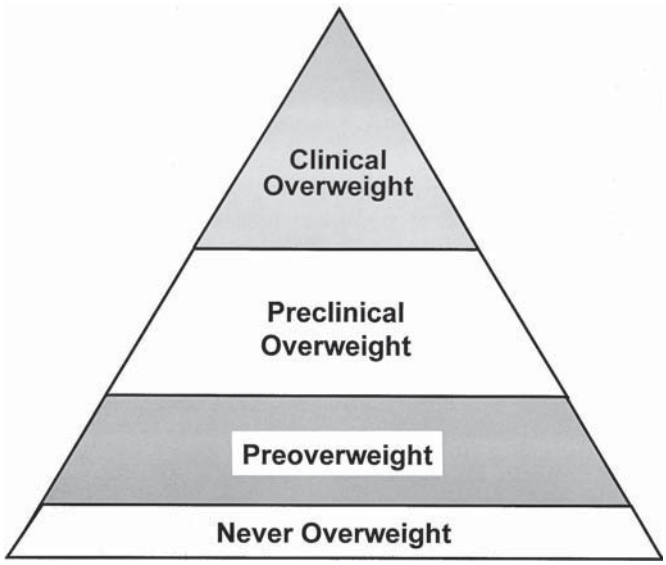

Fig. 4. Progression to clinical overweight. Many individuals who become overweight will not have diabetes, hypertension, or other diseases. These are classed preclinical overweight. Those who develop clinical disease are clinically overweight.

tion into five subgroups: never overweight, preoverweight, preclinical overweight, overweight, and clinical overweight (*see* Fig. 3). For individuals who will never become overweight, we may only be able to identify them in retrospect. Because most preoverweight people will become overweight during their lifetime, it is important to have as much insight into the risk factors as possible. Table 6 lists some predictors of weight gain. These predictors fall into two broad groups: demographic and metabolic. The second group is the preoverweight individuals who have a BMI <25 kg/m^2. When individuals become overweight without clinically significant problems, they manifest what I call "preclinical overweight." With the passage of time or a further increase in weight, they may show clinical signs of diabetes, hypertension, gallbladder disease, or dyslipidemia. I call this group "clinical overweight." The relationship of one to the other may be depicted as a pyramid (Fig. 4). At the base is the reservoir of never overweight and preoverweight individuals, many of whom will become overweight in their adult life. Some of these will, in turn, show signs of clinical disease and become clinically overweight. This approach to the natural history of overweight can be used in the approach to treatment of obesity.

Overweight Developing Before Age 10 (Prepubertal)

Prenatal Factors

Caloric intake by the mother may influence body size, shape, and later body composition. Birth weights of identical and fraternal twins have the same correlation ($r = 0.63$), indicating that birth weight does not predict future obesity. In the first years of life, the correlation of body weight among identical twins begins to converge rapidly, becoming much closer together ($r = 0.9$) whereas dizygotic twins diverge during this same period ($r = 0.5$). Infants born to diabetic mothers have a higher risk of being overweight as children and adults *(1)*. Infants who are small-for-dates, short, or have a small head circumferences are at higher risk of developing abdominal fatness and other comorbidities associated with obesity later in life *(21)*.

Infancy Through Age 3

Body weight triples and body fat normally doubles in the first year of life. This increase in body fat is an important predictor of overweight only in infants with overweight parents. An infant above the 85th percentile at age 1–3 has a fourfold increased risk of adult overweight if either parent is overweight compared with nonoverweight infants. If neither parent is overweight, this infantile overweight does not predict overweight in early adult life (Fig. 5). These observations are similar to the older observations suggesting that 80% of children with two overweight parents were at risk for adult obesity, 40% of those with one overweight parent, and <10% if neither parent was overweight *(1,2)*.

Childhood Obesity from Age 3 to 10

An important period of childhood for developing overweight occurs between the ages of 3 and 10 yr. *Adiposity rebound* is a term used to describe the increase in weight of many children as socialization begins at about age 5–7. About half of the overweight grade school children remain overweight as adults. Moreover, the risk of overweight in adulthood is at least twice as great for overweight children as nonoverweight children. The risk is 3 to 10 times higher if the child's weight is above the 95th percentile for their age. Parental overweight plays a strong role in this group too. Nearly 75% of overweight children ages 3–10 remain overweight in early adulthood if they have one or more overweight parents compared with 25–50% if neither parent is overweight. Overweight 3–10-yr-olds with an over-

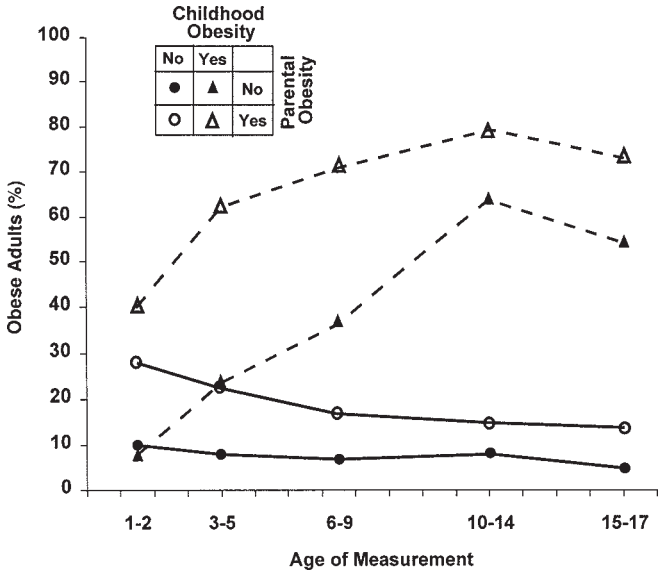

Fig. 5. Effect of parental and childhood weight on weight status during early adulthood. The percentage of overweight adults is plotted in relation to whether the child was overweight at each age and whether the child had one or both overweight parents at the same time. When one parent was overweight, the nonoverweight 1- to 2-yr-old child had a much greater risk of becoming overweight as an adult compared with the child of parents neither of whom was overweight. This effect of parental weight status was no longer evident by age 7–9. The effect of parental overweight declined as children entered adolescence and the tracking of adolescent overweight into early adulthood became much stronger. (Adopted from ref. *22*.).

weight parent is thus an ideal group in whom to utilize behavioral techniques. When there is progressive deviation of body weight from the upper limits of normal in this age group, I label it "progressive obesity" *(2)*; this is usually severe and lifelong, and is associated with an increase in the number of fat cells.

