

## COMPETENCE AND TRAINING STATEMENT

# ACCF/AHA/ACP 2009 Competence and Training Statement: A Curriculum on Prevention of Cardiovascular Disease

A Report of the American College of Cardiology Foundation/American Heart Association/  
American College of Physicians Task Force on Competence and Training (Writing Committee to  
Develop a Competence and Training Statement on Prevention of Cardiovascular Disease)

*Developed in Collaboration With the American Academy of Neurology; American Association of  
Cardiovascular and Pulmonary Rehabilitation; American College of Preventive Medicine; American Diabetes  
Association; American Society of Hypertension; Association of Black Cardiologists; National Lipid Association;  
and Preventive Cardiovascular Nurses Association*

### Writing Committee Members

C. Noel Bairey Merz, MD, FACC, FAHA, *Chair*\*

Mark J. Alberts, MD, FAHA†

Gary J. Balady, MD, FACC‡

Christie M. Ballantyne, MD, FACC\*

Kathy Berra, MSN, ANP, FAHA, FAAN§

Henry R. Black, MD||

Roger S. Blumenthal, MD, FACC, FAHA\*

Michael H. Davidson, MD, FACC¶

Sara B. Fazio, MD#

Keith C. Ferdinand, MD, FACC, FAHA\*\*

Lawrence J. Fine, MD, DRPH††\*\*\*

Vivian Fonseca, MD‡‡

Barry A. Franklin, PhD, FAHA§§

Patrick E. McBride, MD, MPH, FACC, FAHA\*

George A. Mensah, MD, FACC, FACP,

FAHA|||\*\*\*

Geno J. Merli, MD, FACP#

Patrick T. O'Gara, MD, FACC, FAHA§§

Paul D. Thompson, MD, FACC¶¶

James A. Underberg, MD, MS, FACPM, FACP##

\*American College of Cardiology Foundation Representative; †American Academy of Neurology Representative; ‡American Association of Cardiovascular and Pulmonary Rehabilitation Representative; §Preventive Cardiovascular Nurses Association Representative; ||American Society of Hypertension Representative; ¶National Lipid Association Representative; #American College of Physicians Representative; \*\*Association of Black Cardiologists Representative; ††National Heart, Lung, and Blood Institute Representative; ‡‡American Diabetes Association Representative; §§American Heart Association Representative; |||Centers for Disease Control and Prevention; ¶¶American College of Sports Medicine Representative; ##American College of Preventive Medicine Representative; \*\*\*The findings and conclusions in this competence and training statement reflect ACCF policy and do not necessarily represent the views of the Centers for Disease Control and Prevention or the National Institutes of Health, by whom Drs. Fine and Mensah are employed.

This document was approved by the American College of Cardiology Foundation Board of Trustees in October 2008, by the American Heart Association Science Advisory and Coordinating Committee in November 2008, and by the American College of Physicians Board of Regents in April 2009.

The American College of Cardiology Foundation requests that this document be cited as follows: Bairey Merz CN, Alberts MJ, Balady GJ, Ballantyne CM, Berra K, Black HR, Blumenthal RS, Davidson MH, Fazio SB, Ferdinand KC, Fine LJ, Fonseca V, Franklin BA, McBride PE, Mensah GA, Merli GJ, O'Gara PT, Thompson PD, Underberg JA. ACCF/AHA/ACP 2009 competence and training statement: a curriculum on prevention of cardiovascular disease: a report of the American College of Cardiology Foundation/American Heart Association/American College of Physicians Task Force on Competence

and Training (Writing Committee to Develop a Competence and Training Statement on Prevention of Cardiovascular Disease). *J Am Coll Cardiol* 2009;54:1336-63.

This article has been copublished in the September 29, 2009, issue of *Circulation*.

Copies: This document is available on the World Wide Web sites of the American College of Cardiology ([www.acc.org](http://www.acc.org)) and the American Heart Association ([my.heart.org](http://my.heart.org)). For copies of this document, please contact Elsevier Inc. Reprint Department, fax (212) 633-3820, e-mail [reprints@elsevier.com](mailto:reprints@elsevier.com).

Permissions: Modification, alteration, enhancement, and/or distribution of this document are not permitted without the express permission of the American College of Cardiology Foundation. Please contact Elsevier's permission department at [healthpermissions@elsevier.com](mailto:healthpermissions@elsevier.com).

