Comprehensive Clinical Assessment of Modifiable Cardiometabolic Risk Factors

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Cardiovascular disease (CVD) is associated with significant morbidity and mortality. Some of the risk factors for CVD are unmodifiable such as male gender, family history, race, and increasing age. There are, however, several modifiable risk factors for CVD, including tobacco use, hypertension, obesity, diabetes, physical inactivity, and dyslipidemia. Patients' overall or global risk of CVD, which takes into account the presence or absence of both modifiable and unmodifiable risk factors, should be determined periodically. Patients found to be at increased risk due to the presence of modifiable risk factors should be counseled on how to make lifestyle changes and thereby decrease their risk of developing CVD. Weight loss and increased physical activity have a positive effect on most of the modifiable risk factors. Some patients, however, will also require pharmacotherapy to address such risk factors as hypertension, obesity, diabetes, and dyslipidemia. (*Clinical Cornerstone*. 2008;9[Suppl 1]:S9–S19) © 2008 Excerpta Medica Inc.

Cardiovascular disease (CVD) is a major cause of morbidity and mortality in the United States. A 2007 report from the American Heart Association (AHA) estimated that ~79 million Americans, or 1 out of every 3 individuals, have ≥1 type of CVD, including coronary heart disease (CHD), congestive heart failure, myocardial infarction (MI), and stroke.[1] Approximately one third of the deaths each year in the United States are attributed to CVD, meaning that CVD accounts for more deaths than does any other single or group cause.[1] In fact, CVD is responsible for the loss of more lives than are cancer, chronic lower respiratory diseases, accidents, and diabetes combined.[1] Although there has been some decline in deaths due to CVD over the past 2 decades, clearly, there is still opportunity for further reduction of morbidity and mortality.[2]

There are substantial economic costs associated with CVD. In 2007, the estimated total (direct and indirect) cost of CVD in the United States was $431.8 billion[1]; therefore, if the incidence of CVD can be reduced, a substantial savings in health care costs can be realized.

Numerous risk factors have been associated with the development of CVD.[3] These risk factors include *modifiable factors*, such as tobacco use, hypertension, obesity, diabetes, physical inactivity, and dyslipidemia, as well as *unmodifiable factors*, such as male gender, family history, race, and increasing age. Studies have demonstrated that by affecting positive changes in modifiable risk factors, the morbidity and mortality associated with CVD can be reduced. For example, there were 341,745 fewer deaths from CHD in 2000 than in 1980, with ~44% of this decrease attributable to reductions in levels of total cholesterol and systolic blood pressure and increases in physical activity.[2]

This paper addresses modifiable risk factors. Ideally, all patients should be assessed regularly for the presence of CVD risk factors, and when modifiable factors are identified, efforts should be made to encourage lifestyle changes to reduce CVD risk. Pharmacotherapy also may be required to further reduce risk.

MODIFIABLE RISK FACTORS

Tobacco Use

Although tobacco use has decreased over the last 50 years, as many as 44.5 million Americans, or 21% of the adult population, still use tobacco.[4] Tobacco use of all kinds (ie, cigarettes, cigars, and chewing tobacco) increases the risk of CVD.[5] Tobacco use also increases the

KEY POINT

Ideally, all patients should be assessed regularly for the presence of CVD risk factors, and when modifiable factors are identified, efforts should be made to encourage lifestyle changes to reduce CVD risk.

risk of death from cancer and chronic obstructive pulmonary disease and may contribute to diabetes.[4,6] The yearly economic cost of lost productivity due to CVD attributable to smoking has been estimated at $35.6 billion.[6]

Interventions that result in a decrease in tobacco use can reduce not only the risk of CVD but also the risk of cancer, diabetes, and pulmonary disease.[6,7] Ford et al[2] determined that a decline of ~12% in the prevalence of smoking from 1980 to 2000 resulted in 39,925 fewer deaths due to CHD in 2000 than in 1980. Although there is a residual risk of CVD after individuals quit smoking, the risk is substantially lower than if they continued to smoke.[5] For example, 1 year after quitting, their risk of CHD is 50% lower than if they continued to smoke.[6]

KEY POINT

Interventions that result in a decrease in tobacco use can reduce not only the risk of CVD but also the risk of cancer, diabetes, and pulmonary disease.

