0733–8651/01 $15.00 + .00

EXERCISE IN WEIGHT MANAGEMENT OF OBESITY

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Obesity has become an important burden for the health system of industrialized countries and must be regarded as a serious public health issue in our time. Obesity is associated with reduced life expectancy,[38] and it is now well recognized that increased body fat is associated with heart disease, stroke, hypertension, dyslipidemia, type 2 diabetes mellitus, gallbladder disease, osteoarthritis, sleep apnea and respiratory problems and numerous cancers (endometrial, breast, prostate and colon).[2, 21] The American Heart Association has stated that obesity is a major modifiable risk factor for heart disease.[40, 42] This article reviews basic regulatory aspects of human adipose tissue metabolism with implications for the cardiologist in terms of exercise prescription and the role of exercise and aerobic physical training in the management of obesity.

EPIDEMIOLOGY

The incidence of obesity in the United States has increased progressively since 1960.[104] In the United States and in Europe, the incidence of being overweight and obese have reached epidemic proportions.[2, 61] Furthermore, in the past decade, the percentage of overweight and obese individuals in the United States and in some countries in Europe has increased to over 50% of adults aged 20 years or older.[61, 104] Childhood obesity is also an alarming problem, and opportunities for physical activity have been lost in the youth.[6] Obesity in the youth may, in part, have been created by structural changes that have reduced the ability to make healthy choices (ie, it is unsafe for children to play outside). As many clinicians have often observed, weight reduction is difficult to achieve and even more difficult to maintain. The reduced-obesity state is a self-perpetuating condition, wherein homeostatic mechanisms attempt to restrain further weight loss.[41] Obesity is a very complex chronic disorder that results from the interaction of genotypic versus environmental factors[2, 19] and involves multifaceted interactions among numerous potential determinants (humoral, neural, metabolic, psychological etc). Of great consequence, the relative risk of diabetes increases by approximately 25% for each additional unit of body mass index (BMI) over 22 kg/m^2,[23] and the practice of regular physical activities has been associated with the prevention of diabetes[45, 78] and decreased mortal-

Support has been provided by grants from The Québec Heart Institute and Le Fond de Recherche en Santé du Québec (PP), the Canadian Institutes for Health Research (J-PD), the Natural Sciences and Engineering Research Council (J-PD), the Canadian Diabetes Association (J-PD), and the Heart and Stroke Foundation of Canada (J-PD).

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ity in obese individuals and those with type 2 diabetes.[62, 111] Likewise, insulin resistance is frequently associated with obesity, and it should always be kept in mind that insulin-resistant individuals are at increased risk of heart disease. It was recently reported in three European cohorts (>17,000 men) followed for over 20 years that nondiabetic men with higher blood glucose had a significantly higher risk of death from cardiovascular and coronary heart disease.[14] Therefore, asymptomatic glucose intolerance should no longer be considered a benign metabolic condition, and features associated with the insulin resistance syndrome should be taken seriously by the medical community. Indeed, data from the Quebec Cardiovascular Study have suggested that hyperinsulinemia resulting from insulin resistance might be an independent risk factor for coronary artery disease.[34] This observation further stresses the importance of exercise and aerobic physical training as effective interventions to increase insulin sensitivity.[35, 87, 93, 102]

Obesity is age-dependent, with most subjects increasing their fat stores when they become older. Aging is associated with a decline in physical activity that contributes to decreased exercise tolerance, decreased lean body mass, and increased fat mass, with alterations in glucose and lipoprotein metabolism. Therefore, while aging, our population is becoming obese. This trend has important public health implications, and proper nonpharmacologic management of obesity and associated comorbidities is mandatory to decrease the burden of obesity on the health system. Unfortunately, data from the 1998 Behavioral Risk Factor Surveillance System (BRFSS) indicate that two thirds of overweight persons trying to lose weight reported using physical activity as a strategy for weight loss, but only one fifth reported being active at the recommended levels.[4, 79]