Overweight Developing from Adolescence to Midlife

Adolescence

Weight in adolescence becomes a progressively better predictor of adult weight status *(23)* (Fig. 5). In a 55-yr follow-up of adolescents, the weight status in adolescence predicted later adverse health events *(24)*. Those adolescents who are above the 95th percentile had a 5- to 20-fold greater likelihood of being overweight in adulthood. In contrast with younger ages, parental overweight was less important, or had already had its effect. Whereas 70–80% of overweight adolescents with an overweight parent were overweight as young adults, the numbers were only modestly lower (54–60%) for overweight adolescents without overweight parents. In spite of the importance of childhood and adolescent weight status, however, it remains clear that most overweight individuals develop their problem in adult life *(1,2)*.

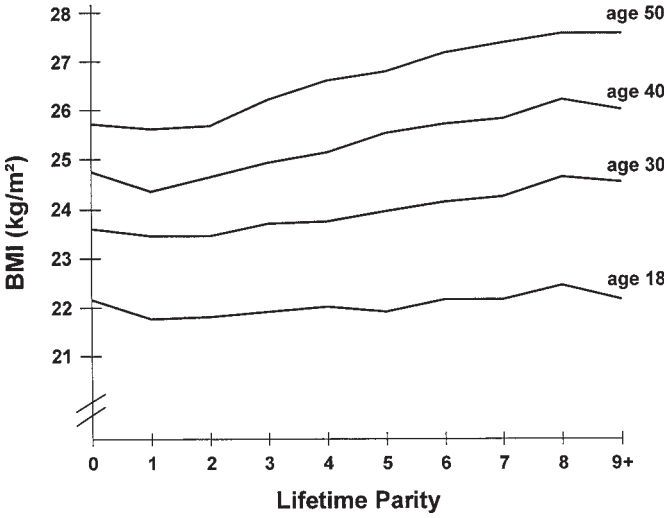

Fig. 6. Effect of pregnancy on weight gain. Weight status shifts further with age in women having more children *(26)*.

Adult Women

Most overweight women gain their excess weight after puberty. This weight gain may be precipitated by several events, including pregnancy, oral contraceptive use, and menopause.

Pregnancy

Weight gain during pregnancy, and the effect of pregnancy on subsequent weight gain, are important events in the weight gain history of women. A few women gain a considerable amount of weight during pregnancy, occasionally more than 50 kg. Pregnancy itself may leave a legacy of increased weight as suggested by one study that evaluated women prospectively between the ages of 18 and 30 *(25)*. Women who remained nulliparous ($n = 925$) were compared with women who had a single pregnancy of 28 wk of duration during that period and who were at least 12 mo postpartum. The primiparas gained 2 to 3 kg more weight and had a greater increase in waist-to-hip ratio (WHR) compared with the nulliparas during this period. As shown in Fig. 6, overweight at each decade increased with increasing parity *(26)*. The overall risk of weight gain associated with childbearing after age 25, however, is quite modest for women in the United States *(27)*.

Oral Contraceptive Use

Oral contraceptive use may initiate weight gain in some women, although this effect is diminished with low-dose estrogen pills. One study evaluated 49 healthy women initiating treatment with low-dose oral contraceptives (30 μg of ethinyl estradiol plus 75 μg of gestodene). Anthropometric measurements before and after the initiation of this formulation were used to compare 31 age- and weight-matched women *(28)*. Baseline BMI, percentage of fat, percentage of water, and WHR did not change significantly after six

Table 7
Clinical and Laboratory Data for Evaluating the Overweight Patient

Name: _____ Identifying Number: _____

Age: _____ Today's Date: _____ (month / day / year)

Date of Birth: _____ (month / day / year)

MEASURED DATA			DERIVED DATA	
Height (in or cm)	in	cm	BMI (kg/m^2)	
Weight (lb or kg)	lb	kg	Weight Gain Since Age 20	
Weight at Age 20	lb	kg	Waist Circumference / Hip Circumference (WHR)	
Waist Circumference	in	cm		
Hip Circumference	in	cm		
Blood Pressure (mmHg)	syst	diast		
Triglycerides	mg/dL	mmol		
HDL-Cholesterol	mg/dL	mmol		
Fasting Glucose	mg/dl	mmol		
Sleep Apnea				
Medications that Increase Weight	Y	N		
Major Etiologic Cause (if known)				

cycles in the birth control pill users. A similar number of women gained weight in both groups (30.6% of users, 35.4% of controls); the typical weight gain in the pill user group was only 0.5 kg. But the small weight gain in these women was owing to the accumulation of fat, not body water. Approximately 20% of women in both groups lost weight.

Menopause

Weight gain and changes in fat distribution occur after menopause. The decline in estrogen and progesterone secretion alters fat cell biology so that central fat deposition increases. Central or abdominal fat deposition (as estimated clinically from waist circumference or WHR) is an important determinant of cardiovascular risk.

Estrogen replacement therapy does not prevent the weight gain, although it may minimize fat redistribution *(29)*. A prospective study of 63 early postmenopausal women compared 34 who initiated continuous estrogen and progesterone therapy with the remaining women who refused it. Body weight and fat mass increased significantly in both the treatment (73.2–75.6 kg) and control groups (71.5–73.5 kg). However, WHR increased significantly only in the control group (0.80–0.85). Caloric and macronutrient intake did not change in either group. A 2-yr trial with estrogen in postmenopausal women also showed an increase in body fat.

Adult Men

The transition from an active lifestyle during the teens and early twenties to a more sedentary lifestyle thereafter is associated with weight gain in many men. A rise in body weight continues through the adult years until the sixth decade (*see* Figs. 2 and 3). After ages 55–64, relative weight remains stable and then begins to decline. There is evidence from the Framingham study and studies of men in the armed services that men have become progressively heavier for height during the twentieth century.

Steps in Evaluating the Overweight Patient

To evaluate overweight patients, one needs both clinical and laboratory information. The criteria recommended here are in line with the U.S. Preventive Services Task Force *(30)*, but rely more heavily on the American Obesity Association *(31)*, the World Health Organization reports *(31)*, and the National Heart Lung and Blood Institute (NHLBI) report. The focus of this approach is to characterize the type of obesity and to emphasize preventing its progression. This approach assumes that there are many types of overweight individuals with differing degrees of associated risk. Some types of overweight can be separated from one another and the relative risks identified. The importance of evaluating

