

- gum as an adjunct to physician's advice against cigarette smoking. *JAMA* 1986;256:1315-8.
29. Tsevat J. Impact and cost-effectiveness of smoking interventions. *Am J Med* 1992;93:43S-7S.
 30. Krumholz HM, Cohen BJ, Tsevat J, Pasternak RC, Weinstein MC. Cost-effectiveness of a smoking cessation program after myocardial infarction. *J Am Coll Cardiol* 1993;22:1697-702.
 31. Oldridge NB, Guyatt GH, Fischer ME, Rimm AA. Cardiac rehabilitation after myocardial infarction. Combined experience of randomized clinical trials. *JAMA* 1988;260:945-50.
 32. O'Connor GT, Buring JE, Yusuf S, et al. An overview of randomized trials of rehabilitation with exercise after myocardial infarction. *Circulation* 1989;80:234-44.
 33. Oldridge N, Furlong W, Feeny D, et al. Economic evaluation of cardiac rehabilitation soon after acute myocardial infarction. *Am J Cardiol* 1993;72:154-61.
 34. Berlin JA, Colditz GA. A meta-analysis of physical activity in the prevention of coronary heart disease. *Am J Epidemiol* 1990;132:612-28.
 35. Wood PD, Stefanick ML, Dreon DM, et al. Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. *N Engl J Med* 1988;319:1173-9.
 36. Arroll B, Beaglehole R. Does physical activity lower blood pressure: a critical review of the clinical trials. *J Clin Epidemiol* 1992;45:439-47.
 37. Consensus Development Conference on Diet and Exercise in Non-Insulin-Dependent Diabetes Mellitus. National Institutes of Health. *Diabetes Care* 1987;10:639-44.
 38. Wood PD, Stefanick ML, Williams PT, Haskell WL. The effects on plasma lipoproteins of a prudent weight-reducing diet, with or without exercise, in overweight men and women. *N Engl J Med* 1991;325:461-6.
 39. Grady D, Rubin SM, Petitti DB, et al. Hormone therapy to prevent disease and prolong life in postmenopausal women. *Ann Intern Med* 1992;117:1016-37.
 40. Tosteson AN, Rosenthal DI, Melton LJ, Weinstein MC. Cost effectiveness of screening perimenopausal white women for osteoporosis: bone densitometry and hormone replacement therapy. *Ann Intern Med* 1990;113:594-603.
 41. Cairns JA, Markham BA. Economics and efficacy in choosing oral anticoagulants or aspirin after myocardial infarction. *JAMA* 1995;273:965
 42. Glick H, Cook J, Bourassa M, et al. Projections of the costs and benefits of enalapril treatment in patients with symptomatic heart failure [abstract]. *J Am Coll Cardiol* 1994;23:284A.
 43. Goldman L, Sia STB, Cook EF, Rutherford JD, Weinstein MC. Costs and effectiveness of routine therapy with long-term beta-adrenergic antagonists after acute myocardial infarction. *N Engl J Med* 1988;319:152-7.
 44. Wong JB, Sonnenberg FA, Salem DN, Pauker SG. Myocardial revascularization for chronic stable angina: Analysis of the role of percutaneous transluminal coronary angioplasty based on data available in 1989. *Ann Intern Med* 1990;113:852-71.
 45. Weinstein MC, Stason WB. Cost-effectiveness of coronary artery disease. *Circulation* 1982;66 Suppl III:III-56-66.
 46. Hatzianreou EI, Koplan JP, Weinstein MC, Caspersen CJ, Warner KE. A cost-effectiveness analysis of exercise as a health promotion activity. *Am J Public Health* 1988;78:1417-21.
 47. Tsevat J, Duke D, Goldman L, et al. Cost-effectiveness of captopril therapy after myocardial infarction. *J Am Coll Cardiol* 1995;26:914-9.
 48. Doubilet P, McNeil BJ, Weinstein MC. The decision concerning coronary angiography in patients with chest pain: a cost-effectiveness analysis. *Med Decis Making* 1985;5:293-309.
 49. Fineberg HV, Scadden D, Goldman L. Care of patients with a low probability of acute myocardial infarction: cost effectiveness of alternatives to coronary-care-unit admission. *N Engl J Med* 1984;310:1301-7.
 50. Sox HC, Blatt MA, Higgins MC, Marton KI. *Medical Decision Making*. Stoneham (MA): Butterworth, 1988.
 51. Oster G, Epstein AM. Primary prevention and coronary heart disease: the economic benefits of lowering serum cholesterol. *Am J Public Health* 1986;76:647-56.

Task Force 7. Evaluation and Management of Risk Factors for the Individual Patient (Case Management)

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Several factors that increase risk for atherosclerosis in general, and coronary disease in particular, have been identified and are generally accepted in medical practice as valid and relevant (1). These include cigarette smoking, sedentary life-style, hypertension, abnormal blood lipid levels and a thrombogenic tendency, among others. However, conclusive evidence from population-based studies of reduction of all-cause mortality by modification of such risk factors has been lacking (2-4). Nevertheless, significant (although modest) reduction in coronary artery disease (CAD) mortality and morbidity has been found in these population studies. This benefit is greatest in patients with evidence of coronary disease, or in studies in which the control subjects are at a mortality risk of 4% or greater per year (5,6).

The term prevention—"primary" or "secondary"—is widely used to encompass reduction of these common risk factors in normal persons or in patients with coronary heart disease, respectively. "Primary prevention" implies becoming a non-smoker, maintaining a normal blood pressure and a desirable body weight and consuming no more than 10% of calories from saturated fat. In these healthy people, behavior modification, diet and exercise programs are designed to promote a healthy life-style. Also, these measures will allow some people to avoid or delay the development of atherosclerosis. "Secondary prevention" relates to the treatment of coronary artery disease in patients who have had a clinical event (for example, angina pectoris or acute myocardial infarction) by reduction of conventional risk factors but also includes the use of cardio-

Table 1. Priorities of Coronary Heart Disease Prevention in Clinical Practice

1. Patients with established CHD or other atherosclerotic vascular disease
2. Asymptomatic subjects with particularly high risk (subjects with severe hypercholesterolemia or other forms of dyslipidemia, diabetes or hypertension; subjects with a cluster of several risk factors)
3. Close relatives of patients with early-onset CHD or other atherosclerotic vascular disease; asymptomatic subjects with particularly high risk
4. Other subjects met in connection with ordinary clinical practice

CHD = coronary heart disease.

active, vasoactive, lipid-lowering and other drugs. Clearly, this activity properly belongs within the more formal structure of clinical practice. The designation of this activity as "prevention" is misleading (7,8) and a preferable description would be "the medical management of coronary atherosclerosis." As a practical matter, "preventive" care services may not be compensated in many insurance and managed care programs. Further, the qualifier "secondary" carries an implication of lesser importance to patient and provider. Hence, although these terms have been established by epidemiologic convention, we prefer the designation "optimal medical management" in this task force report.