DEFINITION

Overweight is defined as a body mass index (BMI; weight in kilograms divided by the square of height in meters) of 25 kg/m^2 to 29.9 kg/m^2 and obesity as a BMI $\geq$30 kg/m^2.[2] All overweight and obese adults aged >18 years with a BMI $\geq$25 kg/m^2 are considered at risk for developing cardiovascular comorbidities.[84] However, it is important to emphasize that obesity is characterized by a remarkable metabolic heterogeneity.[27–29] Thus, the challenge for the health care professional and the cardiologist is to identify the overweight/obese individuals "at high risk" of cardiovascular disease (CVD).[84] In this regard, abundant literature published over the last 20 years has emphasized that abdominal obesity was more important than excess fatness as a correlate of the complications that had been in the past considered as the consequence of obesity per se.[20, 26, 59, 90] It has been shown that waist circumference is positively correlated with abdominal fat content and that it is the most practical anthropometric measurement for assessing a patient's abdominal fat content.[86] It has been recently suggested that two simple clinical variables, waist circumference ($\geq$90 cm) and fasting plasma triglyceride concentrations ($\geq$2.0 mmol/L), might be useful as screening tools to unmask men characterized by an atherogenic profile characteristic of the insulin-resistance syndrome.[64] Box 1 shows some of the atherogenic profile features associated with abdominal obesity.

ADIPOSE TISSUE METABOLISM AND CARDIAC ADAPTATION IN OBESITY

Numerous enzyme pathways and hormones are implicated in adipose tissue metabolism.[83] Lipoprotein lipase (LPL) is synthesized in adipose tissue and, by hydrolysing circulating triglyceride-rich lipoproteins, is important in the provision of fatty acids for their uptake and storage as triglycerides

Box 1. Abnormalities Associated with "At Risk" Obesity (Insulin Resistance Syndrome)

- ↑ Triglycerides
- ↓ HDL cholesterol
- ↑ Apolipoprotein B levels
- ↑ Proportion of small, dense LDL
- ↑ Ratio of cholesterol/HDL cholesterol
- ↑ Insulin
- ↑ Glucose intolerance
- ↑ Fibrinogen
- ↑ Factor VII activity
- ↑ Factor VIIIc activity
- ↑ TPA antigen
- ↑ PAI-1 antigen and activity
- ↑ C-reactive protein

(TG).[39] Insulin increases adipose tissue LPL.[83] There are site differences in the regulation of lipolysis in vitro and in vivo in normal-weight subjects.[63] In normal-weight men, adipose tissue LPL activity is higher in the abdominal wall than in the gluteal/femoral region[11]; in women, the opposite has been reported.[11, 15, 89] This may be of clinical importance because these site differences are more marked in women than in men, and might explain why women have more fat in peripheral sites than men. The most important lipolytic hormones in men are the catecholamines, and their lipolytic effect is dependent upon the balance between α and β adrenergic stimulation. The hormonal regulation of lipolysis, in particular the action of catecholamines, is impaired in obesity, and in vivo studies have shown blunted catecholamine induced lipolysis in obese subjects.[24] Thus, resistance to the lipolytic effect of catecholamines and a greater antilipolytic effect of insulin are found in subjects with peripheral as compared to abdominal obesity.[24] However, in obese women[60, 114] and men,[69] fasting adipose tissue LPL activities have not been shown to be different between the abdominal wall and gluteal regions, and the regulation of the enzyme by insulin[114] and exercise[60] is also similar. This is due to increased α-2 adrenoceptor function and decreased β-adrenoceptor expression in the gluteal/femoral fat cells.[63, 71, 91] These results are in contrast to what has been reported in normal-weight individuals, in whom adipose tissue LPL differs between fat depots. Therefore, the alterations in lipolysis associated with obesity favor weight maintenance.

The β-adrenoceptors that mediate vasodilatation in adipose tissue are mainly the β_1-type, whereas in skeletal muscle, the β_2-subtype is mainly responsible for vasodilatation. Although there are differences between blood flow in adipose tissue and other organs, the increment in blood flow with increasing adiposity is not proportional to the increment in adipose tissue mass.[66] Increases in total body fat result in higher total blood flow secondary to the enlarged vascular bed, but in line with this, it is important to keep in mind that the adipose tissue is less vascularized than lean tissue with increasing obesity. Accordingly, the increase in systemic blood flow seen in obesity[74] cannot solely be explained by increased requirements due to adipose tissue perfusion, but most probably occurs by the concomitant increase in lean body mass in these individuals. Higher cardiac output from increased stroke volume and expanded intravascular volume are features of the higher metabolic demands generated by obesity.[55, 74] In obesity, left ventricular filling pressure and volume increase, shifting left ventricular function to the left on the Frank-Starling curve. A decrease in central blood volume accompanies weight reduction and, when present, relief of edema and dyspnea accompanies this improvement.[55, 56] Of importance, because of the need to move excess body weight, at any given level of activity, the cardiac workload is greater for obese subjects than for nonobese individuals.

