**INTRODUCTION**

Although medical evidence suggests that the mortality rates for cardiovascular disease can be significantly reduced by lowering blood cholesterol levels, cardiovascular disease ranks as the leading cause of mortality in the United States. Approximately 100 million American adults have total blood cholesterol levels of 200 milligrams per deciliter (mg/dL) and higher, and about 40 million of these have levels of 240 mg/dL or higher. Individuals with total blood cholesterol levels of 240 mg/dL or higher are at increased risk for coronary heart disease (CHD). Since 1988, the National Cholesterol Education Program (NCEP) has issued guidelines identifying low-density lipoprotein cholesterol (LDL-C) as the primary target of cholesterol therapy. The initial guidelines, Adult Treatment Panel (ATP) I, outlined a strategy for primary prevention of CHD in persons with high levels of LDL-C, defined at that time as 160 mg/dL or higher, or those with borderline-high LDL-C and multiple risk factors. ATP II restated the importance of this approach as well as recommending the intensive management of LDL-C in persons with established CHD. For such patients, ATP II set a new, lower LDL-C goal of 100 mg/dL or lower. Like its predecessors, ATP III, issued in May 2001, emphasizes the role of diet and exercise in decreasing the risk for developing CHD. Unlike the earlier versions, the revised guidelines advocate a more aggressive approach to testing and management of LDL-C, with a new focus on CHD prevention in persons with multiple risk factors. Key features of ATP III include:

- a change in the minimum accepted level of high-density lipoprotein cholesterol (HDL-C) as a major heart disease risk factor
- a new set of therapeutic lifestyle changes (TLC), with more power to improve cholesterol levels
- a sharper focus on a cluster of heart disease risk factors known as the metabolic syndrome
- introduction of the concept of CHD risk equivalent—conditions that confer the same short-term (10-year) risk for CHD for a major coronary event (such as myocardial infarction [MI] or coronary death) as those with CHD
- identifying diabetes as a CHD risk equivalent
- increased attention to the treatment of high triglyceride levels.

The new guidelines are expected to substantially expand the number of Americans being treated for high cholesterol, including an increase in the number of individuals undergoing dietary treatment from about 52 million to about 65 million and an increase in the number of patients prescribed a cholesterol-lowering drug from about 13 million to about 36 million. This article will present an overview of specific ATP III recommendations.

**ASSESSING RISK**

ATP III advises that risk assessment be based pri-
mainly on measurement of LDL-C as part of lipoprotein analysis and identification of accompanying risk determinants. A 9-12 hour fasting lipid profile should be obtained in all adults 21 years of age and older every 5 years. ATP III recommends a full panel profile, including total cholesterol, triglycerides, HDL and LDL-C. Of particular concern is an elevated LDL-C level, a major cause of CHD. The higher the LDL-C level, the greater the CHD risk. This relationship is reflected in the LDL-C classification system, which has been revised in ATP III; the optimal LDL-C level is 30mg/dL or <100 mg/dL for patients who have CHD or CHD risk equivalents. (Table 1).

A high HDL-C level is now defined as ≥60 mg/dL; while 35 mg/dL was defined as the minimum acceptable level in previous guidelines, new recommendations advise levels of ≥40 mg/dL or higher. HDL-C levels of ≥60 mg/dL are considered a negative risk factor; in patients with other risk factors for CHD, having a high HDL-C level negates the equivalent of one risk factor from the total count.

A significant point is that ATP III recommends in-hospital assessment of LDL-C for patients with CHD-related events, such as MI, unstable angina, and revascularization. When patients present with an acute CHD-related event, the guidelines suggest obtaining a lipid profile within the first 24 hours. If the LDL-C level is high, starting therapy in the hospital is strongly recommended, particularly because inpatients are usually very motivated to undertake risk-lowering therapy. In addition, a large treatment gap has been traditionally seen in patients who were hospitalized for an acute CHD-related event and remained untreated for months following discharge. However, ATP III cautions that in interpreting LDL-C levels in such patients, clinical decisions should take into account the fact that LDL-C levels begin to decline in the first few hours after a CHD event, are significantly decreased after 24 hours, and may remain low for weeks.