Although the best strategy is to educate people so that they never start using tobacco,[4] health care professionals must encourage those who do smoke to quit. Health care professionals should ask patients regularly about their use of tobacco. Whenever a medical history is obtained from a patient, it should include a question regarding the use of tobacco,[7] and because even a brief cessation of smoking may reduce associated health risks, all those who use tobacco should be encouraged to stop.[6] Furthermore, many patients who smoke want to quit, and advice from a physician on the importance of quitting has been shown to improve rates of cessation.[4,8] To facilitate quitting, patients should be provided with suggestions for social support, skills training, and pharmacotherapy, including nicotine replacement, bupropion, and varenicline.[4,7,8] The US Department of Health and Human Services has a clinical practice guideline ("Treating Tobacco Use and Dependence")[7] that provides suggestions on how to inquire about tobacco use and how to assist those who use tobacco in quitting. These guidelines suggest that clinicians address the "5 A's" when inquiring about tobacco use:

- **Ask** about tobacco use
- **Advise** to quit
- **Assess** willingness to quit
- **Assist** in the quit attempt (counseling and pharmacotherapy)
- **Arrange** a follow-up

Although inquiring about tobacco use and encouraging those who smoke to quit may seem obvious, studies have shown that smoking status is identified in only about two thirds of clinic visits, and counseling regarding the benefits of smoking cessation is provided in only about one fifth of smokers' visits.[7] Furthermore, studies show that tobacco cessation treatments are generally offered only to those patients who are suffering from tobacco-related diseases.[7]

Hypertension

Hypertension is another common modifiable risk factor for CVD. More than 50 million Americans and 1 billion people worldwide are thought to have high blood pressure.[9] In the 2004 National Ambulatory Medical Care Survey,[10] hypertension was the most common primary care diagnosis. Hypertension is particularly common among individuals with diabetes.[9]

With every 20/10-mm Hg increase in blood pressure, there is a doubling of CVD risk.[9] In the Framingham Heart Study,[11] individuals with high-normal blood pressure levels (130/85–139/89 mm Hg) had at least a 2-fold greater increase in the risk of CVD than did those with optimal blood pressure levels (<120/80 mm Hg). Furthermore, studies have shown that treatments that reduce blood pressure to <140/90 mm Hg (<130/80 mm Hg in those with diabetes or renal disease) are associated with

a decrease in CVD complications and possibly also a decrease in diabetes-related mortality.[9] Unfortunately, many patients with hypertension are undiagnosed, and many of those who are diagnosed do not receive appropriate treatment.[9]

KEY POINT

Every 20/10-mm Hg increase in blood pressure is associated with a doubling of CVD risk.

It is important to accurately measure blood pressure at each patient visit.[9] Ideally, this should be accomplished using the auscultatory method of blood pressure measurement. Blood pressure assessment should be conducted only by individuals who have appropriate training and are using regularly inspected and validated equipment. Patients should be seated quietly in a chair for ≥5 minutes with their feet on the floor and their arms supported at the level of their heart. They should be encouraged to avoid caffeine, smoking, and exercise for 30 minutes prior to the examination. The bladder of the cuff used should encircle ≥80% of the arm. Whenever blood pressure levels are assessed, ≥2 measurements should be made and the average of the measurements should be recorded. For some individuals, self-measurement or ambulatory blood pressure monitoring may also be indicated.[9] The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure considers optimal blood pressure to be <120/80 mm Hg, blood pressure levels of 120/80 to 139/89 mm Hg to be prehypertension, and blood pressure levels ≥140/90 mm Hg to be hypertension.[9] Because a healthy lifestyle is imperative to the prevention and management of hypertension, it should be promoted whenever blood pressure is measured. Discussions regarding a healthy lifestyle should include information on diet, exercise, the importance of maintaining a healthy body weight, moderate alcohol consumption, and avoidance of tobacco use (**Table I**).[9]