ADIPOSE TISSUE METABOLISM AND EXERCISE

Whereas α-adrenergic mechanisms regulate lipolysis at rest, β-adrenergic activity controls the lipolytic rate during exercise.[10] Free fatty acid (FFA) availability is maximal at 25% to 40% of $\dot{V}O_2max$, and shifts in energy substrate mobilization and utilization occur as exercise intensity increases, particularly at intensities above 70% to 80% of $\dot{V}O_2max$.[92] Therefore, above a certain degree of intensity, the muscle preferentially operates on glycogen stored in situ. However, aerobic physical exercise involves respiratory and circulatory systems as well as the enzymatic machinery adaptations that facilitate the muscle to work more effectively. When adaptation to endurance exercise has taken place, the muscle is characterized by more oxidative enzyme activity and is now better equipped to work at low intensity for long duration and to use FFA as the main substrate. There are also changes occurring at the site of lipid mobilization in adipose tissue depending on the physical training fitness status.[72] These include a more sensitive mechanism for activation of hepatic sensitive lipase (HSL),[9] and in untrained individuals, an increase in the lipolytic response of adipose tissue to catecholamines after exercise both in vitro[94, 98, 108] and in vivo.[81] Lipolysis is anything but rate limiting during exercise, and is probably in excess of that required.[92] Even if β-blockade reduces the release of FFA from adipocytes, this reduction in energy supply to the exercising muscle is probably not clinically relevant.[103]

COMMON RISK FACTORS ASSOCIATED WITH OBESITY

Dyslipidemia

As we have alluded to previously, some very obese individuals may nevertheless show a fairly normal metabolic risk factor profile, whereas others may present all the features of an atherogenic and diabetogenic metabolic profile[27-29, 64] (see Box 1). Indeed, there is remarkable metabolic heterogeneity among obese subjects, and the presence of visceral obesity generally worsens the metabolic portrait. Accumulation of visceral fat has been associated with type 2 diabetes mellitus, hypertension, and coronary artery disease.[65] For instance, disturbances in lipoprotein metabolism, coagulation systems, plasma insulin-glucose homeostasis and an elevated blood pressure have all been reported in subjects with visceral obesity[22, 22, 48, 67, 97, 97] (see Box 1). The dyslipidemic profile commonly associated with abdominal obesity has been shown to include high TG, low HDL cholesterol and elevated apolipoprotein B concentrations as well as an increased proportion of small, dense LDL. All these features of an atherogenic dyslipidemic profile can be improved by the incorporation of regular exercise in daily life activities.[27, 28, 33] Fat loss through dieting and/or exercise produces comparable and favorable changes in HDL cholesterol and its subfractions HDL_2 and HDL_3 as well as in TG.[43, 112] Furthermore, long-term aerobic exercise training could even normalize the metabolic risk profile of obese subjects despite the fact that subjects were still classified as "obese" at the end of the program.[100] In addition, the improvement in the plasma lipid profile observed with the use of aerobic exercise in patients with type 2 diabetes is also probably mediated mainly through body fat loss.[82, 109] Of interest, exercise seems to confer no additional benefit to weight loss when hemostatic factors are considered.[97]

Hypertension

Although it is well accepted that hypertension is a major CVD risk factor, an elevated blood pressure is an underrecognized and therefore an undertreated condition.[1] It is important to keep in mind that obesity and hypertension often coexist; the majority of patients with high blood pressure are overweight, and hypertension is about six times more frequent in obese than in lean subjects.[96] Moreover, weight gain in young people is an important risk factor for subsequent development of hypertension.[84] Weight reduction is one of the rare antihypertensive strategies that decreases blood pressure in normotensive as well as hypertensive persons.[2, 3] As little as a 10% reduction in body weight can decrease blood pressure among obese hypertensive patients.[18, 68] It has also been suggested that an exaggerated blood pressure response to exercise may be a better predictor of target organ damage than resting blood pressure.[47, 50] Again, it has been reported that weight loss could decrease blood pressure and heart rate measured both at rest and at all exercise intensities.[16] Interestingly, it has been shown that weight reduction induced by a modest exercise prescription and by a hypocaloric diet could decrease left ventricular mass, which is a well-recognized CVD risk factor, regardless of blood pressure in obese subjects.[54]