ATP III also creates a new emphasis on triglyceride levels as well as a new classification system for these blood lipids. This is described in greater detail as part of the discussion on metabolic syndrome.

In addition to the patient’s lipid profile, accompanying CHD risk factors are considered in overall risk assessment. A basic principle of prevention in the new guidelines is that the intensity of risk-reduction therapy should be adjusted to the individual’s absolute risk for a major event (such as MI or cardiovascular death). Hence, the first step in evaluating the patient for treatment is to assess the patient’s risk status.

The patients at highest risk are those with known CHD or a history of ischemic stroke. Also considered at risk are patients who may not have a CHD event,

| Table 1. ATP III Classification of LDL Cholesterol (mg/dL) |
|-----------------------------|------------------------------------------------------|
| <100                        | Optimal                                              |
| 100-129                     | Near optimal/above optimal                           |
| 130-159                     | Borderline high                                      |
| 160-189                     | High                                                 |
| ≥190                        | Very high                                            |

Table 2. Major Risk Factors (Exclusive of Cholesterol) That Modify LDL Goals*

- Cigarette smoking
- Hypertension (BP ≥140/90 mm Hg or on an antihypertensive medication)
- Family history of premature CHD (CHD in male first-degree relative <55 years; CHD in female first degree relative <65 years)
- Age (men ≥45 years; women ≥55 years*)

*Risks have been broadened from previous guidelines to include diabetes as a CHD risk equivalent.
but who are still at risk for MI or cardiac death in the next 10 years according to the Framingham projections of 10-year absolute CHD risk. Major risk factors according to these projections include cigarette smoking, age, and presence of hypertension (Table 2).

A significant change noted in ATP III is recognition of the seriousness of risk for CHD in patients with diabetes. Because such patients frequently display multiple risk factors, even in the absence of CHD, and have the same risk for CHD as nondiabetics with CHD, ATP III defines diabetes as equivalent to existing CHD in terms of projecting coronary risk. Persons with diabetes who have an MI have an unusually high cardiovascular death rate, either in the short-term or long-term, and a more intensive therapeutic strategy is therefore warranted.

Also worth noting is the fact that although cardiologists have generally focused on the risks incurred by patients with a history of ischemic stroke, MI, and unstable angina, the new guidelines suggest that more attention be given to patients who have peripheral arterial disease, abdominal aortic aneurysm, symptomatic carotid artery disease, or who have undergone revascularization procedures. Risks for such patients are also considered to be equivalent to those of patients with known CHD. Certain patients who have multiple (2 or more) risk factors and a Framingham risk score of >20% over the next 10 years are classified as having a CHD risk equivalent because of the number and severity of their risk factors. Patients with multiple metabolic risk factors, also known as the metabolic syndrome, are candidates for more intensive lifestyle therapy.

Counting Major Risk Factors to Establish Therapeutic Goals

ATP III sets LDL-C goals based on the degree of patient risk. For example, in patients with CHD or CHD risk equivalents, such as diabetes and atherosclerotic disease, the risk for major coronary events within the next 10 years is greater than 20%. Treatment for such patients is intended to be aggressive, with the
goal of decreasing LDL-C to <100 mg/dL. Patients with 2 or more of the risk factors described in Table 2 are considered to be at intermediate risk, with the LDL-C goal being dependent on the individual’s degree of risk. For those whose 10-year risk for CHD is 10% to 20%, the LDL-C goal is <130 mg/dL, while those whose 10-year CHD risk is less than 10% have an LDL-C goal of 160 mg/dL. Patients with 1 or 0 risk factors generally have a 10-year risk of less than 10% and have a more lenient LDL-C goal of <160 mg/dL.