**Task Force Members**

Jonathan L. Halperin, MD, FACC, *Chair*  
Mark A. Creager, MD, FACC, FAHA<sup>†††</sup>  
Gordon L. Fung, MD, PhD, FACC, FAHA  
David R. Holmes, Jr, MD, FACC<sup>‡‡‡</sup>  
Geno J. Merli, MD, FACP  
Ira S. Nash, MD, FACC, FACP

L. Kristin Newby, MD, FACC, FAHA  
Ileana Piña, MD, FACC, FAHA  
George P. Rodgers, MD, FACC, FAHA<sup>‡‡‡</sup>  
Cynthia M. Tracy, MD, FACC<sup>‡‡‡</sup>  
Howard H. Weitz, MD, FACC, FACP

<sup>†††</sup>Former Task Force chair during the writing effort; <sup>‡‡‡</sup>Former Task Force member during the writing effort.

**TABLE OF CONTENTS**

<b>Preamble</b> .....	1338	<b>9. Adherence and Disease Outcome Interdisciplinary Programs</b> .....	1346
<b>1. Introduction</b> .....	1338	<b>9.1. Justification</b> .....	1346
<b>2. Cardiovascular and Vascular Biology</b> .....	1339	<b>9.2. Minimal Knowledge</b> .....	1347
<b>2.1. Justification</b> .....	1339	<b>10. Nutrition Management</b> .....	1347
<b>2.2. Minimal Knowledge</b> .....	1339	<b>10.1. Justification</b> .....	1347
<b>3. Clinical Epidemiology and Biostatistics</b> .....	1339	<b>10.2. Minimal Knowledge</b> .....	1347
<b>3.1. Justification</b> .....	1339	<b>11. Lipid Management (Management of Dyslipidemia)</b> .....	1347
<b>3.2. Minimal Knowledge</b> .....	1343	<b>11.1. Justification</b> .....	1347
<b>4. Cardiovascular Pharmacology (Complex Multipharmacologic Understanding)</b> .....	1343	<b>11.2. Minimal Knowledge</b> .....	1348
<b>4.1. Justification</b> .....	1343	<b>12. Thrombosis Management</b> .....	1348
<b>4.2. Minimal Knowledge</b> .....	1343	<b>12.1. Justification</b> .....	1348
<b>5. Genetics and Cardiovascular Disease in Individuals and Families</b> .....	1344	<b>12.2. Minimal Knowledge</b> .....	1348
<b>5.1. Justification</b> .....	1344	<b>13. Hypertension Management</b> .....	1349
<b>5.2. Minimal Knowledge</b> .....	1344	<b>13.1. Justification</b> .....	1349
<b>6. Behavioral and Psychosocial Programs (Financial and Socioeconomic Factors)</b> .....	1344	<b>13.2. Minimal Knowledge</b> .....	1349
<b>6.1. Justification</b> .....	1344	<b>14. Smoking Cessation</b> .....	1349
<b>6.2. Minimal Knowledge</b> .....	1345	<b>14.1. Justification</b> .....	1349
<b>7. Advanced Risk Assessment (Renal, Inflammatory Diseases)</b> .....	1345	<b>14.2. Minimal Knowledge</b> .....	1349
<b>7.1. Justification</b> .....	1345	<b>15. Obesity Management (Behavioral Programs)</b> .....	1350
<b>7.2. Minimal Knowledge</b> .....	1345	<b>15.1. Justification</b> .....	1350
<b>8. Subclinical Atherosclerosis Assessment (Imaging and Nonimaging)</b> .....	1345	<b>15.2. Minimal Knowledge</b> .....	1350
<b>8.1. Justification</b> .....	1345	<b>16. Exercise Physiology, Physical Activity Management, and Cardiac Rehabilitation (Secondary Prevention)</b> .....	1351
<b>8.2. Minimal Knowledge</b> .....	1346	<b>16.1. Justification</b> .....	1351
		<b>16.2. Minimal Knowledge</b> .....	1351
		<b>17. Prediabetes, Metabolic Syndrome, Insulin Resistance, and Diabetes Management</b> .....	1352
		<b>17.1. Justification</b> .....	1352
		<b>17.2. Minimal Knowledge</b> .....	1352