In addition to lifestyle modifications, pharmacotherapy is recommended for all patients with hypertension.[9] In most patients, ≥2 antihypertensive agents are required to adequately control blood pressure levels. Many agents, including diuretics, calcium channel blockers, angiotensin-converting enzyme inhibitors, and angiotensin receptor blockers, have been shown to reduce blood pressure and decrease CVD risk.[9,12] Although β-blockers reduce blood pressure levels, these agents may not be associated with the same reduction in CVD risk that may be achieved with other antihypertensive agents; therefore, β-blockers are not recommended as first-line therapy for the treatment of hypertension.[13–15] Regardless of which agents are used, it is important that patients be followed carefully to ensure that blood pressure goals are achieved. Generally, the goal of treatment is a blood pressure level of

TABLE I. LIFESTYLE MODIFICATIONS TO PREVENT AND MANAGE HYPERTENSION.*

Modification	Recommendation	Reduction in SBP, Range†
Weight reduction	Maintain normal body weight (BMI, 18.5–24.9 kg/m^2)	5–20 mm Hg/10 kg
DASH eating plan	↑ Consumption of fruits, vegetables, and low-fat dairy products ↓ Consumption of saturated and total fat	8–14 mm Hg
Dietary sodium reduction	↓ Intake to ≤100 mmol/d (2.4 g sodium or 6 g sodium chloride)	2–8 mm Hg
Physical activity	Engage in regular aerobic activity (eg, brisk walking) ≥30 min/d most days of the week	4–9 mm Hg
Moderate alcohol consumption	Limit consumption to ≤2 drinks/d for men and ≤1 drink/d for women and lighter-weight persons‡	2–4 mm Hg

SBP = systolic blood pressure; BMI = body mass index; DASH = Dietary Approaches to Stop Hypertension; ↑ = increase; ↓ = reduce.
*For overall cardiovascular risk reduction, tobacco use should be stopped.
†The effects of implementing these modifications are dose- and time-dependent and could be greater for some individuals.
‡One drink equals 12 oz beer, 5 oz wine, or 1.5 oz 80-proof whiskey.

<140/90 mm Hg; however, in patients with chronic kidney disease or diabetes, the goal is <130/80 mm Hg.[9]

Obesity

Although the prevalence of some of the modifiable risk factors for CVD may have decreased over the past several decades, the prevalence of obesity has increased at an alarming rate.[2] Throughout the 1980s and 1990s, the prevalence of obesity increased by ~50% per decade.[16] It is estimated that 97 million American adults are overweight or obese.[17] Worldwide an estimated 1.1 billion adults are overweight and 312 million are obese.[18]

There is a correlation between CVD risk and excess body fat as measured by body mass index (BMI) and waist circumference.[6,17] In an analysis of the age-adjusted death rate for CHD, an increase in BMI from 25.6 kg/m^2 in 1980 to 28.8 kg/m^2 in 2000 was found to account for an 8% increase in deaths over the same period.[2] Increased weight is also associated with an increased risk of hypertension, dyslipidemia, type 2 diabetes mellitus (DM), gallbladder disease, osteoarthritis, sleep apnea, respiratory problems, and cancer.[17] Studies have demonstrated, however, that when weight is reduced, there is a reduction in the risk of CVD.[17] Furthermore, weight loss may also have a positive effect on other CVD risk factors, including hypertension, lipid profile, and blood glucose levels.[19]

KEY POINT

There is a correlation between CVD risk and excess body fat as measured by BMI and waist circumference.

Every patient visit is an opportunity to assess BMI and waist circumference. Patients with a BMI of 25 to 29.9 kg/m^2 are considered overweight, and those with a BMI of ≥30 kg/m^2 are considered obese. BMI is calculated by dividing weight in kilograms by height in meters squared. A waist circumference ≥40 inches (102 cm) in men and ≥35 inches (88 cm) in women is associated with an increased risk of CVD. **Figure 1** demonstrates how to measure waist circumference.[17]

Patients who have an increased BMI or waist circumference should be counseled on diet modification, increasing their physical activity levels, and behavioral therapy. Some patients may benefit from pharmacotherapy or weight loss surgery as well.[17] It is also important to implement a strategy to assist patients in maintaining their weight loss.[17] The AHA Web site (www.americanheart.org) has several suggestions for managing weight, including diet and exercise programs.