Detailed point scoring systems for men and women used to determine therapeutic goals is detailed by ATP III, using Framingham point score assessments of 10-year CHD risk intended for persons with 2 or more risk factors who do not have heart disease or a CHD risk equivalent (Figures 1 and 2). These scores are intended for patients who do not have known heart disease or a CHD risk equivalent, and are not intended as a substitute for a medical examination. Risk estimates are based on the number and severity of cardiovascular risk factors. Risk is calculated separately for men and women based on age, total cholesterol level, HDL-C level, systolic blood pressure, and cigarette smoking status.

Ten-year risk is determined by totaling the points the patient receives in all 5 major risk categories. The point total correlates with 10-year risk by percentage, as shown at the bottom of the figures. Increasing levels of total cholesterol add substantially to risk, as indicated by the point scores in each age category. ATP III terms a low HDL-C as an HDL-C level of 40 mg/dL. An important point to note is that the LDL-C level is not a component of risk score estimates simply because the Framingham database has far more information on total cholesterol and HDL-C levels.

ATP III has removed diastolic blood pressure measurements from risk scoring because systolic blood pressure has been identified as a more accurate predictor of CHD risk than diastolic blood pressure, particularly after age 50. At any blood pressure level, patients who use medication to lower their blood pressure are given more points because the presence of hypertension suggests that some degree of coronary endothelial damage may have already taken place. Cigarette smoking, especially at younger ages, is a potent risk factor for CHD, as demonstrated by the point scores from ages 20 through 49 years.

Prior to age 40, women receive a larger negative number for age than men do, and after age 64, women receive more points for age than men. For total cholesterol levels >200 mg/dL, women in all age categories receive higher point scores than men. Women receive more points than men for each increment of systolic blood pressure level >120 mm Hg. In addition, women who smoke receive a higher number of points than men in similar age categories until the age of 70, when this point total equalizes for both sexes.

For both men and women, the 10-year risk for a CHD event is determined by adding up the points for each of the 5 risk factors:

Points for age + Total cholesterol + HDL-C + Smoking status + Systolic blood pressure = Point total

The 10-year absolute risk for a hard CHD event is determined from the point total according to sex. The
point system differs significantly for men and women because, in general, men have a higher CHD risk than do women, particularly in middle age. For example, a point total of 15 for a woman is associated with a 3% absolute risk, whereas for a man it is associated with a 20% absolute risk (Figure 3).

**Diagnosing the Metabolic Syndrome**

ATP III defines the metabolic syndrome as a potential secondary target of therapy. In aggregate, the risk factors for the metabolic syndrome, shown in Table 3, enhance the risk for CHD. ATP III bases the diagnosis of the metabolic syndrome on the presence of 3 or more of the following risk factors: abdominal obesity, triglycerides ≥150 mg/dL, low HDL-C level (<40 mg/dL in men, <50 mg/dL in women), increased blood pressure (≥130/≥85 mm Hg), and fasting glucose ≥110 mg/dL. Note that ATP III raises the HDL-C cutpoint to less than 50 mg/dL in women for purposes of defining it as a risk factor. This upward adjustment reflects the general tendency for women to have higher HDL-C levels than men.

Management of the metabolic syndrome has 2 objectives: to reduce the underlying causes (obesity and physical inactivity) with intensified TLC; and to treat associated nonlipid and lipid risk factors. Because patients with the metabolic syndrome are frequently overweight or obese and sedentary, treatment should focus on weight reduction and increased physical activity. This approach addresses the underlying causes, obesity or overweight and physical inactivity, and reduces associated lipid and nonlipid risk factors. Reaching the LDL-C target level remains the primary goal of treatment.