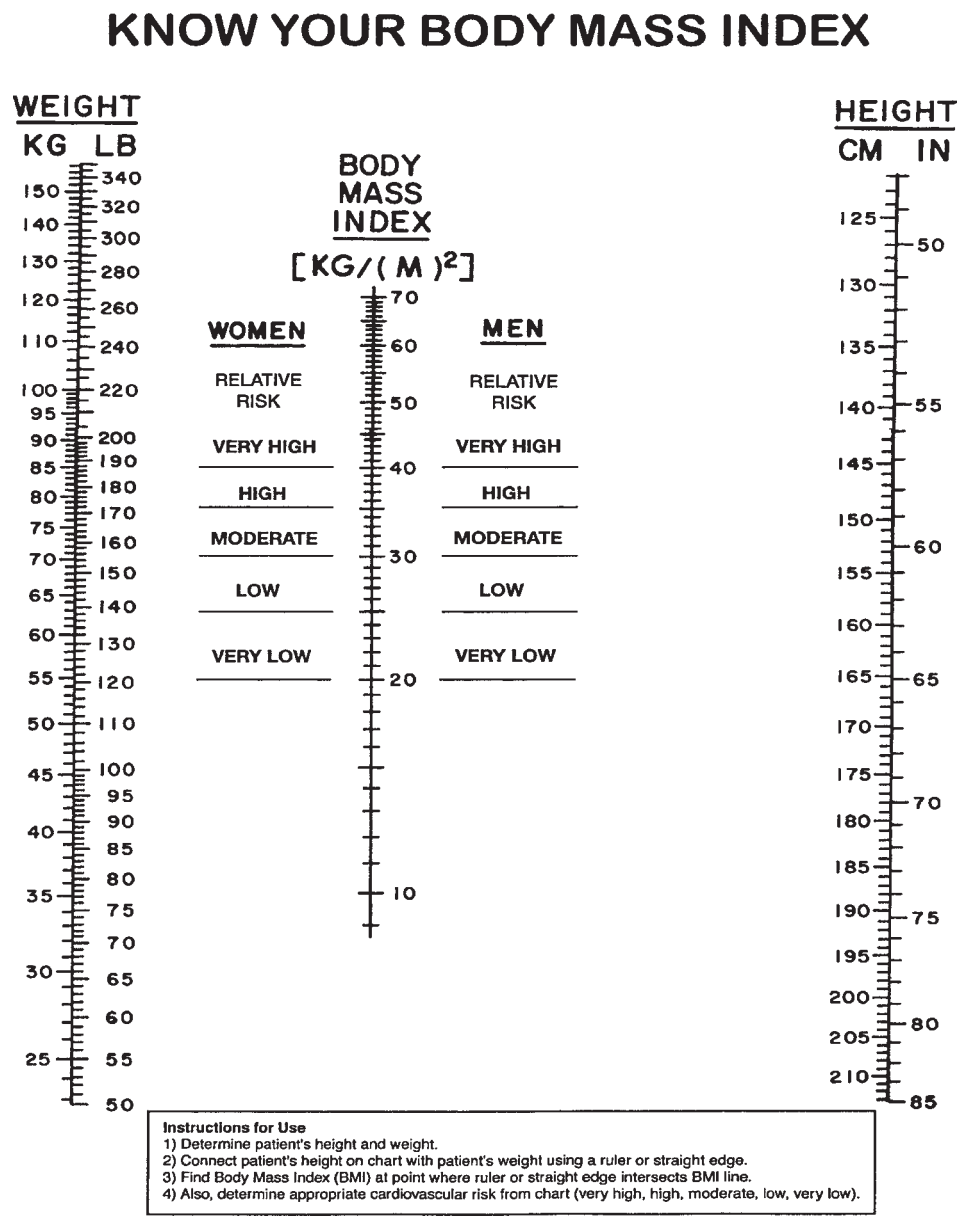

Fig. 7. Nomogram for determining BMI. To use this nomogram, place a ruler or other straight edge between the body weight in kilograms or pounds (without clothes) located on the left-hand line and the height in centimeters or in inches (without shoes) located on the right-hand line. The BMI is read from the middle of the scale and is in metric units. (Copyright 1978 George A. Bray. Used with permission.)

all types of overweight has increased as the epidemic of obesity has worsened and the number of potential treatments has increased.

Several reports have been prepared to provide guidance for this evaluation. These reports come from the American Obesity Association *(32)*, the American Association of Clinical Endocrinologists *(33)*, Scottish Intercollegiate Guidelines Network *(34)*, the World Health Organization Consultation *(30)*, and the National Heart, Lung and Blood Institute *(35)*.

Body Weight

Table 7 provides a form to record relevant clinical and laboratory data when evaluating an overweight patient, and it will help categorize them as preoverweight, preclinical overweight, or clinical overweight. Accurate measurement of height and weight is the initial step in the clinical assessment of an overweight individual *(36,37)*. The most practical single method to evaluate the degree of overweight from weight and height is the BMI. This index is calculated as the body weight (kilograms) divided by the stature (height [meters]) squared (weight/[height]^2) and can be obtained from the nomogram in Fig. 7 or from Table 8. The BMI is well correlated with body fat and is relatively unaffected by height. The BMI shows a curvilinear relation to risk (Fig. 8). From this Fig. 8 several levels of risk can be identified. These cut points are derived from data collected on Caucasians. Whether they apply accurately to other eth-

Table 8
Body Mass Index[a]

	Good Weights						Increasing Risk														
	BMI																				
Height	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

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nic groups is not yet clear, but until other criteria become available, they are the best we have. Data in Hispanic and African Americans suggest that increased body fat produces a greater risk of diabetes, but less impact on heart disease. Fig. 8 also identifies cut points to separate preoverweight from preclinical overweight and clinical overweight using BMI alone. After treatment begins, regular measurement of body weight is one important way to follow the progress of any treatment program.

Regional Fat Distribution

Visceral fat and central fat can be evaluated by several methods. The most accurate are the CT and MRI scan, but these are expensive and not generally available for this purpose. The waist circumference or the WHR are the most practical clinical alternatives. The waist circumference is measured with a flexible tape placed in a horizontal plane at the level of the natural waistline or narrowest part of the torso as seen from the anterior view *(37)*. The hip circumference is measured in the horizontal plane at the level of maximal circumference including the maximum extension of the buttocks posteriorly. A nomogram for calculating WHR is shown in Fig. 9. Figure 10 displays the risk associated with WHR across various ages.

Table 9 shows the risk associated with different levels of waist circumference or WHR. Measurement of changing waist circumference is a good tool to use in following the progress of weight loss. It is particularly valuable when patients become more physically active. Physical activity may slow loss of muscle mass and thus slow weight loss while fat is continuing to be mobilized. The waist circumference can help in making this distinction. The waist circumferences for men and women that are equivalent to WHR values for low, moderate, and high risk are presented in Table 9. As with BMI, the relationship of central fat to risk factors for health varies between populations as well as within them. Japanese-Americans and Indians from South Asia have relatively more visceral fat and are thus at higher risk for a given BMI or total body fat than Caucasians living in the same region. Thus, the risk assigned to the waist circumference or WHR in Table 9 must be tempered by the group to which it is applied. Even though the BMI is <25 kg/m^2, central fat may be increased, and, thus, adjustment of BMI for central adiposity is important particularly in the BMI range of 22–29 kg/m^2.