The Patient With Coronary Artery Disease

Atherosclerosis is a complex polygenic, multifactorial vascular disorder associated with many differing and changing metabolic, anatomic and clinical manifestations (9). Two markedly differing general mechanisms—instability of the coronary artery wall in an area of nonobstructive atherosclerosis, and the severity of vascular obstruction locally or in sum—are responsible for ischemic symptoms and adverse events (10,11). Instability of the coronary endothelial surface is associated with the inflammatory reaction, the lipid content of the atherosclerotic plaque, the state and thickness of the fibrous cap, local fluid dynamics and vasomotion, as well as with lipid levels. Readily definable characteristics other than a prior coronary event also place other subjects at high risk (Table 1). These groups include individuals with familial hypercholesterolemias, a strong family history of premature coronary artery disease, diabetes mellitus, other lipid profile abnormalities, hypertension, obesity and adverse behavior practices, such as excessive cigarette smoking alone or in combination. In these subjects, the probability of adverse events, including myocardial infarction, is great. In patients with coronary disease, risk factor modification has proved to be of benefit in regard to all-cause mortality and cardiovascular mortality and morbidity, and in several recent studies the reduction in disease and death is of a sufficient magnitude to be important to individual patients over a relatively short time. This may reflect improved endothelial cell function, plaque stabilization and altered coagulation—alone or in combination. Major or minor genetic traits, abnormalities of vascular biology, hemostasis and glucose intolerance, among others, interrelate in a complex manner. The detailed management of dyslipidemias in at-risk

patients requires a thorough understanding of these interrelationships and of the clinical pharmacology of lipid-lowering drugs. Thus, treatment strategies may differ importantly between individual patients, and mandate a high standard of physician knowledge, skill, motivation and performance.

Patients with clinical angina pectoris have a high probability of an obstruction of at least one major coronary artery. While those who have sustained an infarction have demonstrated a propensity to thrombosis, all patients with coronary artery disease and those with an adverse coronary profile are at increased risk for further adverse outcomes. Risk factor modifications favorably affect several processes central to atherogenesis and thrombosis. Serum fibrinogen, a powerful predictor of coronary events (12), is now undergoing study. Future research will serve to define the details of adverse platelet function, fibrinogen and antifibrins, plasminogen activators and inhibitors, the trigger role of the autonomic nervous system (13), the composition of the vessel wall and the reactions of the coronary endothelial surface. Risk factors, known and unknown, and their interaction one with the other are sufficiently complex as to require special expertise in their analysis and details of management. In the future, as these matters are resolved, the therapeutic strategies will change dramatically, and will prove to be more specific, effective and efficient.

The prognosis of established coronary artery disease appears to be improving. This in turn exerts an impact on the predictive accuracy of current measures of risk stratification. New demographic information, and more sensitive noninvasive and invasive testing methods, are now available. Although extrapolation from older data bases may not be ideal, it will have to suffice until new data bases are available. Despite these favorable trends in prognosis, it is now clear that effective management of common risk factors in patients with coronary artery disease will result in further improvement in the frequency and severity of subsequent coronary events.

The Current Dilemma

Despite the scientific evidence for the significance of common risk factors on outcomes and the availability of effective remedies, the ongoing long-term medical management of the patient with coronary disease remains fragmented and is often unsatisfactory (8). Patients with coronary disease receive their care from both general physicians and cardiovascular specialists. The latter usually participate in the acute management of unstable angina pectoris and myocardial infarction, perform diagnostic testing and participate in invasive revascularization procedures. The former are concerned primarily with longer term medical management in the clinically quiescent phase. Despite the overwhelming evidence as to benefit, effective risk reduction strategies are not consistently pursued (14,15). In many instances, cardiovascular specialists are best positioned to direct the patient with coronary disease in or into a long-term management plan, yet fail to do so. Many general physicians and some cardiovascular specialists do not seem to

be aware that patient benefit requires a patient-specific plan, a regular follow-up to ensure attainment of specific goals, modification of goals and treatment strategies when necessary and a lifelong commitment. Both general physicians and cardiovascular specialists require careful initial and continuing medical education in the evaluation and management of risk factors in the patient with coronary disease.

The Objective

Recognizing that cardiovascular disease management requires insights into the entire spectrum of disease manifestations from initiation through quiescence to acute illness, a commitment now to reducing adverse outcomes by favorably altering risk factors is crucial. The objectives of risk reduction strategies are to reduce all-cause and cardiovascular mortality, ischemic symptoms, myocardial infarction, arrhythmias, heart failure, other manifestations of atherosclerosis and to improve general health. Although benefit in the elderly has been questioned (16), evidence for benefit of risk factor reduction in other patient groups is overwhelming (17-21).

Implementation

Whether in the acute or quiescent phase of cardiovascular disease, effective and efficient management requires a well founded clinical strategy described in a clear protocol and an integrated health care delivery system, as, for example, in approaches to the emergent management of the patient with new-onset chest pain. Within such a system, which aligns the patient with physicians, other health care personnel and the insurer, positive incentives are necessary so that the necessary attention is directed to risk stratification and effective risk reduction. A "system" may be implemented by solo practitioners, small group practices, hospital-based or other clinics, as well as by large providers, as, for example, managed care organizations or the Department of Veterans Affairs. *System* implies the organizational structure necessary to implement an effective, efficient management protocol using resources available and appropriate to patient need. For example, a trained nurse practitioner working closely with a motivated primary care physician might require advice from an independent qualified dietitian from time to time, whereas a large clinic could employ such personnel. Likewise, a general internist might have an arrangement for periodic review by a cardiovascular specialist to implement an improved evaluation and new treatment approaches, including drug therapy. In other circumstances a larger organization would provide comprehensive in-house cardiology services. The cardiovascular specialist is also qualified to provide the necessary expertise in the direct management of the acute coronary syndromes and their complications.