ATP III places a new focus on elevated triglyceride levels, which have been significantly linked to the degree of heart disease risk. The new guidelines recommend lowering the accepted values for triglyceride levels with more emphasis on treating moderate elevations in triglyceride levels. According to the revised guidelines, a triglyceride level of <150 mg/dL is normal, 150 to 199 mg/dL is borderline high, 200 to 499 mg/dL is high, and ≥500 mg/dL or greater is very high. The primary aim of therapy for patients with elevated triglyceride levels is to reach the target LDL-C level, achieved with TLC for patients with levels >150 mg/dL. If the LDL-C goal is reached but the triglyceride level is >200 mg/dL, a second goal of non-HDL-C is set, with a target calculated by subtracting HDL-C from the total cholesterol level. The final number reflects serum concentrations of LDL-C plus very-low-density lipoprotein (VLDL). The non-HDL-C target is set at 30 mg/dL greater than the LDL-C target.

Drug treatment may be required to reach the non-HDL-C target, which might include higher doses of LDL-C-lowering drugs or the addition of nicotinic acid or fibrate to further lower VLDL. A similar treatment strategy is followed for low HDL-C (<40 mg/dL) if the triglyceride level is high (200 mg/dL or higher). However, in cases of isolated low HDL-C, physicians should consider treating with nicotinic acid or fibrate.

**The Role of C-Reactive Protein**

The role of highly sensitive C-reactive protein (hs-CRP) in predicting CHD risk is of particular interest to researchers. One recent analysis, based upon the Women's Health Study, of hs-CRP levels in patients...
with high total cholesterol levels shows that patients at highest risk for CHD have a high total cholesterol/HDL ratio and a high hs-CRP level. However, the study also suggests that regardless of hs-CRP level, the higher the total cholesterol/HDL ratio, the higher the patient's absolute CHD risk. The Air Force/Texas Coronary Atherosclerosis Prevention Study (AFCAPS/TEXCAPS) study showed that for patients with an LDL of < 150 mg/dL with a normal hs-CRP, statin therapy was not notably beneficial. However, for lower LDL levels and an above-average hs-CRP level, statins produced significant therapeutic benefits. Clearly, hs-CRP plays an increasingly important role in treatment of LDL-C since reliable data for both men and women suggests that elevated levels of hs-CRP are independently associated with increased risk. In fact, some data suggest that elevated hs-CRP can also increase the risk for MI and stroke, even in symptomatic patients. Markers of inflammation, such as hs-CRP, may also identify the highest-risk patients who present with acute coronary syndrome.

The relevance of CRP was first documented in 1988 in a landmark paper that refuted the premise that lesions with tight stenoses and small lumen were the most frequent cause of heart attacks. This study illustrated that mild to moderate lesions tend to have large lipid cores and thinner, fibrous caps. Most researchers believe that the shoulder regions of the edges of these fibrous caps contain a high concentration of activated macrophages and smooth muscle cells. Recent research suggests that if numerous inflammatory cells are found at the edges of the fibrous cap, one result may be an increase in CRP secreted by the liver. The current paradigm is that the macrophages lead to an increase in systemic cytokines in the circulation, such as IL-6 and TNF alpha, which prompts the liver to secrete CRP. In prediabetic patients, the adipose tissue also plays this role, causing an increased secretion of CRP.

The role of coronary calcification in predicting CHD risk and in primary prevention is also of interest. Research suggests that the adventitial layer of the arteries expands and plaque accumulates in the wall of the arteries as part of the aging process. With this compensatory expansion, a very high level of plaque burden may exist with the lumen still relatively preserved. In the patient with stable coronary disease, this condition may progress to increased encroachment of the lumen and, ultimately, the development of angina. In an asymptomatic middle-aged patient with a family history of heart disease and no signs of coronary calcification, lifestyle modification, rather than drug therapy, is considered by many cardiologists to be the most appropriate initial therapeutic approach for borderline hyperlipidemia. If that person is unable to optimize lipid levels with lifestyle changes alone, then statin therapy should be used to decrease the LDL-C level to < 130 mg/dL.

**Cutpoints for Treatment According to Risk Category**

Given the abundant clinical evidence showing that therapy for lowering LDL-C levels reduces CHD risk, the primary goals of therapy and the cutpoints for initiating treatment are stated in terms of LDL-C. ATP III defines cutpoints for initiating TLC and drug therapy according to category of risk (Table 4). TLC includes dietary modifications stressing low intake of saturated fats and cholesterol, increased consumption...