Diabetes

Another CVD risk factor that is becoming more prevalent is diabetes.[20] Worldwide, the number of people with diabetes has been projected to increase from 171 million in 2000 to 366 million by 2030.[18] The increase in the prevalence of diabetes is closely related to the increased prevalence of obesity; indeed, ~90% of type 2 DM is attributable to excess body weight.[18]

The increased prevalence of diabetes has had a significant impact on the prevalence of CVD. Individuals with diabetes, impaired fasting glucose (IFG), or impaired glucose tolerance (IGT) are at an increased risk of developing CVD,[21] as well as an increased risk of dying from it.[22] An analysis of the age-adjusted death rate for CHD from 1980 to 2000 demonstrated that an increase in the prevalence of diabetes from 6.5% in 1980 to 9.4% in 2000 accounted for a 9.8% increase in deaths due to CHD over the same period.[2]

KEY POINT

Individuals with diabetes, IFG, or IGT are at an increased risk of developing CVD, as well as an increased risk of dying from it.

The American Diabetes Association[21] recommends that regular blood glucose evaluations be performed on all people, with and without diabetes. Every 3 years, asymptomatic individuals ≥45 years of age should be screened for diabetes with a fasting plasma glucose (FPG) test, a 2-hour oral glucose tolerance test (OGTT), or both. More frequent screenings may be appropriate in asymptomatic individuals who are overweight and have ≥1 other risk factor for type 2 DM. Other risk factors include habitual physical inactivity, having a first-degree relative with type 2 DM, being a member of an

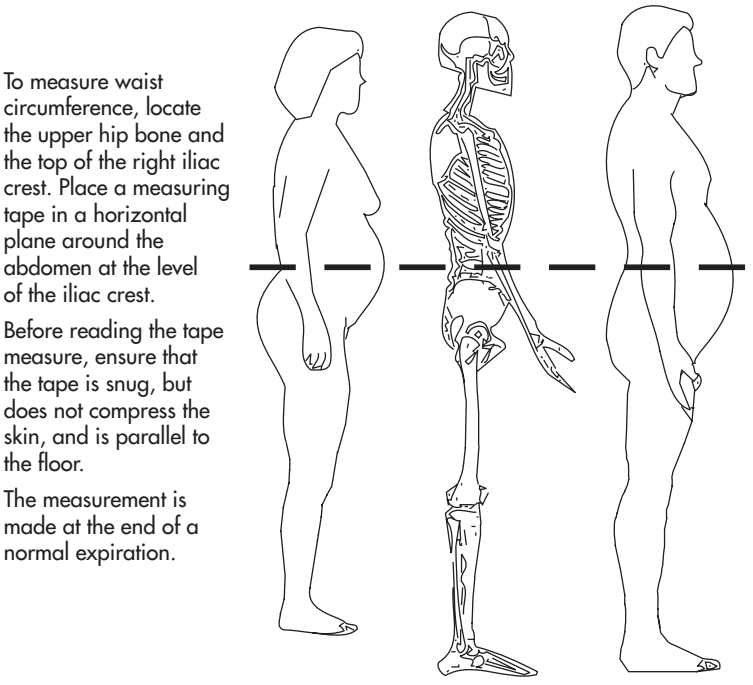

Figure 1. Measuring waist circumference.[17]

ethnic population (eg, African American, Latino, Native American, Asian American, or Pacific Islander), being diagnosed with gestational diabetes or delivering a baby weighing >9 pounds, being diagnosed with hypertension, and having low levels of high-density lipoprotein cholesterol (HDL-C) or high levels of triglycerides. Risk factors also include having polycystic ovary syndrome, a condition associated with insulin resistance, or a history of vascular disease. Consideration should be given to screening individuals <45 years of age if they are overweight and have other risk factors for diabetes. **Table II** explains how to interpret FPG levels and plasma glucose levels obtained 2 hours after an OGTT.[21]

For individuals with IFG or IGT, a diet and exercise program is recommended to prevent conversion from IGT to type 2 DM.[21,23] Individuals diagnosed with diabetes should be counseled on diet and exercise, and appropriate pharmacotherapy should be prescribed (**Figure 2**).[21] To assess treatment response, glycosylated hemoglobin (A1C) levels should be monitored regularly—at least twice a year.[21] Although the goals of diabetes treatment should be based on the individual patient, in general, an A1C of <7% is desired.[21]

TABLE II. DIAGNOSTIC VALUES FOR DIABETES AND PREDIABETES.