The Clinical Strategy

The clinical strategy requires a specific protocol to clarify logistic issues and responsibilities. Risk factor assessment and

current cardiac status (the necessary data sets for initial evaluation and regular follow-up) and further invasive or noninvasive testing are among the clinical matters to be implemented. Goals for blood pressure, blood lipids, nutrition, physical activity and stress levels are required for patient education and behavior modification. Knowledge of the effects, side effects, dosing protocols and monitoring of drugs used to control blood pressure, reduce lipids and modify coagulation processes must be available.

Such a strategy mandates the development of a long-term program that includes an initial risk assessment (including the impact of existing cardiac or other disease), a specific treatment plan and an outcomes-based long-term assessment for each patient as an individual, and an effective follow-up for compliance. Specially trained physicians (e.g., cardiologists, internists and family practitioners) must attain those qualifications necessary to provide the expertise and direction in risk assessment and long-term management of patients with cardiovascular disease (22). It is mandatory for physicians who take such leadership responsibilities to become knowledgeable of the basic relations of lipids, hemostatic and other factors related to atherosclerosis and vascular stability. These responsible physicians must design and, if necessary, modify the details of long-term management, even when it is to be delivered by other health care providers. Expert ongoing review is required as new information emerges to ensure that management strategies continue to correct or favorably modify risk factors, particularly at times when untoward events occur. The concept that an appropriate level of care can be provided by health care personnel with a minimal understanding of these complex processes is naive, and could prove inadequate for many individual patients.

Initial Evaluation

Each patient determined to have or to be at risk for coronary artery disease will require an individualized evaluation and an identification of the contribution of the various metabolic, hemostatic, hormonal and autonomic nervous system elements contributing to that risk. Low socioeconomic status must also be taken into account as an important predictor of cardiovascular risk. In addition to a careful medical history and physical examination, a detailed patient summary is required regarding coronary disease and death in kindred, and accurate blood pressure, body weight and height measurements. A 12-lead electrocardiogram is necessary. Laboratory studies must include at least one fasting lipid profile, including total cholesterol, high density lipoprotein (HDL) cholesterol, total/HDL cholesterol ratio, triglycerides, calculated low density lipoprotein (LDL) cholesterol and fasting blood glucose values (Fig. 1). Isolated total serum cholesterol values alone may be misleading. In the future, other laboratory studies, including lipoprotein(a), fibrinogen and ferritin levels as well as genetic markers may refine even more accurately the establishment of individual risk. When appropriate, functional stress testing is useful to define physi-

Risk Elements

Age _____; Gender _____

History of Preventive Coronary Disease in a First-Degree Relative? (Male <55 years; female <65 years) Yes _____; No _____

Socioeconomic status? Low _____; Not Low _____

Weight, Height? _____ lbs.; _____' _____" (ft., inches)

Total Cholesterol, _____ mg/dl; Triglycerides, _____ mg/dl; LDL, _____ mg/dl; HDL, _____ mg/dL

Smoking? _____ >20 cigarettes/day; _____ <20 cigarettes/day; _____ former; _____ never

Sedentary? Type of exercise _____; Number of times per week _____

Hypertension? Yes _____; No _____; If yes, _____ controlled; _____ not controlled

Diabetes Mellitus? Yes _____; No _____; If yes, _____ insulin requiring; _____ non-insulin requiring

Evidence of Atherosclerosis

Carotid disease? _____ present; _____ absent

Peripheral vascular disease? _____ present; _____ absent

Other vascular disease? _____ present; _____ absent

Coronary disease?

Prior coronary event? _____ MI; _____ Stable angina; _____ Unstable angina; _____ PTCA; _____ CABG

Angiographic results? _____ 1VD; _____ 2VD; _____ 3VD

Stress Test with/without perfusion imaging? _____ ischemia; _____ no ischemia

Left ventricular function? _____ Normal; _____ Mildly/moderately reduced; _____ Severely reduced

Dysrhythmias? _____ A trial; _____ VT; _____ Prior VF

Comorbid Conditions

Likely to Limit Life _____

Figure 1. Initial risk appraisal. CABG = coronary artery bypass graft surgery; HDL = high density lipoprotein cholesterol; LDL = low density lipoprotein cholesterol; MI = myocardial infarction; PTCA = percutaneous transluminal coronary angioplasty; VD = vessel disease; VF = ventricular fibrillation; VT = ventricular tachycardia.

cal work capacity and blood pressure response in addition to evidence for inducible ischemia. The presence, frequency and characteristics of arrhythmias at rest and during stress may have important prognostic significance. As defined by the clinical presentation and preliminary stratification, additional appropriate testing may include coronary angiography, stress echocardiography and other imaging techniques. In the future, new imaging techniques may allow screening for the presence of anatomic atherosclerosis in the asymptomatic but at-risk patient.

Establishment of Risk

In the patient with coronary artery disease, individual risk is influenced by multiple conditions other than conventional risk factors, including age, prior infarction, left ventricular dysfunction, arrhythmias, other systemic conditions (including diabetes mellitus, cerebral or peripheral vascular disease, emphysema and additional comorbid states), as well as response to prior interventions. The risk of death, cardiovascular morbidity, and an undesirable quality of life cannot be quantified from