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### Table 4. ATP III: LDL-C Cutpoints for Treatment According to Risk Category

<table>
<thead>
<tr>
<th>Risk category (10-yr CHD risk)</th>
<th>LDL-C Level (mg/dL)</th>
<th>Goal</th>
<th>Initiate TLC</th>
<th>Consider drug therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD or CHD risk equivalent (&gt;20%)</td>
<td>&lt;100</td>
<td>≥100</td>
<td>≥130</td>
<td>100-129 drug optional</td>
</tr>
<tr>
<td>2+ risk factors (≤20%)</td>
<td>&lt;130</td>
<td>≥130</td>
<td>10-year risk: 10-20%</td>
<td>≥130</td>
</tr>
<tr>
<td>0-1 risk factor (≤10%)</td>
<td>&lt;160</td>
<td>≥160</td>
<td>10-year risk: &lt;10%</td>
<td>≥160</td>
</tr>
</tbody>
</table>

TLC = Therapeutic lifestyle changes.

of plant stanol and sterols, weight reduction, and increased physical activity.

In the highest-risk category, the guidelines advise initiating drug therapy concurrently with TLC if baseline LDL-C is \( \geq 130 \) mg/dL. For LDL-C levels between 100 and 129 mg/dL, the use of drug therapy relies on clinical judgment. Some authorities recommend use of LDL-C–lowering drugs if LDL-C is > 100 mg/dL. This is supported by the recent presentation of the Heart Protection Study at the 2000 American Heart Association Meeting. If the borderline LDL-C is accompanied by low HDL and elevated triglycerides, some prefer drugs that modify HDL-C or triglyceride levels, such as nicotinic acid or fibrates as the initial therapy.

For patients with multiple risk factors, the cutpoint for initiating TLC is an LDL-C level of 130 mg/dL or higher. Two subcategories of risk determine when drug therapy should be considered, as shown in Table 4. While patients with 0 to 1 risk factors are generally advised to undertake TLC, drug therapy should be considered when LDL-C is \( \geq 190 \) mg/dL. For those with an LDL-C level of 160 to 189 mg/dL, drug therapy is an option based on clinical judgment. The presence of a severe single risk factor (cigarette smoking, significant hypertension, strong family history of premature CHD, or very low HDL-C) or a 10-year risk approaching 10% would favor the use of drugs to reach an LDL-C level of \(< 160\) mg/dL and probably \(< 130\) mg/dL.

**The Heart Protection Study**

A landmark study, The Heart Protection Study, will likely change the way many clinicians practice lipid-lowering therapy. Inclusion criteria for the study included men and women between the ages of 40 and 80 who either had CHD, diabetes, or hypertension and total cholesterol levels between 130 and 270 mg/dL, in whom the treating physician did not believe statins or vitamins were clearly indicated. Patients were randomized to a drug treatment group of 40 mg of simvastatin or simvastatin placebo, and to an antioxidant vitamin cocktail treatment group, or a vitamin placebo group. Among the 20,000 subjects, compliance was a significant issue, with nearly 10% terminating statin therapy after year 1. This was largely due to the difficulty that some participants had complying with regularly scheduled 3-month follow-up appoint-

ments. In the placebo group, by year 3, 20% of the subjects had initiated statin therapy as the treating physicians kept abreast of emerging scientific evidence supporting the therapeutic value of statin use. The number within the placebo group who changed to statin therapy increased to 25% by year 3, and to 33% by year 4, significantly decreasing the statistical power of the study to show a difference in mortality between the statin and statin placebo groups.

In the beginning of year 1, the average LDL reduction was about 60 mg/dL in the statin group, and as each year progressed with an increasing number of placebo subjects taking statins, the differential reduction in LDL decreased proportionately. By the end of the trial, only a 30 mg/dL difference in LDL-C was observed between the statin and statin placebo groups. Despite this small overall reduction, the intention-to-treat analysis showed that the incidence of all strokes was reduced by about 27% in the statin therapy arm, with significantly fewer hemorrhagic strokes in the statin group.