	Diabetes*	Prediabetes (IFG/IGT)
FPG, mg/dL†	≥126	100–125
OGTT (2-h postload), mg/dL‡	≥200	140–199

IFG = impaired fasting glucose; IGT = impaired glucose tolerance; FPG = fasting plasma glucose; OGTT = oral glucose tolerance test.

*Diabetes is diagnosed if a patient has an elevated FPG level, an elevated glucose level as measured by 2-hour postload OGTT, or symptoms of diabetes (ie, polyuria, polydipsia, and unexplained weight loss) *and* a casual plasma glucose level ≥200 mg/dL.

† No caloric intake for ≥8 hours.

‡ Performed using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.

Physical Inactivity

Physical inactivity not only contributes to the development of hypertension, obesity, and diabetes, but it is also an independent risk factor for CVD.[21,24,25] Recent data suggest that only about half of Americans meet the cur-

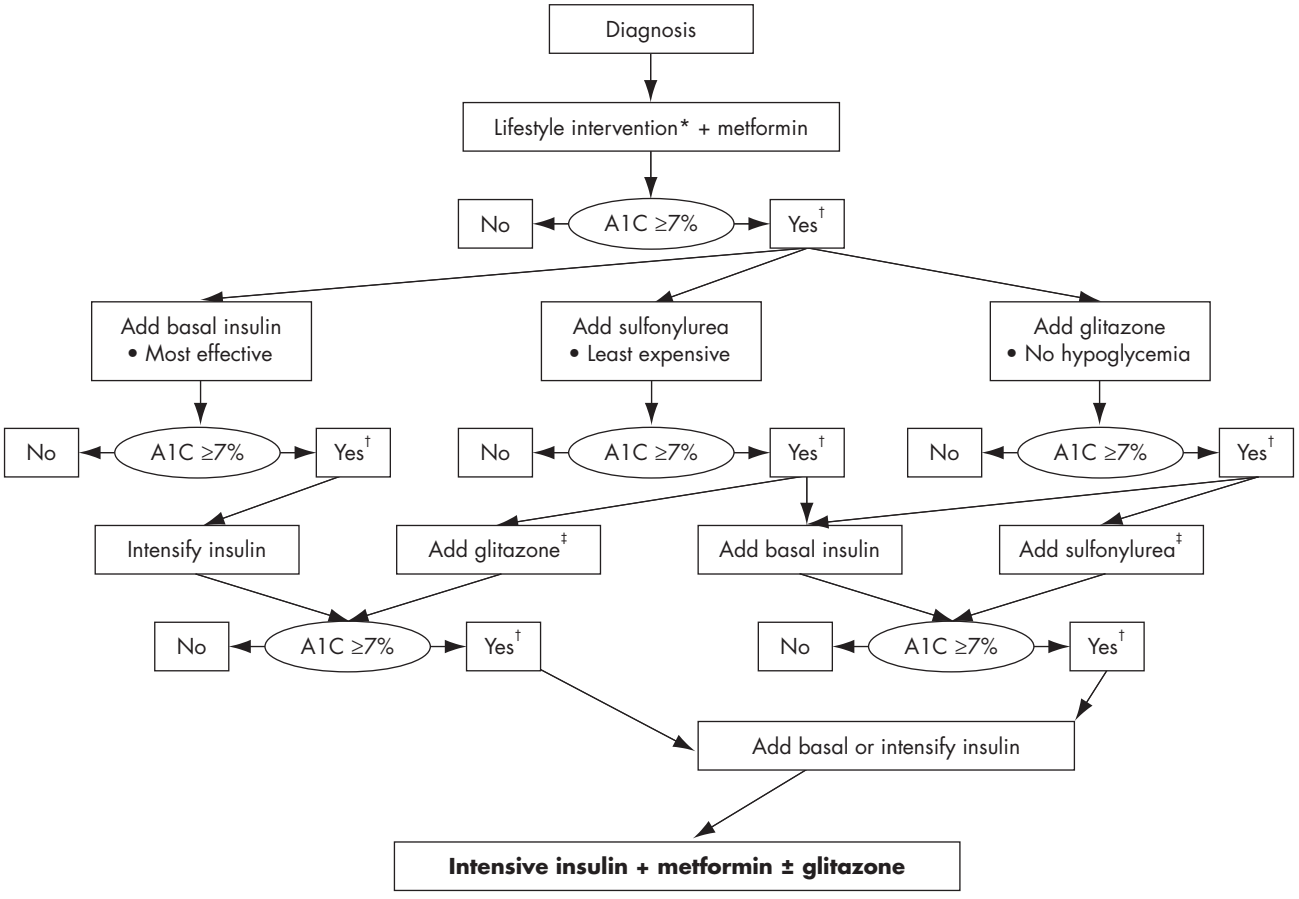

Figure 2. Algorithm for the management of type 2 diabetes mellitus. *Reinforce lifestyle intervention at every visit. †Check glycosylated hemoglobin (A1C) every 3 months until it is <7%, and at least every 6 months thereafter. ‡Although 3 oral agents can be used, initiation and intensification of insulin therapy is preferred based on effectiveness and expense. Adapted with permission.[21]

rent recommendations for physical activity.[26] Therefore, questions regarding physical activity should be included routinely when obtaining a patient history.

Based on the most recent recommendations of the American College of Sports Medicine and the AHA,[27] adults should perform moderate-intensity aerobic activity (exercise that accelerates the heart rate) for a minimum of 30 minutes 5 days a week or vigorous-intensity aerobic activity (exercise that causes rapid breathing and a substantial increase in heart rate) for a minimum of 20 minutes 3 days a week. Whereas the recommendations for exercise are similar for younger and older adults, the type of activity that constitutes moderate-intensity or vigorous-intensity exercise sometimes differs.