Risk Intervention	Recommendations			
Smoking: <u>Goal</u> complete cessation	Strongly encourage patient and family to stop smoking. Provide counseling, nicotine replacement, and formal cessation programs as appropriate.			
Lipid management: <u>Primary goal</u> LDL<100 mg/dL <u>Secondary goals</u> HDL>35 mg/dL; TG<200 mg/dL	Start AHA Step II Diet in all patients: ≤30% fat, <7% saturated fat, <200 mg/dL cholesterol. Assess fasting lipid profile. In post-MI patients, lipid profile may take 4 to 6 weeks to stabilize. Add drug therapy according to the following guide:			
	LDL<100 mg/dL No drug therapy	LDL 100 to 130 mg/dL Consider adding drug therapy to diet, as follows:	LDL>130 mg/dL Add drug therapy to diet, as follows: Suggested drug therapy TG <200 mg/dL TG 200 to 400 mg/dL TG >400 mg/dL Statin Statin Consider combined Resin Niacin drug therapy (niacin, Niacin fibrate, statin)	HDL<35 mg/dL Emphasize weight management and physical activity. Advise smoking cessation. If needed to achieve LDL goals, consider niacin, statin, fibrate.
Physical activity: <u>Minimum goal</u> 30 minutes 3 to 4 times per week	Assess risk, preferably with exercise test, to guide prescription. Encourage minimum of 30 to 60 minutes of moderate-intensity activity 3 or 4 times weekly (walking, jogging, cycling, or other aerobic activity) supplemented by an increase in daily lifestyle activities (eg, walking breaks at work, using stairs, gardening, household work). Maximum benefit 5 to 6 hours a week. Advise medically supervised programs for moderate- to high-risk patients.			
Weight management:	Start intensive diet and appropriate physical activity intervention, as outlined above, in patients >120% of ideal weight for height. Particularly emphasize need for weight loss in patients with hypertension, elevated triglycerides, or elevated glucose levels.			
Antiplatelet agents/ anticoagulants:	Start aspirin 80 to 325 mg/d if not contraindicated. Manage warfarin to international normalized ratio=2 to 3.5 for post-MI patients not able to take aspirin.			
ACE inhibitors post-MI:	Start early post-MI in stable high-risk patients (anterior MI, previous MI, Killip class II [S ₃ gallop, rales, radiographic CHF]). Continue indefinitely for all with LV dysfunction (ejection fraction≤40) or symptoms of failure. Use as needed to manage blood pressure or symptoms in all other patients.			
Beta-blockers:	Start in high-risk post-MI patients (arrhythmia, LV dysfunction, inducible ischemia) at 5 to 28 days. Continue 6 months minimum. Observe usual contraindications. Use as needed to manage angina rhythm or blood pressure in all other patients.			
Estrogens:	Consider estrogen replacement in all postmenopausal women. Individualize recommendation consistent with other health risks.			
Blood pressure control: <u>Goal</u> ≤140/90 mm Hg	Initiate lifestyle modification—weight control, physical activity, alcohol moderation, and moderate sodium restriction—in all patients with blood pressure>140 mm Hg systolic or 90 mm Hg diastolic. Add blood pressure medication, individualized to other patient requirements and characteristics (ie, age, race, need for drugs with specific benefits) if blood pressure is not less than 140 mm Hg systolic or 90 mm Hg diastolic in 3 months or if initial blood pressure is >160 mm Hg systolic or 100 mm Hg diastolic.			

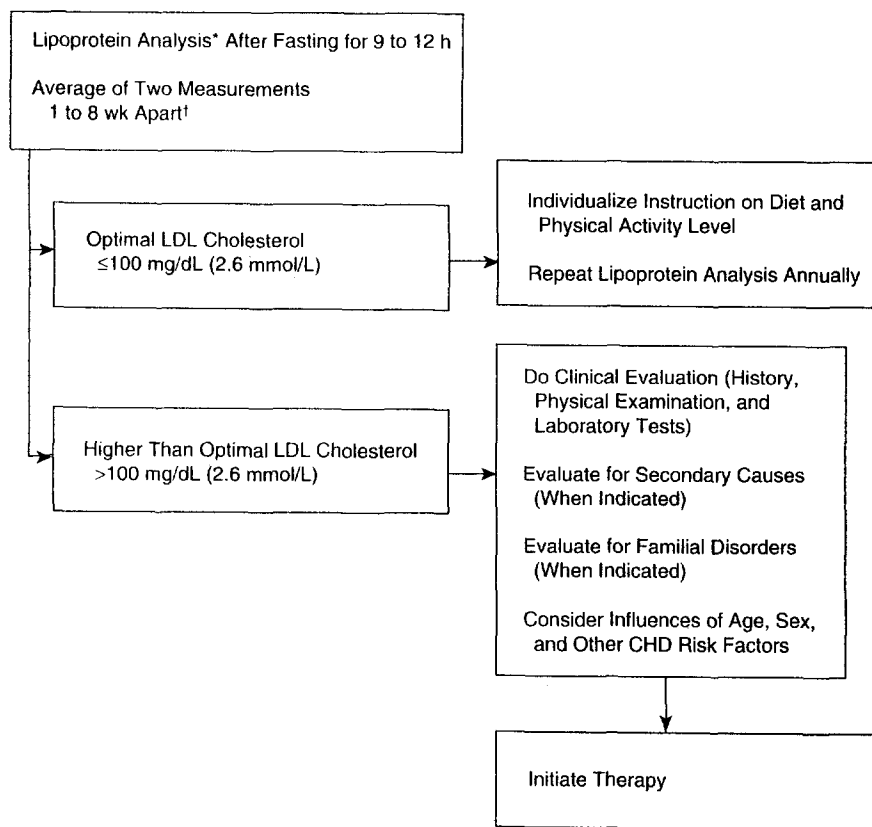
Figure 2. Guide to comprehensive risk reduction for patients with coronary and other vascular disease. ACE = angiotensin-converting enzyme; AHA = American Heart Association; LV = left ventricular; TG = triglycerides; other abbreviations as in Figure 1. Reprinted with permission from Smith et al. (24).

epidemiologic data alone with a precision sufficient to manage the individual patient.

After an initial comprehensive evaluation, it is possible to assign a relative hazard as a baseline to develop an appropriate

management strategy. The stroke and cardiac event risk factor prediction charts (see tables in Task Force 5), based on the Framingham Heart Study (23) of initially normal persons, is helpful, but not specific for the patient with coronary disease. Every patient who has had a cardiac event is at increased risk and deserving of assessment and management. Other factors listed previously must be integrated by the physician to provide a more realistic general prognosis (see Tables 5 to 8 and Figure 3 in Task Force 5).