INFLUENCE OF PHYSICAL ACTIVITY ON ADIPOSE TISSUE METABOLISM

There is an inverse relationship between the amount of daily physical activity and body weight. Exercise requires energy, and the two main sources of fuel for muscle contraction are carbohydrates (CHO) and lipid. The major source of lipid energy for muscle is the TG stored in adipose tissue, but available as FFA. Exercise is one of the most potent physiological stimuli for lipolysis; it is higher during exercise in trained subjects than that reported during critical illness[57] or even after 84 hours of starvation.[58] In obesity, the expanded adipose tissue mass provides abundant lipid substrates to meet the needs of increased energy expenditure associated with exercise. For instance, an 80-kg man with 25% body fat (20 kg) has stored in adipose tissue approximately 180,000 calories. Thus, only 1 kg of adipose tissue TG would be sufficient to provide energy for several marathons. Numerous factors associated with obesity, that is, gender, body fat mass, adipose tissue distribution, and number and size of adipose cells, contribute to the eventual response to exercise training. For instance, in response to a 20-week exercise training program, Tremblay et

al[99] reported that men, matched for body fat mass, with a high fat cell size, lost six times more body fat mass (loss of 4.4 kg) than men with small adipose cells (loss of 0.7 kg). Of note, women in this study with either high or low fat cell size did not lose body fat. Therefore, it seems that there might be a certain morphology (size + number of cells + distribution) that may explain, at least to a certain extent, the susceptibility to lose weight in response to an exercise program.[7] During exercise, the lipolytic rate has been shown to be higher in abdominal subcutaneous than in gluteal/femoral subcutaneous adipose tissue, especially in women,[10] whereas there is no gender difference at rest. Women also exhibit less lipolysis during exercise than men,[31, 32] and this phenomenon may help explain why men decrease body fat more efficiently with physical training than do women.[30, 99] Another important clinical issue that should be kept in mind is that when exercise is considered in the management of obesity, the weight loss may be accompanied by an increase in appetite. Careful attention should be given to this adaptation that may compromise weight loss.[37]

EXERCISE PRESCRIPTION

The minimal objectives of a weight loss and management program are: to prevent further weight gain, to reduce body weight, and to permanently maintain a lower body weight. Regular physical activity is a well-recognized tool for long-term weight maintenance because it contributes to increased energy expenditure through a caloric deficit (although generally small) contributing to weight loss.[102] Although epidemiologic studies have suggested that weight cycling could be associated with an elevated risk of death from CVD,[17, 53] there is little evidence to support the view that weight cycling could be related to an increased prevalence/risk of coronary artery disease.[77] Patients should have their BMI and levels of abdominal fat measured with goals of weight reduction established to favorably impact health outcomes, including the risk of a first or recurrent CVD event.[100, 112] Simply stated, weight reduction depends upon energy intake compared to energy expenditure. Approximately 1 pound per week can be lost with no change in physical activity if caloric intake can be reduced by only 2100 kJ (500 kcal)/d. Although aerobic exercise alone produces a modest weight reduction, generally 2% to 3%, increased physical activity is extremely important in sustaining the weight-reduced state.[36, 73, 101] An intervention combining behavior therapy, a low-calorie diet, and increased physical activity is probably the most successful management approach for weight loss and weight maintenance. In overweight/obese patients who have reached the proper "readiness" state, this approach should be emphasized and sustained for a few months before considering other strategies such as pharmacotherapy. Weight loss programs that result in a slow but steady weight reduction, eg, 1 pound to 2 pounds per week, may be more effective long-term than those that result in rapid weight losses.[105] Indeed, behavioral strategies reinforcing changes in diet and physical activity can produce weight loss in the range of 10% over a period of 6 months in obese adults.[3] Unfortunately, long-term follow-up results of patients undergoing behavior therapy show a return to baseline weight for the majority of subjects in the absence of continued behavior intervention.[80, 106, 107] These negative results reinforce the importance of incorporating daily exercise in the lifestyle of overweight/obese patients. Because of the presence of high left ventricular filling pressure and, as in type 2 diabetes, left ventricular diastolic dysfunction, the usually recommended exercise prescription may be inappropriate for the obese individuals.[85] Obesity is associated with persistence of elevated cardiac filling pressures during exercise,[5, 13] and the average left ventricular filling pressure rises with exercise similarly (about 20 mm Hg) after weight loss. Of interest, reduced ventricular compliance characterized by left ventricular diastolic dysfunction during exercise does not always regress with weight loss.[5, 13]