[28] For example, in young adults, a brisk walk is considered moderate-intensity exercise and jogging is considered vigorous-intensity exercise. In some older adults, however, a slow walk may be considered moderate-intensity exercise and a brisk walk may be considered vigorous-intensity exercise. The recommended duration of aerobic activity may be accumulated in a continuous session (eg, one 30-minute brisk walk) or in a series of short sessions (eg, three 10-minute brisk walks).[29] In addition to aerobic

KEY POINT

Physical inactivity not only contributes to the development of hypertension, obesity, and diabetes, but it is also an independent risk factor for CVD.

exercise, all adults should perform activities that maintain or increase muscular strength and endurance for a minimum of 2 days each week. Activities that maintain or increase flexibility and balance are also recommended for older adults.[27,28]

Inactive patients should be encouraged to gradually increase their activity level in a stepwise manner. They should be informed that physical activity may help reduce levels of triglycerides, blood glucose, blood pressure, and weight, and it may also help reduce the risk of developing CVD and diabetes.[6,25,30] In addition, regular physical activity may reduce the risk of developing osteoporosis, cancer, anxiety, and depression.[27] Patients should also be commended for any increase in their activity levels. The AHA (www.americanheart.org) and the American College of Sports Medicine (www.acsm.org) Web sites offer many suggestions for exercise programs.

Dyslipidemia

The risk of CVD is also increased in individuals with dyslipidemia. Although increased levels of low-density lipoprotein cholesterol (LDL-C) and triglycerides and decreased levels of HDL-C have all been associated with an increased risk of CVD, the greatest risk is associated with increased levels of LDL-C. Consequently, lipid goals are generally stated in terms of LDL-C.[31]

KEY POINT

Increased levels of LDL-C and triglycerides and decreased levels of HDL-C have all been associated with an increased risk of CVD; however, the greatest risk is associated with increased levels of LDL-C.