Figure 3. Guide to lipid management in patients with established cardiovascular disease. *Lipoprotein analysis should be performed when the patient is not in the recovery phase from an acute coronary or other medical event that would lower the usual low density lipoprotein cholesterol (LDL) levels. †If the first two low density lipoprotein cholesterol test results differ by >30 mg/dl (0.7 mmol/liter), a third test should be obtained within 1 to 8 weeks and the average value of the three tests used. CHD = coronary heart disease. Reprinted with permission from reference 30.



As discussed in Task Force 6, risk factor modification is most cost effective and most efficient in patients with coronary disease, less so for patients with multiple severe risk factor abnormalities. Younger patients with mild angina, small infarcts, as well as those who previously have undergone revascularization, are most appropriate for efforts to reduce and improve adverse risk characteristics. Less favorable outcomes are to be expected in the elderly, patients with extensive irreversible cardiac damage or those with other organ system damage.

Ongoing Care

Initially, several physician visits may be necessary to establish an optimal management strategy, monitor responses and adjust subsequent treatment. Implementation and maintenance of longer term care in conjunction with the primary care physician usually is appropriate, with follow-up by the cardiovascular specialist for unanticipated changes in clinical events or further guidance in overall management. Data concerning blood pressure, body weight, lipid and glucose values should be reviewed at regular intervals (annually or semiannually). It is essential that the patient recognize the overall nature of the plan, the need for a team approach and his or her essential and long-term role in its accomplishment. Management is a lifetime process, as in patients with a variety of other chronic diseases. A continuum of reliable information relative to the initial level of risk and the efficacy of interventions in the

modification of risk is essential. An appropriate lifetime flow-chart is a simple task with current computer technology and can function as a valuable motivator for both patient and physician.

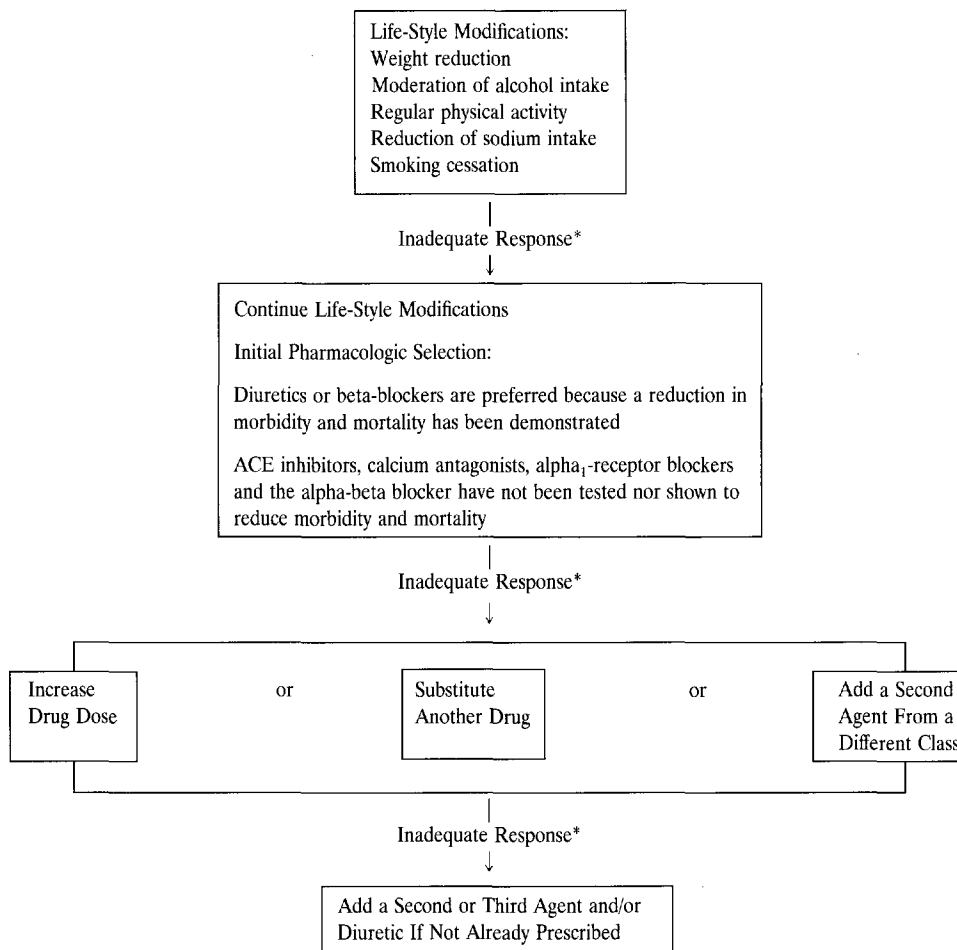
Management Strategies

A detailed and comprehensive discussion of management strategies is outside the scope of this task force report. Several carefully considered published recommendations provide useful guidelines. General guidelines from the American Heart Association and endorsed by the American College of Cardiology are presented in Figure 2 (24,25).

Brief comments on the risk factors designated category I (Task Force 3) follow:

Smoking. Other than for those addicted, cigarette smoking is without redeeming social value in the population at large and is the single most obvious and important risk factor for coronary heart and vascular disease (26). It must be eliminated in the management of all patients at high risk for or with evident coronary artery disease. Increased taxation on the product, restriction of smoking in communal environments and other policy level inhibitors of smoking continue to confer significant benefits to patients with coronary disease and to the general population.