Weight Gain

Weight gain is associated with increased risk to health; Table 10 presents this risk. Three categories of weight gain are identified: <5 kg (<11 lb), 5–10 kg (11–22 lb), and

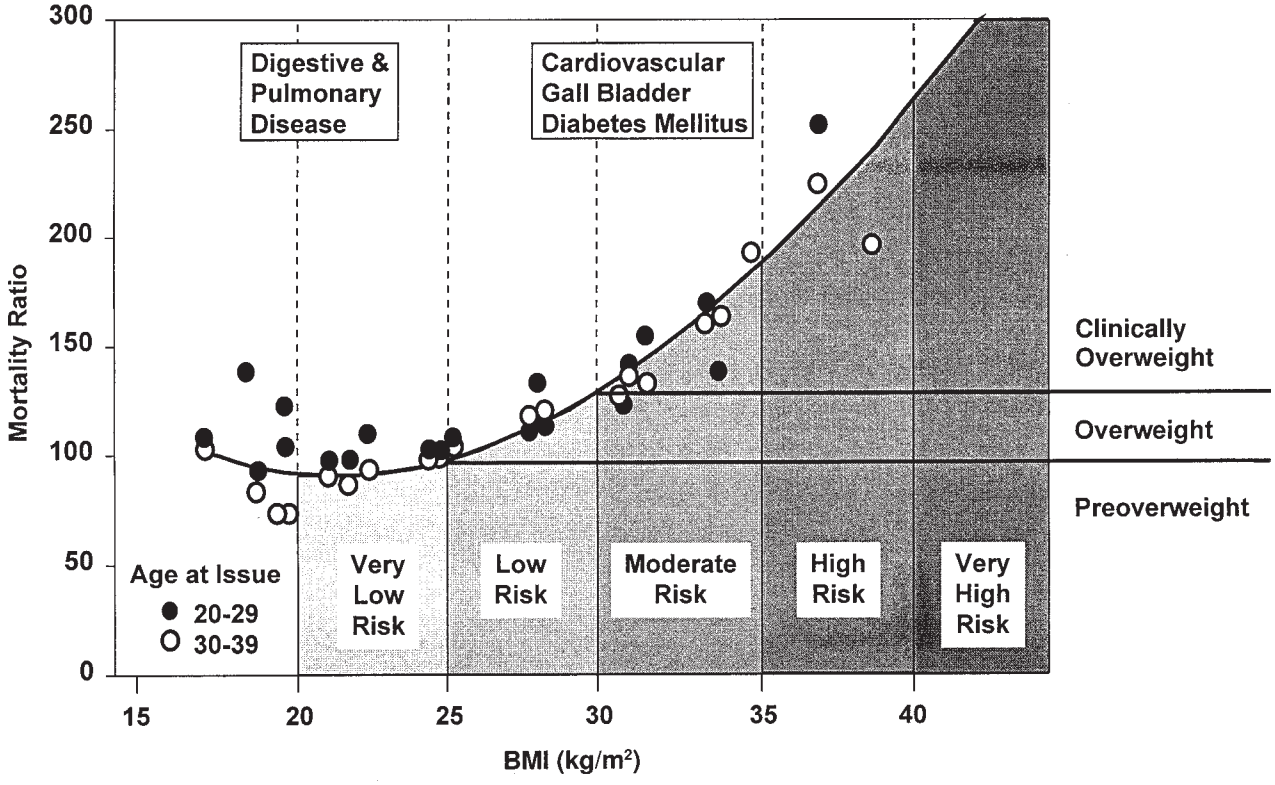

Fig. 8. Relative risk and BMI. The categories of risk are shown at the bottom and those for overweight are shown on the right. Individuals with a BMI <25 are called preoverweight, those with a BMI of 25–30 are preclinical overweight, and those above 30 are clinically overweight.

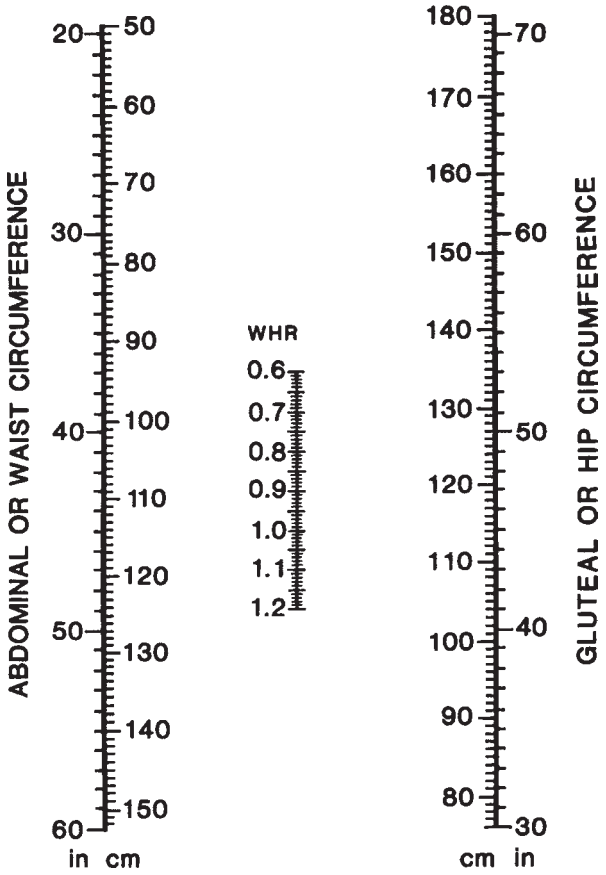

Fig. 9. Nomogram for determining WHR. Place a straight edge between the column for waist circumference and the column for hip circumference and read the ratio from the point where this straight edge crosses the WHR line. The waist circumference is the smallest circumference below the rib cage and above the umbilicus, and the hip circumference is taken as the largest circumference at posterior extension of the buttocks. (Copyright 1987 George A. Bray. Reproduced with permission.)

>10 kg (>22 lb). The adjusted score will be used later to calculate a risk-adjusted weight index.

Sedentary Lifestyle

A sedentary lifestyle also increases the risk of early death. An individual with no

regular physical activity is at higher risk than an individual with modest levels of physical activity.