It is recommended that all adults ≥20 years of age have a fasting lipoprotein profile, which includes measures of total cholesterol, LDL-C, HDL-C, and triglycerides, once every 5 years. These profiles can be interpreted using the National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) classification of cholesterol levels (**Table III**).[31]

TABLE III. CLASSIFICATIONS OF TOTAL CHOLESTEROL, LDL-C, HDL-C, AND TRIGLYCERIDES.[31]

Total cholesterol, mg/dL	
Desirable	<200
Borderline high	200–239
High	≥240
LDL-C, mg/dL	
Optimal	<100
Near/above optimal	100–129
Borderline high	130–159
High	160–189
Very high	≥190
HDL-C, mg/dL	
Low	<40
High (desirable)	≥60
Triglycerides, mg/dL	
Normal	<150
Borderline high	150–199
High	200–499
Very high	≥500

LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol.

The goals of treatment for dyslipidemia depend on whether or not a patient has other risk factors for CVD (eg, tobacco use, hypertension, low HDL-C levels, family history of premature CHD, or age ≥45 years for a man and ≥55 years for a woman).[31] If a patient has CHD or a CHD risk equivalent (eg, diabetes or atherosclerotic disease), the LDL-C goal is <100 mg/dL. The 10-year risk of a major coronary event for such a patient is >20% (ie, >20 out of 100 of these individuals will develop CHD or have a recurrent CHD event within 10 years). If a patient does not have CHD or a CHD risk equivalent but has ≥2 other risk factors, the LDL-C goal is <130 mg/dL; these patients have a 10-year CHD risk of ≤20%. Finally, if a patient has ≤1 risk factor, the LDL-C goal is <160 mg/dL, as these individuals have a 10-year risk for CHD of <10%. Whereas a low HDL-C level is considered a positive risk factor, an HDL-C level of ≥60 mg/dL is considered a negative risk factor, and its presence removes 1 risk factor from a patient's total count.

For all patients with elevated LDL-C levels,[31] lifestyle changes such as reduced intake of saturated fats and cholesterol, weight reduction, and increased physical activity are recommended. Pharmacotherapy is recommended for those with CHD or CHD risk equivalents if their LDL-C level is ≥130 mg/dL.[31] Pharmacotherapy is rec-

ommended for individuals with ≥2 CHD risk factors if their LDL-C level is ≥130 mg/dL and their 10-year risk for CHD (based on Framingham risk scoring) is 10% to 20% or if their LDL-C level is ≥160 mg/dL and their 10-year risk for CHD is <10%.[31] For individuals with ≤1 risk factor, pharmacotherapy is recommended if their LDL-C level is ≥190 mg/dL.[31] Treatment with lipid-lowering agents to reduce LDL-C levels has been associated with a reduction in the risk of CVD.[32,33]

GLOBAL ASSESSMENT OF RISK FACTORS

Many patients have >1 of the individual CVD risk factors described above. For example, ~86% of patients with type 2 DM also have hypertension,[34] and ~90% of patients with type 2 DM are overweight.[18]

In some patients, multiple risk factors of metabolic origin such as low HDL-C or high triglyceride levels, hypertension, elevated blood glucose levels, and increased waist circumference, can be identified. *Metabolic syndrome* is the term used to characterize the presence of ≥3 of these factors (**Table IV**).[35] It is estimated that ~24% of the US population, or 47 million people, have metabolic syndrome.[36] Patients with metabolic syndrome are at high risk for developing CVD and diabetes.[35]

For individuals with >1 CVD risk factor, clinicians should use tools that consider the impact of multiple factors on a patient's overall risk of CVD. This is an important point since the *overall* risk of CVD may affect the desired goals for each *individual* risk factor. Furthermore, the cumulative effect of several mildly elevated risk factors may produce a high overall CVD risk. This concept was considered by the NCEP ATP III in the development of LDL-C goals.[31]