An important issue is whether one needs to focus on exercise intensity in order to achieve metabolic improvements and reduce the risk of coronary heart disease in obese individuals. Concomitant with diet therapy, low-intensity training of 30% to 50% $\dot{V}O_2$max of long duration (90 minutes to 240 minutes) and high frequency has been proposed for losing body fat. This recommendation is based on the premise that the dominant fuel for energy during the first 20 minutes of exercise is glycogen; exercising more than 30 minutes will

increase the usage of fat stores.[51] Of note, as obese people have a lower mechanical efficiency (defined by the relation between oxygen uptake and external work), the most appropriate approach to prescribe exercise is to base work intensity on the oxygen cost relative to $\dot{V}O_2$max.[46] This notion has clinical importance, since subjects with obesity usually get standard recommendations to lose weight by decreasing food intake and increasing physical activities. For instance, normal-weight subjects use about 35% $\dot{V}O_2$max when walking at a self-selected, comfortable pace.[12] This activity is generally considered a convenient and mild form of training. It is accessible to everyone and carries a low risk of injuries, which are increased in obesity because of the burden of extra weight on the joints. Joint considerations should, however, not limit physical activity, since exercise combined with diet leads to improvement in pain, disability, performance and gait in obese older adults with knee osteoarthritis.[75]

However, individuals often find the exercise prescription difficult to follow, since they get extremely tired while walking at the pace recommended by the clinician. $\dot{V}O_2$max and heart rate during brisk walking is higher in obese than in normal-weight individuals.[70] Thus, even walking may represent a difficult exercise modality for obese individuals, since they can use as much as 56% $\dot{V}O_2$max (some using between 64% to 98% $\dot{V}O_2$max) to meet the demand of such an activity compared to only about 35% in normal-weight subjects. Therefore, long and brisk walks should not be regarded as low-intensity forms of training for obese people in general. Since the average work load during a day causes fatigue if it exceeds 30% to 40% $\dot{V}O_2$max,[12] it seems natural that walking for exercise may be too demanding for many obese patients. In addition, walking outdoors can be demanding because of uneven or slippery surfaces, and heavy outdoor clothes during the autumn and winter seasons add to body weight. Moreover, severe obesity may impair the ability to properly walk, especially when the obesity is of the gynoid form. Gluteal fat increases the friction on clothing and skin, making it even more unpleasant to walk. This common problem is often neglected in clinical practice. Increasing obesity and age, abnormal gait pattern, degenerative pain, friction of clothes and skin problems may increase the relative oxygen cost. Clinical assessment may be difficult, because neither BMI, walking speed rate, perceived exertion nor pain (Borg scale) seem to predict the degree of effort of walking.[70] In the clinical setting, it is most of the time impractical to measure $\dot{V}O_2$max before giving advice on physical activity. Nevertheless, measuring heart rate after a 4- to 6-minute walk may be a simpler way to judge the relative cost of walking. Heart rate exceeding 100 beats per minute during walking is generally associated with an exercise intensity of about 50% $\dot{V}O_2$max (Fig. 1).