Etiologic Factors

The presence of etiologic factors and the natural history of obesity should be identified if possible *(38,39)*. They should be noted in Table 7 if they are identified. The algorithm in Fig. 11 will help identify etiologic factors *(40)*. Questions begin in the upper left-hand corner and proceed via the appropriate arrows. At each point a positive answer to the question leads to suggestions for clinical evaluation. For example, once the presence of overweight has been

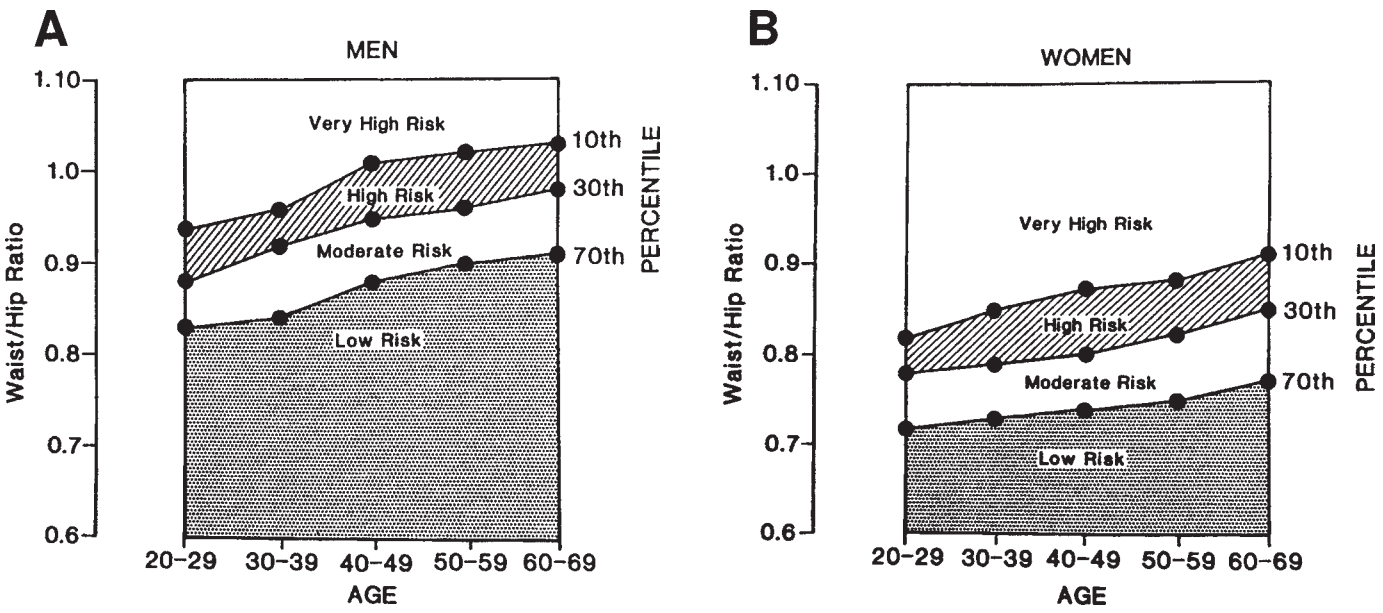

Fig. 10. Percentiles for fat distribution in men and women. The percentiles for the ratio of abdominal circumference to hip circumference are depicted for men **(A)** and women **(B)** by age groups. The relative risk for these percentiles is indicated based on the available information. (Plotted from tabular data in the *Canadian Standardized Test of Fitness,* 3rd ed. 1986. Copyright 1987. George A. Bray.)

Table 9
Risk Associated with Different Levels of Central Fat[a]

	Risk		
BMI adjustment if initial BMI is between 22 and 29	Low (0)	Moderate risk score (+2)	High (+4)
Men			
Waist circumference (in.)	<37	37–40	>40
Waist circumference (cm)	<94	94–102	>102
WHR	<0.90	0.90–1.00	>1.00
Women			
Waist circumference (in.)	<32	32–35	>35
Waist circumference (cm)	<80	80–88	>88
WHR	<0.75	0.75–0.85	>0.85

[a]This evaluation and adjustment of BMI for the added risk of central fat is done for individuals with a BMI <30 kg/m^2.

Table 10
Risk Associated with Weight Gain

	Weight gain since Age 20 (kg)		
	<5	5–10	>10
Risk	Low	Moderate	High
Risk score	0	+1	+2

established, the possibility of hypertension is addressed. If hypertension is present, you should search for clinical signs of Cushing syndrome. If these are present, a urinary-free cortisol test is recommended to be followed by dexamethasone-CRH test if indicated. If Cushing syndrome is not suspected, a hypertension workup is suggested. In turn, the algorithm directs you to search for clinical clues of hypothyroidism, glucose intolerance, dyslipidemia, hypoventilation sleep-apnea syndrome, central nervous system lesions, polycystic ovary syndrome, and congestive heart failure.

Acanthosis nigicans deserves a brief comment. This is a clinical condition with increased pigmentation in the folds of the neck, along the exterior surface of the distal extremities and over the knuckles. It may signify increased insulin resistance or malignancy, and this should be evaluated.

Laboratory Evaluation

Several laboratory tests are outlined in the algorithm in Fig. 11. These include measurements of metabolic fitness,