<b>18. Chronic Disease Management</b> .....	1352
<b>18.1. Justification</b> .....	1352
<b>18.2. Minimal Knowledge</b> .....	1353
<b>References</b> .....	1353
<b>Appendix 1. Author Relationships With Industry and Other Entities</b> .....	1359
<b>Appendix 2. Reviewer Relationships With Industry and Other Entities</b> .....	1362

## Preamble

The American College of Cardiology Foundation (ACCF)/American Heart Association (AHA)/American College of Physicians (ACP) Task Force on Clinical Competence was formed in 1998 to develop recommendations for attaining and maintaining the cognitive and technical skills necessary for the competent performance of a specific cardiovascular service, procedure, or technology. These documents are evidence-based, and where evidence is not available, expert opinion is utilized to formulate recommendations. Indications and contraindications for specific services or procedures are not included in the scope of these documents. Recommendations are intended to guide curriculum development and assist those who judge the competence of cardiovascular healthcare providers entering practice for the first time and/or those in practice who undergo periodic review of their expertise or who apply for privileges at a new institution. The assessment of competence is complex and multidimensional; therefore, isolated recommendations contained herein may not necessarily be sufficient or appropriate for judging overall competence. The current document addresses a curriculum for developing competence in the prevention of cardiovascular disease (CVD) and is authored by representatives of the ACCF, AHA, ACP, the American Academy of Neurology; American Association of Cardiovascular and Pulmonary Rehabilitation; American College of Preventive Medicine; American College of Sports Medicine; American Diabetes Association (ADA); American Society of Hypertension; Association of Black Cardiologists; Centers for Disease Control and Prevention; National Heart, Lung, and Blood Institute; National Lipid Association; and the Preventive Cardiovascular Nurses Association. The recommendations contained herein recognize the broader context of clinical training and the importance of systems of care in improving patient outcomes. Trainees should be aware of and responsive to the larger context of systems-based health care and utilize all available resources to provide optimum care. Similarly, the development of competence embodies knowledgeable incorporation of technological advances for the evaluation of health and

disease based on ongoing familiarity with the emerging scientific and social literature.

The ACCF/AHA/ACP Task Force makes every effort to avoid actual or potential conflicts of interest that may arise as a result of an outside relationship or personal interest of a member of the ACCF/AHA/ACP Writing Committee. Specifically, all members of the writing committee are asked to provide disclosure statements of all such relationships that might be perceived as real or potential conflicts of interest relevant to the document topic. These statements are reviewed by the writing committee and updated as changes occur. The relationships with industry for authors and peer reviewers are published in [Appendixes 1 and 2](#) of the document.

*Jonathan L. Halperin, MD, FACC  
Chair, ACCF/AHA/ACP Task Force  
on Competence and Training*

## 1. Introduction

The mission of many organizations is providing optimal care to those with or at risk of developing CVD (primary and secondary prevention). Over the past 2 decades, there have been dramatic increases in knowledge concerning specific risk factors in atherosclerosis, hypertension, thrombosis, and other forms of vascular dysfunction. Clinical trials have proven that strategies aimed at the appropriate detection and modification of risk factors can slow progression of atherosclerosis, diabetes mellitus, and hypertension and reduce the occurrence of clinical cardiovascular events in both primary and secondary prevention settings. More recently, it has been shown that atherosclerosis can be stabilized or even modestly reversed. Finally, a new and growing knowledge base of molecular genetics applied to the study of the cardiovascular system has potential relevance to the clinical practice of preventive cardiovascular medicine.

Despite the fact that clinical outcomes can be improved by promotion of favorable life habits and behaviors and by the proper use of drug treatment, the application of primary and secondary preventive interventions in clinical practice is not optimal. Prevention of CVD in both the primary and secondary prevention setting, while dominantly the responsibility of the primary care provider, is increasingly challenged given the ever expanding new knowledge as well as the ongoing problems related to adherence to recommendations. New knowledge in the area of preclinical disease detection has presented increasingly challenging scenarios to primary care healthcare providers relative to the decisions regarding the need for further risk stratification and aggressive medical regimens. Furthermore, increasingly complex patients are surviving with CVD, many of whom can benefit from advanced knowledge and expertise with regard to risk factor management and rehabilitation that is beyond the

traditional general primary and cardiology practitioner's scope of practice.