Dietary regulation. For many years diet modification, in particular, reduction in the consumption of animal fat, has been a cornerstone of cholesterol control. Over the years, the



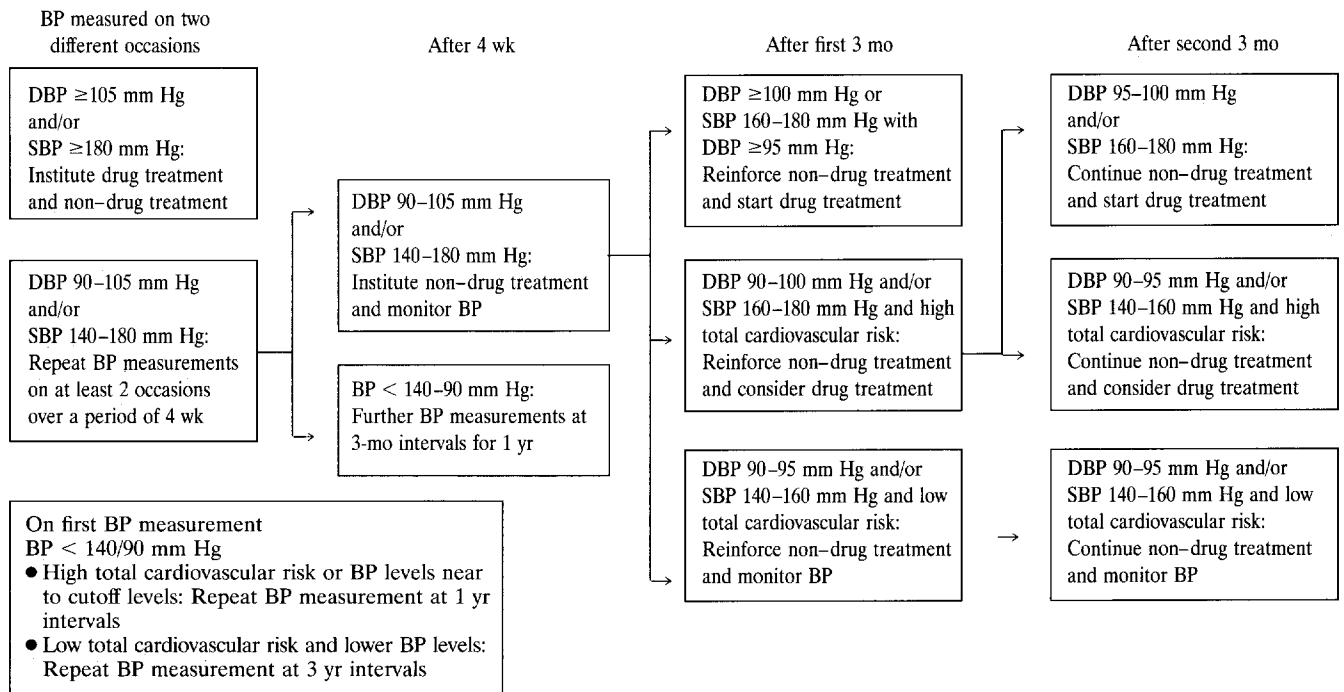
*Achieved goal blood pressure or patient making progress toward this goal. ACE = angiotensin-converting enzyme.

Figure 4. Treatment algorithm. Reprinted with permission from reference 33.

level of serum cholesterol in the U.S. population has declined, contributing to the reduction in cardiovascular disease mortality rates. The objective of diet therapy is to reduce body weight and blood lipids to a level sufficient to reduce coronary risk. However, even under controlled clinical trial conditions (27), commonly recommended diets alone cause only a modest average fall in total and LDL cholesterol, with few subjects showing reductions exceeding 10%. While more rigorous vegetarian diets may result in satisfactory lipid lowering, they may cause considerable disruption in many homes so that a more acceptable solution is achieved by addition of lipid-lowering drugs (28). Low fat diets also facilitate the action of lipid-lowering drugs, and may favorably affect other important nonlipid mechanisms. Moreover, diets low in animal fat and high in fruits and vegetables will often reduce obesity, particularly in older patients, and favorably influence blood pressure and triglyceride levels. Although several population studies indicate significant risk reduction associated with alcohol consumption, it would seem that initiation or encouragement of alcohol consumption has considerable risk for other adverse health and behavioral consequences (29). Users of alcohol,

in moderation (one to two drinks per day), should not be discouraged.

Serum lipids. In any patient who has sustained a coronary event, it should be suspected that the preexisting specific lipid levels are excessive (or deficient for HDL) for that patient and must be altered (Fig. 3) (30,31). Multiple studies with angiographic end points have shown the early and dramatic benefit of reduction of LDL cholesterol to under 100 mg/dl (21). Hence, virtually all coronary patients are candidates for lipid reduction. If dietary modification is insufficient, several drug interventions to lower lipids are available. In each, it is essential for the management team to define the specific action of the drug and then follow the resulting change by charting. The HMG-CoA reductase inhibitor class of drugs are effective in lowering total and LDL cholesterol by approximately 30% and cause a modest increase in HDL cholesterol, and appear, currently, to be the drugs of choice (32). The results of the Scandinavian Simvastatin Survival Study (4S) trial could be generalized to male and female younger and older patients and those with high and low initial lipid levels. As a class, statins have not been associated with a high rate of serious adverse



symptoms, although there is a low incidence of hepatic dysfunction and myocytolysis, particularly in the elderly. The resins colestipol and cholestyramine and the fibric acid derivative gemfibrozil are effective in reducing coronary event rates, but they do not induce as great an LDL lowering, and their unpleasant characteristics impair compliance. Nicotinic acid facilitates the cholesterol lowering achieved by diet and raises HDL, but also has unpleasant side effects. Probuco has been shown to not affect outcome, may prolong the QT interval and should be used in patients with low HDL values. Those pharmacologic interventions that are associated with dose-related adverse symptoms are more likely to be met with noncompliance in a practice setting than in the conditions of controlled clinical trials.

Blood pressure. The Joint National Committee (JNC-V) report on hypertension recommends that cuff blood pressure values, which correctly obtained and exceeding 140 mm Hg systolic and 90 mm Hg diastolic (rest), deserve attention, in particular in the patient with coronary disease (33). A treatment algorithm and a practical guide are presented in Figures 4 and 5. A comprehensive team approach, with regular follow-up, offers the ideal circumstance for ensuring compliance in effective drug therapy.

Aspirin. Aspirin in low dose has proven benefit in all-cause mortality and in coronary event incidence (25). It is effective, inexpensive and should be part of the management of every patient at coronary risk, other than those with specific contraindications. The role of warfarin sodium (Coumadin), heparin and newer antithrombotic and anticoagulants remains to be defined.