Even with the relatively high number of subject drop-ins and drop-outs, major coronary and cardiovascular events were reduced by 25% to 30% overall. For those who were compliant with their assigned therapy, the reduction was about 35%. As time went on, a greater separation in the curves between statin and statin placebo was observed.

Interestingly, the effect of the statin on vascular events by prior disease apparently had little or no impact on the incidence of prior MI or other vascular disease. Patients still showed a 25% reduction in major vascular events—even those with existing diabetes or peripheral vascular disease. An examination of the correlation between age and incidence of disease also shows some very interesting results: subjects older than 75 years of age tended to have a greater risk reduction than younger patients, and females had comparable risk reductions to males. Even more striking is the fact that even subjects whose LDL-C level was \(< 100\) mg/dL had a 25% reduction in vascular events with statin therapy. Statins and placebo groups exhibited a nearly identical safety profile, measured by liver function tests. The main conclusions drawn from the study, presented at the American Heart Association meeting, were that after taking into consideration the noncompliance among study subjects, 40 mg of statin safely reduced the risk of MI, stroke, and revascularization by about one third. Therefore, 5 years of statin treatment may be estimated to prevent major vascular events in approximately 100/1000 patients with...
prior MI, 70/1000 with diabetes, and 70/1000 with prior peripheral artery disease or stroke. Vitamins vs vitamin placebo showed no beneficial effects among study subjects.

**ABCs of Cardiovascular Disease Risk Management**

Our group at the Ciccarone Center for Prevention of Heart Disease at The Johns Hopkins University School of Medicine has developed a checklist, “ABCs of Preventive Cardiovascular Disease Risk Factor Management,” based on recommendations from the American Heart Association and the American College of Cardiology (Table 5).

**A: Antiplatelets and Anticoagulants**

Aspirin is the prototypical antiplatelet agent based on its proven efficacy, safety, and low cost when it comes to reducing cardiovascular disease (CVD) risk.

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**Table 5. ABCs of Cardiovascular Disease Risk Factor Management**

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Agents</th>
<th>Treatment Goals</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A: Antiplatelets/anticoagulants</strong></td>
<td>Aspirin</td>
<td>All high-risk patients should be treated with one of these agents, especially diabetics, to optimize blood pressure.</td>
</tr>
<tr>
<td>ACES/ARBs</td>
<td>Clopidogrel</td>
<td>Relief of anginal symptoms to allow patient to exercise.</td>
</tr>
<tr>
<td></td>
<td>Warfarin, eg, ramipril, losartan</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nitrates Long-acting CCBs Beta-blockers</td>
<td></td>
</tr>
<tr>
<td><strong>B: Blood pressure control</strong></td>
<td>Diuretics, beta-blockers ACE inhibitors, ARBs, CCBs</td>
<td>Aim for blood pressure &lt;130/80 mm Hg</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>eg, metoprolol</td>
<td>Especially post-MI to achieve resting heart rate ≤60</td>
</tr>
<tr>
<td><strong>C: Cholesterol management</strong></td>
<td>Statins, bile resins Fibrates Nicotin</td>
<td>LDL &lt;100 mg/dL if CAD, PVD, DM; LDL&lt;130 mg/dL if ≥2 CRFs; &lt;160 mg/dL if 0-1 CRFs “normal PG less than 150”</td>
</tr>
<tr>
<td></td>
<td></td>
<td>TG &lt;150 mg/dL; HDL ≥40 men; ≥ 50 women</td>
</tr>
<tr>
<td>Cigarette-smoking cessation</td>
<td>Bupropion Nicotine replacement</td>
<td>Long-term smoking cessation</td>
</tr>
<tr>
<td></td>
<td>Behavioral modification</td>
<td></td>
</tr>
<tr>
<td><strong>D: Dietary/weight counseling</strong></td>
<td>Low-saturated fat diet supplemented with fruits and vegetables</td>
<td>Achieve optimal BMI</td>
</tr>
<tr>
<td>Diabetes management</td>
<td>Metformin, glitazones Insulin</td>
<td>Achieve HbA1C &lt;7%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Improve physical fitness and optimize other CAD risk factors</td>
</tr>
<tr>
<td><strong>E: Exercise</strong></td>
<td>Less than 3x/week; &gt;30 min/d</td>
<td></td>
</tr>
<tr>
<td>Education of patient/families</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; CCB = calcium-channel blocker; EF = ejection fraction; MI = myocardial infarction; LDL = low-density lipoprotein; CAD = coronary artery disease; PVD = pulmonary vascular disease; DM = diabetes mellitus; CRF = corticotropin-releasing factor; TG = triglyceride; HDL = high-density lipoprotein; BMI = body mass index; HbA1C = glycosylated hemoglobin.