Obese individuals who enjoy walking and who can tolerate this form of physical activity without too many unpleasant side effects should certainly continue to do so. In general, however, recommendations should focus on training regimens, not generating pain over time, otherwise compliance will obviously be impaired. It is also important to keep in mind that various aerobic training modalities (walking, cycling, swimming) may have a different impact on weight loss. Notably, swimming protocols have generated rather disappointing results regarding weight loss.[52] Health care professionals should also emphasize that heart rate should be properly assessed at the wrist level, because the carotid pulsation may be difficult to find in obese patients. It is also very important to inform the patients about the results to be expected from the recommended exercise regimen in order to avoid unrealistic expectations on weight loss. Body weight normalization should obviously not be the target, but rather some weight loss associated with improvements in the risk factor profile. As an example, a working model of an algorithm for the

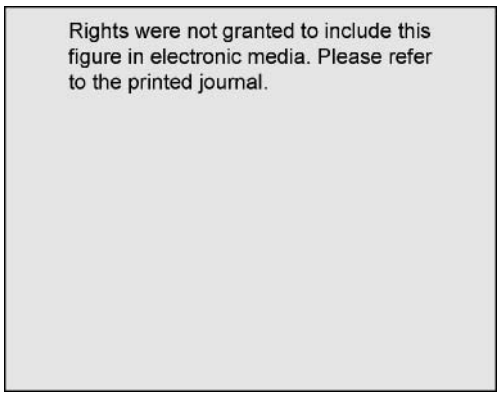

Figure 1. Simple regression between percentage of $\dot{V}O_2$max and heart rate during walking in obese women. $r = 0.63$; $P < .0001$. (*Adapted from* Mattsson E, Larsson UE, Rossner S: Is walking for exercise too exhausting for obese women? Int J Obes Relat Metab Disord 21:380–386, 1997; with permission.)

detection and management of the high-risk obese individuals is suggested in Figure 2.

Reduced-Obese Individuals

A better understanding of adipose tissue metabolism in weight-reduced obese subjects is important because of the high recidivism. Weight loss by hypocaloric diet decreases lipolysis and fat oxidation, adaptations that may predispose individuals to weight regaining. The blunted utilization of fat as fuel during a 60-minute bout of exercise at 50% $\dot{V}O_2max$ contributes to a positive fat balance and possibly weight gain in formerly obese individuals.[88] However, studies of adipose tissue function in vitro have shown that the addition of exercise training to a hypocaloric diet counteracts the decline in lipolytic responsiveness, fat oxidation and resting metabolic rate in weight-reduced postmenopausal women.[76, 88] Moreover, the lipolytic adaptations are of the same magnitude between subcutaneous abdominal and gluteal/femoral adipose tissue regions.[76] Thus, a reduction of body weight to a level that would still be considered as overweight is accompanied by a decreased basal rate of lipolysis but by an improved catecholamine-stimulated lipolysis in vitro[8] Insulin sensitivity is also increased after weight reduction under isocaloric maintenance of the reduced-obese state.[113, 115] Although most of the in vivo action of insulin is accounted for by its effect on the skeletal muscle,[25] the ability of increased insulin sensitivity to predict weight regain in reduced-obese subjects[115] is partly explained by the effects of insulin on adipose tissue. Suggested *risk factors for body weight regain* include:

Increased insulin sensitivity
Low resting metabolic rate for a given body size and composition
Low ratio of fat to carbohydrate oxidation (ie, high respiratory quotient)
Low levels of physical activity
Caloric intake