The prevention of cardiovascular morbidity and mortality is a shared responsibility among all health professionals involved in the care of people at risk of developing CVD. This document is directed at those individuals seeking expertise at a leadership level in this field, and includes opportunities for formal training and alternative routes to competence and maintenance of competence in prevention of CVD (Table 1) (1–5), and educational resources for acquisition and maintenance of competence in the prevention of CVD (Table 2) (6–43). To address the expanding fund of knowledge in the area and to ensure that an adequately trained force of preventive cardiovascular leaders will be available to primary care providers, as well as to provide a pool of providers with expertise in running rehabilitation and other programs designed to address the ongoing issue of adherence, the formulation of clinical competency criteria for the cardiovascular preventive specialist is needed. These competency criteria are expected to address issues of expert clinical and scientific leadership, specialty patient care and consultation, and directorship of primary and secondary preventive cardiac programs. Of note and similar to other subspecialty areas of medicine, cardiovascular preventive specialists will have varying areas of expertise and will not necessarily achieve all of the outlined areas of competencies. These clinical competency criteria in the area of specialty treatment and prevention of CVD are needed given the current setting of a rapidly growing field of knowledge ranging from molecular and cellular mechanisms to clinical outcomes in order to translate this into improved patient care.

*C. Noel Bairey Merz, MD, FACC, FAHA  
Chair, ACCF/AHA/ACP Writing Committee to  
Develop a Competence and Training Statement  
on Prevention of Cardiovascular Disease*

## 2. Cardiovascular and Vascular Biology

### 2.1. Justification

Recent advances in cardiac and vascular biology and related molecular and cellular mechanisms provide a sound scientific foundation for the practice of preventive cardiovascular medicine. A basic knowledge of the structure and function of the arterial wall, its interactions with components of the circulating blood, and key pathologic processes such as oxidation, inflammation, thrombosis, and remodeling is important to the application of strategies for the detection, evaluation, and prevention of atherosclerotic CVD (44,45). Similarly, a basic understanding of myocardial cellular and molecular processes is essential for effective application of therapies that address myocardial salvage, regeneration, and remodeling.

### 2.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. The process of atherosclerosis that begins in youth, initially as a fatty streak containing mainly lipid-rich macrophages in the arterial intima (46), and the role of various blood lipoproteins in this process and the factors that promote the initiation and progression of the fatty streak to arterial plaque (including endothelial activation and dysfunction, smooth muscle cell migration and proliferation, collagen production, and arterial remodeling).
2. Disorders of lipid metabolism and major atherogenic risk factors, and the pathophysiological significance of the biological composition of the arterial plaque and its fibrous cap (47,48).
3. A substantial understanding of vulnerable plaque and the crucial role of inflammation, plaque fissuring, erosion, and rupture in the genesis of acute coronary syndromes, should be emphasized. An understanding of the concepts of plaque pathophysiology remodeling and progression should also be understood, as well as an appreciation of the systemic nature of atherosclerosis.
4. Systemic (endocrine) and local (autocrine/paracrine) neurohormonal derangements that lead to an impaired vasoregulatory and fibrinolytic balance, including the biological, social, and environmental determinants of these derangements as well as the pharmacologic and therapeutic lifestyle changes established for their control.
5. Mechanisms of atherosclerosis-specific targeted interventions with the use of combination medications that can be used to slow progression and reverse the process (49).
6. Vascular and hemodynamic benefits of smoking cessation, increased physical activity, and a diet low in saturated fats and rich in fruits, vegetables, fiber, and whole grains, particularly promoted at an early age.

## 3. Clinical Epidemiology and Biostatistics

### 3.1. Justification

Clinical epidemiology is the study of the magnitude, distribution, and trends in the factors that affect health, disease, and their determinants in populations. Within the context of preventive cardiology, clinical epidemiology provides crucial information in the enumeration of CVD events, rates, trends, and outcomes in defined populations and their subgroups. It also permits the identification of populations at different levels of risk for CVD events and the existence of health disparities (51). The surveillance components of clinical epidemiology provide clues to new and emerging CVD threats and permit assessment of the effectiveness of interventions.