Activity. Although sedentary life-style appears to have an adverse effect in all persons, the long-term benefit of formal

Figure 5. Guide to blood pressure (BP) management. Total cardiovascular risk should be assessed first and the major components of risk identified. If 10-year coronary heart disease risk is $>20\%$ or will be $>20\%$ if projected to age 60, more intensive advice for all risk factors will be required. Clinical vascular disease will increase the risk to no more than 20% for most patients and to $>40\%$ for many. DBP = diastolic blood pressure; SBP = systolic blood pressure. Reprinted with permission.

programs of exercise for the patient with coronary disease is not yet clear (34,35). Age, general conditioning, individual preference and motivation must play a part. Modest levels of physical activity offer benefit for weight reduction, and increase the sense of well-being.

Stress. Behavior modification with regard to time management, urgency and hostility has demonstrated benefit (see Task Force 3). Further, counseling by the cardiovascular specialist may be appropriate early after myocardial infarction as well as before and after revascularization. The patient must be assisted in dealing with the consequences of his/of her disease (36).

Estrogen and progesterone. The temporal "advantage" enjoyed by women in regard to coronary disease is mediated in significant part by these hormones. In general, older women and those at above-average risk for coronary disease in particular, will benefit from hormone therapy in a variety of ways (37). Definitive clinical data is required to define the benefit-risk relationship of postmenopausal estrogen administration in women with coronary disease. Those with a family history of breast cancer and at a younger age should not be encouraged to receive hormone therapy.

Other drugs. In patients with an acute myocardial infarction, beta-blocking drugs reduce late mortality and reinfarction (25). Angiotensin-converting enzyme inhibitors decrease mortality in patients with left ventricular dysfunction and also decrease reinfarction rate. Use of these classes of drugs is a necessary component of optimal medical treatment of coronary artery disease.

Summary and Recommendations

Current medical management of atherosclerotic coronary artery disease often fails to reflect advances in current knowledge of risk factors and their effective modification. This failure results in an avoidable occurrence of death and disability and must be remedied. A strategy for optimal medical management of coronary disease requires lifetime attention and a cohesive team approach.

The general principles of evaluation and management presented herein must be modified for each patient according to cardiac status, other medical conditions and specific cardiac risk characteristics. This mandates a standard of physician competence based on a knowledge and understanding of the underlying disturbances of vascular, metabolic and coagulative function, in addition to the conventional cardiologic competencies. In each case, the strategy must include careful and continued documentation of the individual characteristics of the patient. Medical interventions must be individualized to limit risk, and to develop milestones to achieve these goals. If these goals are not met, then the treatment plan should be suitably modified. The trained specialist fills an essential role as consultant, coordinator and team leader of cardiac care. Effective treatment in patients with coronary disease and other at-risk patients can make efficient the consumption of available human and material resources.

A healthy life-style in the general population, as well as in patients, is to be strongly encouraged. Designation of the term "prevention" as related to the problem of coronary heart disease is imprecise, inaccurate and confusing. The task at hand is the optimal medical treatment of atherosclerosis and should be so designated, as in the management of other chronic disease states.

References

1. Neaton JD, Wentworth D. Serum cholesterol, blood pressure, cigarette smoking, and death from coronary heart disease. Overall findings and differences by age for 316,099 white men. Multiple Risk Factor Intervention Trial Research Group. *Arch Intern Med* 1992;152:56-64.
2. Hulley SB, Walsh JM, Newman TB. Health policy on blood cholesterol. Time to change directions. *Circulation* 1992;86(3):1026-9.
3. Muldoon MF, Manuck SB, Matthews KA. Lowering cholesterol concentrations and mortality: a quantitative review of primary prevention trials. *BMJ* 1990;301(6747):309-14.
4. Stamler J, Stamler R, Brown WV. Serum cholesterol. Doing the right thing. *Circulation* 1993;88(4 Pt 1):1954-60.
5. Law MR, Wald NJ, Thompson SG. By how much and how quickly does reduction in serum cholesterol concentration lower risk of ischaemic heart disease? *BMJ* 1994;308:367-72.
6. Smith GD, Song F, Sheldon TA. Cholesterol lowering and mortality: the importance of considering initial level of risk. *BMJ* 1993;306(6889):1367-73.
7. Silberberg JS, Henry DA. The benefits of reducing cholesterol levels: the need to distinguish primary from secondary prevention. 2. Implications for heart disease prevention in Australia. *Med J Aust* 1991;155(10):665-74.
8. Swan HJ. Why cardiologists must be interested in lipids. *Am J Cardiol* 1995;75(15):1067-8.
9. Fuster V, Badimon L, Badimon JJ, Chesebro JH. The pathogenesis of coronary artery disease and the acute coronary syndromes. *N Engl J Med* 1992;326(5):310-8.
10. Falk E, Shah PK, Fuster V. Coronary plaque disruption. *Circulation* 1995;92:657-71.
11. Falk E. Plaque rupture with severe pre-existing stenosis precipitating coronary thrombosis. Characteristics of coronary atherosclerotic plaques underlying fatal occlusive thrombi. *Br Heart J* 1983;50:127-34.
12. Meade TW, Ruddock V, Stirling Y, Chakrabarti R, Miller GJ. Fibrinolytic activity, clotting factors, and long-term incidence of ischaemic heart disease in the Northwick Park Heart Study. *Lancet* 1993;342:1076-9.
13. Muller JE, Abela GS, Nesto RW, Tofler GH. Triggers, acute risk factors and vulnerable plaques: the lexicon of a new frontier. *J Am Coll Cardiol* 1994;23:809-13.
14. Roberts WC. Getting cardiologists interested in lipids. *Am J Cardiol* 1993;72:744-5.
15. Stevenson JC, Godsland IF, Wynn V. Cardiologists rebuked. *Lancet* 1994;344:1557.
16. Krumholz HM, Seeman TE, Merrill SS, et al. Lack of association between cholesterol and coronary heart disease mortality and morbidity and all-cause mortality in persons older than 70 years. *JAMA* 1994;272:1335-40.
17. Brown G, Albers JJ, Fisher LD, et al. Regression of coronary artery disease as a result of intensive lipid-lowering therapy in men with high levels of apolipoprotein B. *N Engl J Med* 1990;323:1289-98.
18. Gotto AM Jr. Lipid lowering, regression, and coronary events. A review of the Interdisciplinary Council on Lipids and Cardiovascular Risk Intervention, Seventh Council Meeting. *Circulation* 1995;92:646-56.
19. Leung WH, Lau CP, Wong CK. Beneficial effect of cholesterol-lowering therapy on coronary endothelium-dependent relaxation in hypercholesterolaemic patients. *Lancet* 1993;341:1496-500.
20. Rossouw JE, Lewis B, Rifkind BM. The value of lowering cholesterol after myocardial infarction. *N Engl J Med* 1990;323:1112-9.
21. Superko HR, Krauss RM. Coronary artery disease regression. Convincing evidence for the benefit of aggressive lipoprotein management. *Circulation* 1994;90:1056-69.
22. Sullivan JM, Frohlich ED, Lewis RP, Pasternak RC. Guidelines for training in adult cardiovascular medicine. Core Cardiology Training Symposium (COCATS). Task Force 10: training in preventive cardiovascular medicine. *J Am Coll Cardiol* 1995;25:33-4.
23. Levy D, Wilson PW, Anderson KM, Castelli WP. Stratifying the patient at risk from coronary disease: new insights from the Framingham Heart Study. *Am Heart J* 1990;119:712-7.
24. Smith SC, Blair SN, Criqui MH, et al. Preventing heart attack and death in patients with coronary disease. *Circulation* 1995;92:2-4.
25. Pearson T, Rapaport E, Criqui M, et al. Optimal risk factor management in the patient after coronary revascularization. A statement for healthcare professionals from an American Heart Association Writing Group. *Circulation* 1994;90(6):3125-33.
26. Jonas MA, Oates JA, Ockene JK, Hennekens CH. Statement on smoking and cardiovascular disease for health care professionals. American Heart Association. *Circulation* 1992;86:1664-9.
27. Hunninghake DB, Stein EA, Dujovne CA, et al. The efficacy of intensive dietary therapy alone or combined with lovastatin in outpatients with hypercholesterolemia. *N Engl J Med* 1993;328(17):1213-9.
28. Roberts WC. The ineffectiveness of a commonly recommended lipid-lowering diet in significantly lowering the serum total and low-density lipoprotein cholesterol levels. *Am J Cardiol* 1994;73(8):623-4.
29. Pearson TA, Terry P. What to advise patients about drinking alcohol. The clinician's conundrum. *JAMA* 1994;272:967-8.
30. Summary of the second report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II). *JAMA* 1993;269(23):3015-23.
31. Pyorala K, De Backer G, Graham I, Poole-Wilson P, Wood D. Prevention of