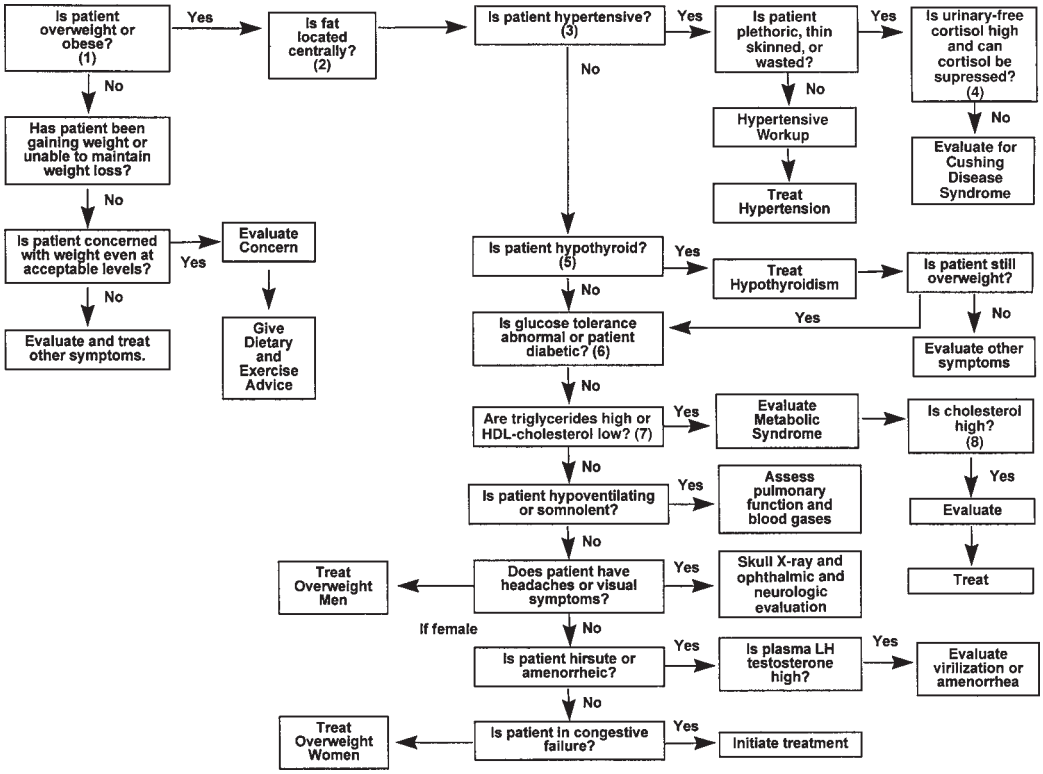

Fig. 11. Algorithm for evaluating the overweight individual. The following numbered list corresponds to the numbers in the figure:

1. Overweight is defined as BMI between 25 and 30 kg/m^2; obesity as a BMI >30 kg/m^2.
2. Fat distribution is defined by waist circumference or the WHR (Table 10).
3. Blood pressure readings are taken with a large cuff that encircles 75% of the arm.
4. Dexamethasone suppression test: Suppression is defined as cortisol <3 μg/dL (80 nmol/L) at 8:00 am, 9 h after 1 mg of dexamethasone orally. If not suppressed, evaluate for Cushing disease.
5. Thyroid function:

	Serum thyroxine (corrected)		Serum thyrotropin
	(μg/dL)	(nmol/L)	(μU/mL = mU/L)
Possible hyperthyroidism	12	154	< 2
Normal	5.5–12.0	71–154	2–7
Borderline hyperthyroidism	4.0–5.5	51–71	7–10
Hypothyroidism	4.0	51	>10

In the presence of severe illness, a low serum thyroxine must be interpreted cautiously; it may be a bad prognostic sign, but not indicative of hypothyroidism unless TSH is elevated.

6. The diagnosis of diabetes in nonpregnant adults is based on the following:
   a. Unequivocal hyperglycemia and classic symptoms of diabetes mellitus.
   b. Fasting venous plasma glucose >126 mg/dL (7.0 mmol/L).
   c. Fasting plasma glucose >126 mg/dL (7.0 mmol/L) at some point between 0 and 2 h, and at 2 h after an oral glucose tolerance test with 75 g of glucose (or for children 1.75 g/kg of ideal body weight, not to exceed 75 g).
7. Triglycerides >2.3 mmol: normal = <200 mg/dL; borderline high = 200–400 mg/dL; high = 400–1000 mg/dL; very high = >1000 mg/dL.
8. LDL cholesterol. Total cholesterol >200 mg/dL determines LDL cholesterol and evaluates risk.

	Diet therapy	Consider drugs	Goal of treatment
Without CHD and <2 risk factors	≥160	≥190	≤160
Without CHD and 2 or more risk factors	≥130	≥160	≤130
With CHD	≥100	≥130	≤100

Risk factors include family history of premature coronary heart disease (CHD) (age <55); hypertension; cigarette smoking; diabetes mellites; and low HDL cholesterol (<0.9 mmol/L or <35 mg/dL). An HDL cholesterol >60 mg/dL (1.6 mmol/L) is protective.

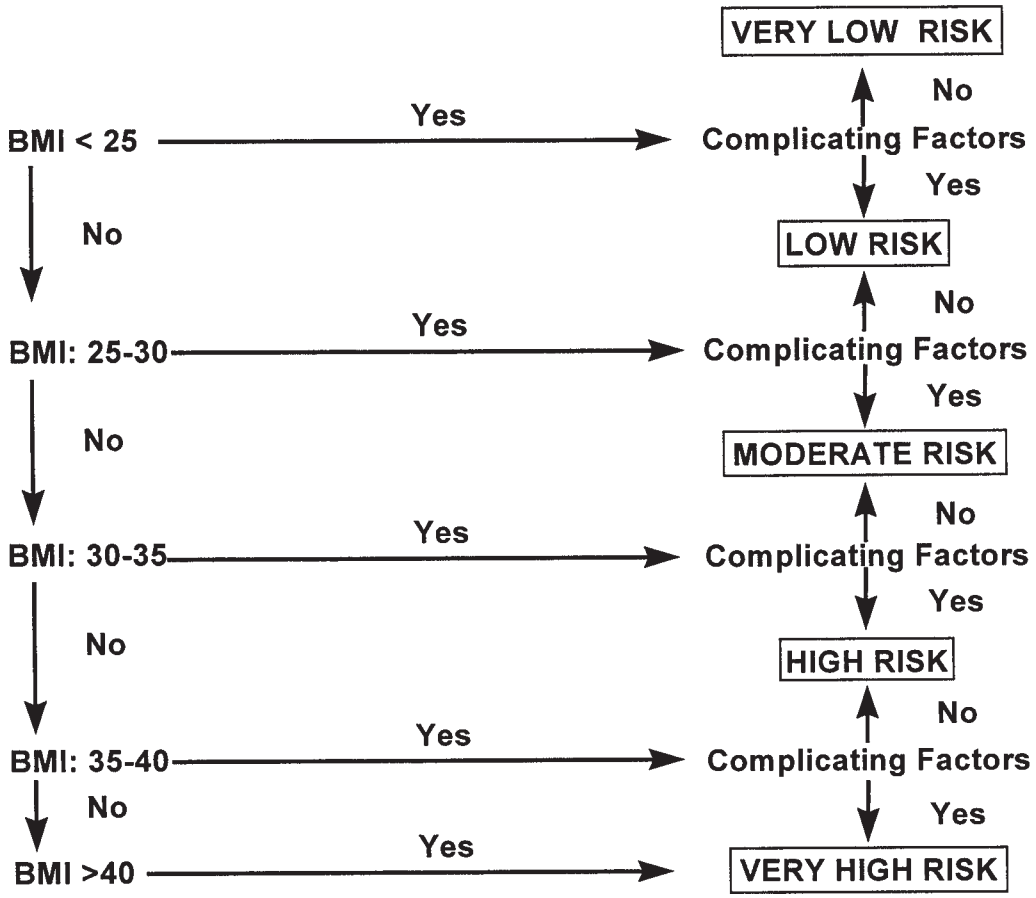

Fig. 12. Algorithm for evaluating the overweight patient developed by Shape-Up America. This algorithm begins with BMI and then proceeds to use items defined in other tables and figures given herein *(32)*.