Recent emphasis on quality, economic end points, and modeling in epidemiologic studies provides an opportunity for epidemiology to inform clinical practice on the cost-effectiveness and health impact of alternative preventive strategies (52–55). In addition, clinical epidemiology serves an important role in informing practitioners about the use of evidence from clinical trials and the strength and general-

**Table 1. Opportunities for Formal Training and Alternative Routes to Competence and Maintenance of Competence in Prevention of Cardiovascular Disease**

Section	A Ways to Achieve Formal Training	B Alternate Routes to Achieve Competence	C Maintenance of Competence
2. Cardiovascular and Vascular Biology		ACCF Self-Assessment programs (ACCSAP, LipidSAP)	CME that focuses on cardiovascular and vascular biology and atherosclerosis is important to receive each year
3. Clinical Epidemiology and Biostatistics			ACCSAP The AHA 10-Day Seminar on the Epidemiology and Prevention of CVD
5. Genetics and Cardiovascular Disease in Individuals and Families		Participation in an active genetic CVD referral clinic under the supervision of expert cardiovascular specialists in the relevant areas	CME that focuses on the genetic aspects of CVD prevention in individuals and families is important to receive each year
6. Behavioral and Psychosocial Programs (Financial and Socioeconomic Factors)		Participation in cardiac rehabilitation program that includes psychosocial assessment, management, and referral under the supervision of expert cardiovascular and other specialists in the relevant areas	CME that focuses on behavioral assessment and management of patients with CVD
10. Nutrition Management	Clinical experience in a preventive cardiology clinic program during formal fellowship training. Clinical experience in nutrition subspecialty programs such as weight loss clinics, lipid clinics, and diabetes management programs		
11. Lipid Management (Management of Dyslipidemia)	A comprehensive understanding of the NCEP ATP III and updates is critical to achieve competence. The ACCF provides a self-assessment program in lipidology, and the National Lipid Association has a self-study program that can provide eligibility for Board certification by the American Board of Clinical Lipidology		National Lipid Association sponsored self-assessment program, self-study modules, masters class, and advanced masters summits in lipidology
12. Thrombosis Management	ACCF/AHA continuing education programs, the American College of Chest Physician Consensus Conference Guidelines, the Peripheral Arterial Diseases Antiplatelet Consensus Group: Antithrombotic and thrombolytic therapy: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (1)	Web sites of the <a href="#">PAD Coalition</a> and <a href="#">AHA</a>	National society meetings with focus on antithrombotic therapy
13. Hypertension Management		<a href="#">American Society of Hypertension Certification</a> as a clinical specialist in hypertension and medical education seminars and didactic sessions with faculty members with expertise in each of the above areas	
14. Smoking Cessation	Formal training in behavioral science and smoking cessation is a critical link to successful office-based or hospital-based smoking cessation programs. Mentoring by faculty/colleagues with expertise in behavioral medicine/science is an important training modality	Medical education seminars, Webcast CME programs, Society for Behavioral Medicine publications and meetings, and CME programs specifically addressing addiction and smoking cessation	<a href="#">National Cancer Institute Web site: Prevention and Cessation of Cigarette Smoking: Control of Tobacco Use (2)</a> <a href="#">MedlinePlus: Quitting Smoking (3)</a> <a href="#">AHA Web site: Smoking and Cardiovascular Disease (4)</a> <a href="#">CDC Web site: Smoking &amp; Tobacco Use</a>