- coronary heart disease in clinical practice. Recommendations of the Task Force of the European Society of Cardiology, European Atherosclerosis Society and European Society of Hypertension. *Eur Heart J* 1994;15:1300-31.
32. Randomized trial of cholesterol lowering in 4444 patients with coronary heart disease: the Scandinavian Simvastatin Survival Study (4S). *Lancet* 1994;344(8934):1383-9.
 33. The fifth report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNC V). *Arch Intern Med* 1993; 153:154-83.
 34. Curfman GD. Is exercise beneficial—or hazardous—to your heart? *N Engl J Med* 1993;329:1730-1.
 35. Schuler G, Hambrecht R, Schlierf G, et al. Regular physical exercise and low-fat diet. Effects on progression of coronary artery disease. *Circulation* 1992;86:1-11.
 36. Ornish D, Brown SE, Scherwitz LW, et al. Can lifestyle changes reverse coronary heart disease? The Lifestyle Heart Trial. *Lancet* 1990;336(8708): 129-33.
 37. Barrett-Connor E, Bush TL. Estrogen and coronary heart disease in women. *JAMA* 1991;265:1861-7.

Task Force 8. Organization of Preventive Cardiology Service

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The expanding knowledge base regarding the pathophysiology, molecular biology, epidemiology and economic aspects of atherosclerotic cardiovascular disease provides a solid foundation for the development of guidelines for risk factor management. Randomized, controlled clinical trials have demonstrated the efficacy of a variety of interventions in the secondary prevention of coronary disease and in the primary prevention of disease in high risk individuals. The evidence and support of these interventions was felt sufficient to provide a consensus statement, endorsed by the American Heart Association and the American College of Cardiology, for the secondary prevention of coronary artery disease (1).

However, the development of a scientific rationale through basic and clinical studies often fails to influence clinical practice. Convincing results of randomized clinical trials and widely disseminated guidelines often fall short of implementation (2-7).

The existence of barriers to the implementation of risk reduction interventions appears obvious. Preventive services, including counseling, are provided less often than experts recommend and less frequently than patients and their physicians prefer (8). In a primary care setting, 75% of patients who smoke say they would attempt to stop smoking if their physician advised them to do so, yet only 40% to 55% report that their physician provided such advice to them (9,10). Only 45% to 65% of patients with hypercholesterolemia had evidence of treatment (3,11,12). The extent to which interventions recommended by the American College of Cardiology (1) are being carried out is poorly described. Table 1 shows estimates of the levels of risk factor management. These estimates deal only with initiation of *any* risk factor management; levels of adequacy of risk factor control could likely be much worse. Barriers that prevent these efficacious and cost-effective inter-

ventions from being deployed could be targetted as a way to correct deficiencies in levels of risk factor management. The objective of this task force report is to identify barriers within health care organizations which impede the provision of preventive services. Health care organizations under consideration range from primary care and cardiovascular specialty practices to hospitals, managed care organizations and third-party payors. Opportunities and strategies for these organizations are then identified and evaluated as to their ability to effectively, feasibly and appropriately influence the provision of preventive cardiovascular services. Finally, a list of recommendations for organizations has been developed, based on the recognized needs of patients and providers and the published evidence supportive of the efficacy of specific strategies.

Barriers to Implementation of Preventive Services: An Overview

A variety of barriers to the successful implementation of preventive services have been identified (Table 2). These include factors at the patient, provider, health care organization and community/societal levels (11,12). These different types of barriers might be considered sequential, in that any one barrier in the chain could result in a lack of provision of preventive service.

Patient factors. A detailed discussion of patient factors is beyond the scope of this task force report. Physicians frequently perceive patients as not motivated or noncompliant, yet patients consistently report preventive services as a high priority for their health care and want physicians to provide life-style and prevention recommendations. Ironically, patients cite physicians's failure to order tests, give information or communicate results as reasons not to request preventive services