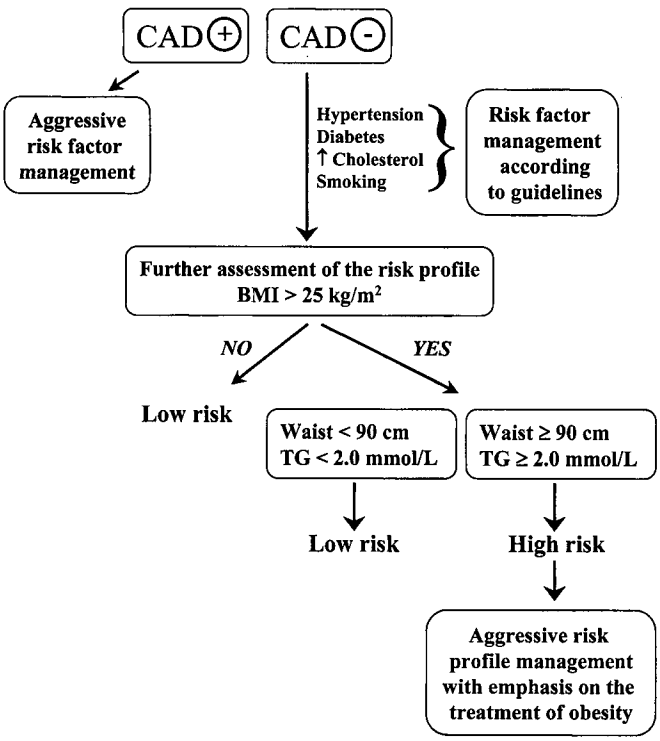

Figure 2. Working model for an algorithm allowing effective and simple identification by health professionals of the high-risk form of overweightness and obesity among individuals asymptomatic for coronary artery disease (CAD). BMI = Body mass index; TG = triglycerides.

SUMMARY

Obesity is a chronic metabolic disorder associated with CVD and increased morbidity and mortality. When the BMI is $\geq$ 30 kg/m^2, mortality rates from all causes, and especially CVD, are increased by 50% to 100%. There is strong evidence that weight loss in overweight and obese individuals improves risk factors for diabetes and CVD. Additional evidence indicates that weight loss and the associated diuresis reduce blood pressure in both overweight hypertensive and nonhypertensive individuals, reduce serum TG levels, increase high-density lipoprotein cholesterol levels, and may produce some reduction in low-density lipoprotein cholesterol concentrations. Of interest, even if weight loss is minimal, obese individuals showing a good level of cardiorespiratory fitness are at reduced risk for cardiovascular mortality than lean but poorly fit subjects.[62] Insulin and catecholamines have pronounced metabolic effects on human adipose tissue metabolism. Insulin stimulates LPL and inhibits HSL; the opposite is true for catecholamines. There is regional variation in adipocyte TG turnover favoring lipid mobilization in the visceral fat depots and lipid storage in the peripheral subcutaneous sites. The hormonal regulation of adipocyte TG turnover is altered in obesity and is most marked in central obesity. There is resistance to insulin stimulation of LPL; however, LPL activity in fasted obese subjects is increased and remains so following weight reduction. Catecholamine-induced lipolysis is enhanced in visceral fat but decreased in subcutaneous fat. Numerous adaptive responses take place with physical training. These adaptations result in a more efficient system for oxygen transfer to muscle, which is now able to better utilize the unlimited lipid stores instead of the limited carbohydrate reserves available. In addition, the reduced adipose tissue mass represents an important mechanical advantage, allowing better long-term work. Gender differences have been reported in the adaptation of adipose tissue metabolism to aerobic exercise training. Physical training helps counteract the permissive and affluent environment that predisposes reduced-obese subjects to regain weight. An exercise program using weight resistance modalities may also be included safely, and it improved program retention in a multidisciplinary weight management program that was designed for obese children.[44,95]

Thirty to 45 minutes of physical activity of moderate intensity, performed 3 to 5 days a week, should be encouraged. All adults should set a long-term goal to accumulate at least 30 minutes or more of moderate-intensity physical activity on most, and preferably all days.[79] Public health interventions promoting walking are likely to be the most successful. Indeed, walking is unique because of its safety, accessibility, and popularity. It is noteworthy that there is a clear dissociation between the adaptation of cardiorespiratory fitness and the improvements in the metabolic risk profile that can be induced by endurance training programs. It appears that as long as the increase in energy expenditure is sufficient, low-intensity endurance exercise is likely to generate beneficial metabolic effects that would be essentially similar to those produced by high-intensity exercise. The clinician should therefore focus on the improvement of the metabolic profile rather than on weight loss alone. Realistic goals should be set between the clinician and the patient, with a weight loss of approximately of 0.5 to 1 pound per week. It should be kept in mind that since it generally takes years to become overweight or obese, a weight loss pattern of 0.5 or 1 pound per week will require time and perseverance to reach the proposed target. However, the use of physical activity as a method to lose weight seems inversely related to patients' age and BMI and directly related to the level of education.[4] Thus, public health interventions helping these groups to become physically active remain a challenge[61] and further emphasize the importance of the one-on-one interaction between the clinician/health care professional with the obese individual "at risk" of CVD. This notion is critical, as it has been shown that less than half of obese adults have reported being advised to lose weight under the guidance of health care professionals.[49,110]

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