including fasting glucose, and if indicated by family history, a 2-h glucose; and a lipid panel including total cholesterol and triglycerides, and if indicated by family history or TC values, a measure of low-density lipoprotein (LDL) cholesterol and high-density lipoprotein (HDL) cholesterol. Evaluating thyroid dysfunction with a TSH test can be particularly useful in older women, in whom the prevalence rate of hypothyroidism may be >4%. If the patient is plethoric, hypertensive, and amenorrheic, a urinary cortisol may be indicated. Sleep apnea is common, particularly in obese men, and testing blood gases may be advisable in some patients. To exclude polycystic ovarian syndrome, the LH and follicle-stimulating hormone (FSH) ratio can be determined, and an ultrasound performed. Ultrasound for gallstones may also be indicated. Several of these tests, including fasting glucose, triglycerides, and HDL cholesterol are used in the interpretation of BMI.

Risk-Benefit Assessment

Once the clinical and laboratory workup is complete, you are ready to evaluate the risk associated with an elevated BMI. Several algorithms can be used for this purpose. The basic strategy I propose is shown in Fig. 12. BMI is determined in Table 7. The initial level of BMI provides the first level of risk. Individuals with a BMI <25 kg/m^2 are at very low risk, but nonetheless nearly half of those in this category at age 20–25 will become overweight by age 60–69. Thus, there is a large group of preoverweight individuals for whom preventive strategies are needed. When the BMI is >25 kg/m^2, the risk rises. The presence of complicating factors increases this risk further. It is thus important to try to provide a quantitative estimate of these complicating factors.

The proposed method for adjusting the BMI for other risk factors is shown in Table 11, which provides the adjustment scores for central fat distribution using waist circumference interpreted in light of the comments discussed about ethnic variability. Use the waist circumference to identify the adjustment score and note it on the appropriate line in Table 11. Table 5 also provides space for the adjustment scores for each of several other metabolic and clinical variables related to obesity already recorded in Table 7. The scores for each are recorded on the right-hand side and added to the patient's BMI (top line) to obtain the risk-adjusted weight index.

Treatments for obesity can be risky and this risk can be seen by reviewing adverse outcomes associated with drug treatment during the past 100 yr (Table 12). Medications

Table 11
Table for Obtaining Risk-Adjusted BMI for Metabolic Variables[a]

Score	Adjustment score 0	+2	+4	Adjustment score
BMI	—	—	—	_____
Weight gain since age 18 (kg)	<5	5–15	>15	_____
Triglyceride/HDL-cholesterol (mg/dL)	<5	5–8	>8	_____
Blood pressur (mmHg(	<140/<90	140–160/90–100		
>160/>100		_____		
Fasting glucose (mg/dL)	<95	96–126	>126	_____
Waist circumference (in[cm])	<32 (81)	32–35 (81–89)	>35 (89)	_____
	<37 (94)	37–40 (94–102)	>40 (102)	_____
Sleep apnea	Absent	—	Present	_____
Physical activity	Regular activity	Sedentary	—	_____
			Risk-adjusted index	

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Table 12
Disasters with Drug Treatments of Obesity

Date	Drug	Outcome
1893	Thyroid	Hyperthyroidism
1933	Dinitrophenol	Cataracts, neuropathy
1937	Amphetamine	Addiction
1967	Rainbow pills (digitalis, diuretics)	Deaths
1971	Aminorex	Pulmonary hypertension
1997	Fenfluramine/phentermine	Valvular insufficiency

Table 13
Weight Goals of 60 Overweight Women[a]

Imagined goal	Weight loss to achieve goal kg (%)	Subjects achieving goal (%)
Dream weight	–37.4 (–38)	0
Happy weight	–31.1 (–31)	9
Acceptable weight	–24.9 (–25)	24
Disappointed weight	–17.2 (–17)	20
Below disappointed weight	—	47

[a]Baseline weight = 99.1 kg. Reproduced from ref. *42*.

for obesity must be taken over the long term, hence the emphasis on the risk-benefit and safety of treatments. Each of the major treatments that has been tried has been associated with a major therapeutic disaster. This must temper enthusiasm for new treatments unless the risk is very low. Because obesity is stigmatized in our society, any treatment approved by the Food and Drug Association will be used for cosmetic purposes by people in the preoverweight group who suffer from the stigma of obesity. Thus, drugs to treat obesity must have very high safety profiles.

When the risk-adjusted weight index is determined, the overall risk assessment and treatment goals can be evaluated (Fig. 13). The algorithm in Fig. 13 divides risk-adjusted BMI into 5-U intervals just as done in Fig. 12. The risk, goals, and potential strategies for treatment are noted opposite each of these intervals. Low levels of the comorbid risks reduce the impact of any level of BMI whereas high levels of comorbid risk factors augment the effect of the BMI. With the risk-adjusted weight index, it is possible to select or rank order the treatments that are available.

Patient Readiness

Before initiating any treatment, it is important to establish that the patient is ready to make changes. A series of questions developed by Brownell *(41)* can be used to make this assessment. When counseling patients who are ready to lose weight, it is essential to accommodate their individual needs as well as ethnic factors, age, and other differences. The approach outlined above is not rigid and must be used to help guide clinical decision making and not serve as an alternative to considering individual factors in developing a treatment plan. Because of increasing complications from obesity, more aggressive efforts at therapy should be directed at people in each of the successively higher risk classifications.

Patient-Doctor Expectations

The realities of treatment for obesity often conflict with the patient's expectations. In one weight loss program *(41)*, patients were asked to give the weights they wanted to achieve in several categories from their dream weight through a weight loss that leaves them disappointed. These are listed in column 1 of Table 13 *(42)*. They then participated in a weight loss program. The percentage achieving each goal level is listed in the right hand column. None of the patients achieved their dream weight, which was an

Body Mass Index	No. of Risk Factors	High Waist Circumference	Risk Category	Treat Risk Factors	Consider as Treatment for Overweight				
					Diet	Exercise	Behavior Therapy	Pharmaco-Therapy	Surgery
<25									
	0	No	Very low	No	Advice for Healthy Living			-	-
	0	Yes	Low	No	+	+	+	-	-
	≥1	Y or N	Moderate	Yes	+	+	+	-	-
25 - <29.9									
	0	No	Low	No	+	+	+	-	-
	0	Yes	Moderate	No	+	+	+	±	-
	≥1	Y or N	Moderate	Yes	+	+	+	+	-
30 - <34.9									
	0	Y or N	Moderate	No	+	+	+	+	-
	≥1	Y or N	High	Yes	+	+	+	+	-
>35									
	0	Y or N	High	No	+	+	+	+	±
	≥1	Y or N	Very High	Yes	+	+	+	+	+

Risk Factors:
Diabetes; IGT or Fasting Glucose 110-126 mg/dL
HDL-Cholesterol M < 35 mg/dL TG > 200
F < 45 mg/dL TG > 200
Treated Hypertension or BP >140/ >90

High Waist Circumference
M > 102 cm > 40 in.
F > 88 cm > 35 in.