Source: Table adapted from *Cardiology in Review*. 2001;9(2).
The higher an individual’s absolute vascular event risk, the more beneficial aspirin appears to be. The role of aspirin in primary prevention has also been demonstrated in prospective trials and meta-analyses.14 Newer antiplatelet agents, such as clopidogrel, may provide more effective inhibition of platelet aggregation. These 2 agents can be combined in high-risk patients. Anticoagulants may be prescribed in conjunction with, or in place of, antiplatelet agents in secondary prevention. For patients with a strong indication for anticoagulants, warfarin is the usual choice.

A: ANGIOTENSIN-CONVERTING ENZYME (ACE) INHIBITORS/ANGIOTENSIN RECEPTOR BLOCKERS (ARBs)

The data supporting the value of ACE inhibitors and ARBs is especially strong for patients with diabetes or impaired ejection fractions, as demonstrated in the Heart Outcomes Prevention Evaluation (HOPE) study.15 More recent studies with ARBs report a decrease in proteinuria and renal decline.16 Data in the HOPE study suggests that patients with diabetes benefited even more than nondiabetics, with 24% reduction in deaths among patients with diabetes treated with an ACE inhibitor.

B: BLOOD PRESSURE AND BETA-BLOCKERS

For patients with hyperlipidemia, the goal should be a normal blood pressure of < 130/85 mm Hg, and even lower for patients with diabetes (< 130/80 mm Hg). The data supporting the value of beta-blockers is especially strong in post-MI patients, those with impaired left ventricular ejection fraction, or those with inducible ischemia.

C: CHOLESTEROL

As previously discussed, the newest guidelines advise striving for an optimal LDL-C level of < 100 mg/dL, a normal triglyceride level of < 150 mg/dL, and an HDL-C level above 40 mg/dL. If the patient smokes, nicotine replacement and medications, such as bupropion or wellbutrin, in addition to behavior modification, is strongly recommended.

D: DIABETES AND DIET

From the cardiologist’s point of view, a desirable HbA1C should certainly be < 7%, with a normal fasting blood sugar level of < 110 mg/dL. Healthcare providers need to better stress the value of a diet that is low in saturated fat, rich in fruits, fiber, and vegetables, and restricted in calories, to achieve a normal body mass index. Weight loss can be best maintained if it is done slowly, at a suggested rate of 2-3 pounds per month.

E: EXERCISE AND EDUCATION

Patients must be reminded of the value of exercise, such as brisk walking, at least 30 minutes 3 times per week. Educating patients and their families is important, emphasizing the tremendous advancements that have been made in lipid management and preventive cardiology as a whole.

CONCLUSION

Because so much scientific evidence now supports aggressive management of hyperlipidemia and hypertension, physicians should be doing a much better job of decreasing patient risks for heart attack and strokes. Education is needed not only for patients, but for their health care professionals, as well. Study after study shows that patients continue to be undertreated. Let us not be seduced by what we think we know, because in practice, we are not doing nearly as well as we might.

REFERENCES