A commonly used tool to assess a patient's overall risk of CVD is the Framingham risk score.[37] The Framingham risk score comprises 6 factors: blood pressure level, smoking status, age, LDL-C or total cholesterol level, HDL-C level, and presence/absence of diabetes. A score is given for each of these factors, and based on the total score of the 6 factors, a 10-year risk of CVD is assigned. This is the tool that NCEP ATP III guidelines use to determine appropriate treatment strategies.[31] However, several assessment tools are available to calculate a patient's CVD risk, some of which are available online.[38] One of these tools, the NCEP ATP III Risk Assessment Tool for Estimating 10-Year Risk of Having a Heart Attack (http://hp2010.nhlbihin.net/atpiii/calculator.asp), quickly calculates a patient's risk based on his or her age, gender, total cholesterol, HDL-C, smoking status, systolic blood pressure, and use of blood pressure medications. Another tool is the Estimating CHD Risk Using Framingham Heart Study Prediction Score Sheets (http://www.nhlbi.nih.gov/about/Framingham/riskabs.htm). This tool assesses only CHD risk (angina pectoris, MI, or CHD death); it does not assess risk for other heart or valvular diseases.

The Framingham risk score best predicts CVD risk in white and African American men and women who live in the United States and are between the ages of 30 and 65 years; however, it tends to underestimate risk in high-risk populations and overestimate risk in low-risk populations.[39] Specifically, the Framingham risk score may not accurately calculate CVD risk in non-Americans; and among Americans, the accuracy is diminished in Hispanic men, Japanese men, Native American women, men and women <30 years of age or >65 years of age, those with diabetes, and those with severe hypertension.[38] Importantly, the Framingham risk score does not include factors for weight, physical activity, or family history in the assessment of risk.

TABLE IV. DIAGNOSTIC VALUES FOR METABOLIC SYNDROME.[35]

Parameter*	Cut Point
Waist circumference	≥102 cm (40 in) in men ≥ 88 cm (35 in) in women
Triglycerides	≥150 mg/dL *or* Taking medication for ↑ triglyceride levels
HDL-C	<40 mg/dL in men <50 mg/dL in women *or* Taking medication for ↓ HDL-C levels
Blood pressure	≥130 mm Hg systolic blood pressure *or* ≥85 mm Hg diastolic blood pressure *or* Taking medication for hypertension
Fasting glucose	≥100 mg/dL *or* Taking medication for ↑ glucose levels

HDL-C = high-density lipoprotein cholesterol; ↑ = elevated; ↓ = reduced.
*Presence of 3 of the 5 parameters defines metabolic syndrome.

Because of the limitations of the Framingham risk score, other global CVD assessment tools have been developed. Many of these tools target populations that were not represented in the data used to develop the Framingham risk score. For example, guidelines have been developed recently to determine global CVD risk based on a tool developed using European data—Systematic COronary Risk Evaluation (SCORE). SCORE is calculated based on gender, smoking status, age, and total cholesterol, and different charts are available for European countries with high or low risk for CVD.[40] Since many women who develop CVD do not have any of the risk factors assessed in the Framingham risk score, Ridker et al[41] recently described a tool specifically designed for the assessment of global cardiovascular risk in women. This tool uses systolic blood pressure, high-sensitivity C-reactive protein, total cholesterol, HDL-C, A1C, smoking status, and family history of premature MI.

If patients have multiple factors that put them at increased risk for CVD, all of these factors need to be addressed. Ideally, interventions that have a positive impact on multiple risk factors should be employed. For example, weight loss and increased physical activity can have positive effects on blood pressure, lipid, and blood glucose levels.[42] If pharmacologic therapy is needed in patients with multiple risk factors, clinicians must ensure that the agent chosen to treat one risk factor does not negatively affect another risk factor. Instead, pharmacologic treatments that have positive effects on numerous risk factors should be sought.

KEY POINT

If patients have multiple factors that put them at increased risk for CVD, all of these factors need to be addressed.

CONCLUSIONS

CVD continues to be a major cause of morbidity and mortality; therefore, it is important to screen patients regularly for the presence of modifiable CVD risk factors and to determine their overall CVD risk. For patients who are found to be at increased risk for CVD, interventions should be undertaken to modify all risk factors and thereby reduce their CVD risk. Whereas lifestyle changes are essential for modifying a multitude of CVD risk factors, many patients also require pharmacotherapy. If pharmacotherapy is necessary, we must ensure that the agent chosen to treat one risk factor does not negatively affect another. As much as possible, the agents chosen should have a positive effect on multiple risk factors.

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