Figure 13. Risk classification algorithm. The patient is placed in a category based on initial BMI. The presence of complicating factors shift risk up or down. (Copyright 1987 George A. Bray. Used with permission).

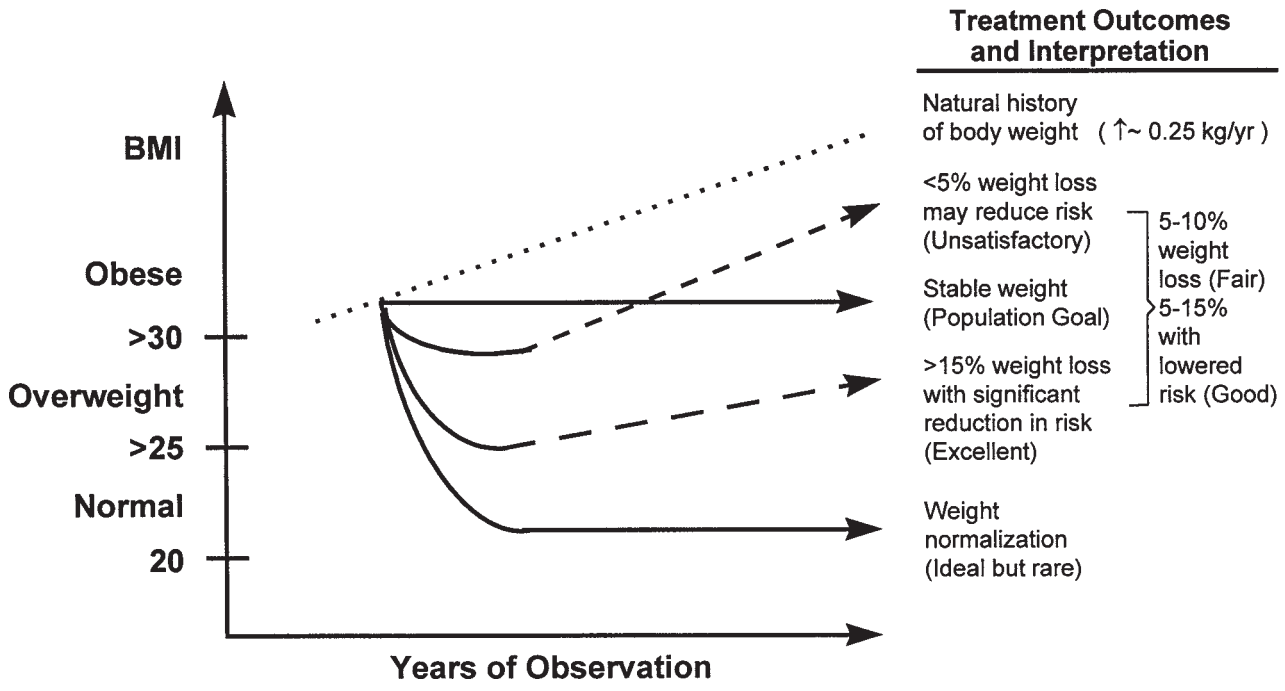

Figure 14. Natural history of weight gain and criteria for successful treatment of overweight. (Adapted from ref. *43*).

average 38% below baseline. Nearly half failed even to achieve a weight loss outcome that would disappoint them. What is desirable from a cosmetic standpoint and what is a realistically achievable goal are almost always at odds. This mismatch between patient expectations and the realities of weight loss provides an important challenge for any therapist and hisor her patient as they begin treatment. A weight loss goal of 5–15% can be achieved by most patients and is a reasonable one.

One complaint about treatments for obesity is that they frequently fail and are thus "no good." An alternative interpretation may be better. Overweight is not a curable disease but can be treated in many ways. When treatments are stopped weight is regained. This is similar to what happens in patients with hypertension who stop their antihypertensive drugs or patients with high cholesterol who stop their hypocholesterolemic drugs. In each case, blood pressure or cholesterol rises. Like overweight, these chronic diseases have not been cured, but palliated.

Criteria for Evaluating Outcomes

Criteria for Weight Loss

Figure 14 shows a model for the natural history of weight change and potential criteria for success *(43)*. For the majority of the population weight rises slowly but inexorably. In a study of 554 subjects ages 30–50 yr with high normal blood pressure, there was an increase of 1.8 ± 5.3 kg at 36 mo follow-up in a usual care setting. The principal preventive strategy should be to stop further weight gain. This alone would dramatically reduce the ravages of weight gain and prevent progression of the epidemic of obesity. For any active treatment program for overweight, a weight loss of <5% would be unsatisfactory. Several levels of efficacy are shown ranging from adequate to ideal. Before initiating treatment with your patients, it is appropriate to review the likely outcome.

An ideal outcome would be a return of body weight to the normal range with no weight gain thereafter. This is rarely achieved and is unrealistic for most patients. Rather, they need guidance in accepting a realistic goal that is usually a loss of 5–15%. A satisfactory outcome is maintenance of a lower body weight over the ensuing years. A good outcome would be a loss of 5–15% and a regain that was no faster than the increase in body weight of the population. Patients should be applauded for achieving this goal. An excellent outcome would be a loss of weight of >15%. An unsatisfactory outcome is a loss of <5% or regain above the projected weight line. In reality, it is difficult to achieve a weight loss of >15% for most patients.

Quality of Life

Quality of life is an important outcome for any patient. This can come in many ways. From the health care perspective, a reduction in comorbidities is a major improvement. Remission of noninsulin-dependent diabetes mellitus or hypertension can reduce costs of treating these conditions as well as delay or prevent the development of disease states. Weight loss can reduce the wear and tear on joints and slow the development of osteoarthritis. Sleep apnea will usually resolve.

Psychosocial improvement is of major importance to the patient. The studies of patients who have achieved long-term weight loss from surgical intervention comment on the improved social and economic function of previously disabled overweight patients.

Loss of 5% or more of initial weight almost always translates into improved mobility, improvement in sleep disturbances, increased exercise tolerance, and heightened self-esteem. A focus on these, rather than cosmetic outcomes, is essential.

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