Continued on next page

**Table 1. Continued**

Section	A Ways to Achieve Formal Training	B Alternate Routes to Achieve Competence	C Maintenance of Competence
15. Obesity Management (Behavioral Programs)	A clinical rotation in both an endocrinology and a bariatric surgery-based obesity clinic to learn how to apply patient-specific behavioral methods to achieve weight loss should be mandatory, with a suggested involvement in the care of 5 patients entering a weight loss management program over the course of formal training. Attendance at an accredited obesity training program for healthcare professionals.		Continued clinical practice as well as yearly CME courses in preventive cardiology with a focus on overweight/obesity management
16. Exercise Physiology, Physical Activity Management, and Cardiac Rehabilitation (Secondary Prevention)	Active instruction in a preventive cardiology/cardiac rehabilitation center that includes access to a multidisciplinary staff (e.g., cardiologists with specific expertise/training in secondary prevention, nurse clinicians, exercise physiologists, registered dietitians, behaviorists, smoking cessation counselors, and pharmacists). A listing of cardiopulmonary rehabilitation programs in the United States and Canada is available through the <a href="#">American Association of Cardiovascular and Pulmonary Rehabilitation</a> . Moreover, the <a href="#">American College of Sports Medicine</a> offers certification examinations and registry programs for exercise physiology, as well as a complete listing of the knowledge, skills, and abilities that comprise the foundations of these relevant certifications (e.g., exercise specialist, registered clinical exercise physiologist), with specific reference to requirements (e.g., educational degree, minimum hours of practical experience) and recommended competencies (5).	Direct clinical training with exercise physiologists, smoking cessation counselors, registered dietitians, and lipid specialists in settings other than formal cardiac rehabilitation programs	Active involvement with cardiac rehabilitation/secondary prevention programs and direct involvement in the supervision and care of cardiac rehabilitation patients each year. CME that focuses on clinical exercise physiology applications, exercise prescription in health and disease, and cardiac rehabilitation/secondary prevention are important to receive each year. Organizations with related regional and national conference programming include: ACCF; AHA; American College of Sports Medicine; and the American Association of Cardiovascular and Pulmonary Rehabilitation
17. Prediabetes, Metabolic Syndrome, Insulin Resistance, and Diabetes Management	Clinical experience in a preventive cardiovascular medicine clinic program during formal fellowship training, as well as rotations in a specialized diabetes clinic. ADA training modules with multiple choice questions and detailed, evidence-based answers.		ADA clinical practice guidelines are updated annually and published as a supplement to <i>Diabetes Care</i>
18. Chronic Disease Management	Clinical rotation in a preventive cardiology center to learn about and apply a patient-specific, systems approach to prevention of CVD in primary and secondary prevention patients		

**A** indicates use of ACCF/AHA training modules with multiple choice questions and detailed, evidence-based answers during training with mentoring by faculty members with expertise in each of the specified areas; **B** indicates medical education seminars and didactic sessions with faculty members with expertise in each of the above areas; and **C** indicates CME activities that focus in each of the specified areas. Use American Board of Internal Medicine recertification modules in the relevant area. ACCF indicates American College of Cardiology Foundation; ADA, American Diabetes Association; AHA, American Heart Association; CDC, Centers for Disease Control and Prevention; CME, continuing medical education; CVD, cardiovascular disease; and NCEP ATP, National Cholesterol Education Program Adult Treatment Panel III.

**Table 2. Educational Resources for Acquisition and Maintenance of Competence in the Prevention of Cardiovascular Disease**

Section	Educational Opportunities
2. Cardiovascular and Vascular Biology	(6)
3. Clinical Epidemiology and Biostatistics	(7–10)
4. Cardiovascular Pharmacology (Complex Multipharmacologic Understanding)	Micromedex is available in many hospitals for dosing and interactions information Facts and Comparisons page is useful to check for drug–drug interactions
5. Genetics and Cardiovascular Disease in Individuals and Families	(6,11) National Coalition for Health Professionals Education in Genomics—competencies in genomics CDC genetics and genomics competencies for public health Inventory of family history tools and resources
6. Behavioral and Psychosocial Programs (Financial and Socioeconomic Factors)	(12–16)
7. Advanced Risk Assessment (Renal, Inflammatory Diseases)	(17,18)
8. Subclinical Atherosclerosis Assessment (Imaging and Nonimaging)	(17–20)
9. Adherence and Disease Outcome Interdisciplinary Programs	Patient compliance information for the professional (21–24)
10. Nutrition Management	(25–27)
11. Lipid Management (Management of Dyslipidemia)	American Board of Clinical Lipidology National Lipid Association Self-Assessment Program (28)
12. Thrombosis Management	See PAD Coalition and AHA for resources (9,29–32)
13. Hypertension Management	(33–35) Council for High Blood Pressure Research and the Councils on Clinical Cardiology and Epidemiology and Prevention
14. Smoking Cessation	(2,36–38) Links to MEDLINE and access to multiple governmental and professional Web sites for smoking cessation support and literature AHA Web site: Smoking and Cardiovascular Disease: includes educational resources for smoking cessation
15. Obesity Management (Behavioral Programs)	(39–41)
16. Exercise Physiology, Physical Activity Management, and Cardiac Rehabilitation (Secondary Prevention)	American College of Sports Medicine Annual Meetings and Regional Conferences American Association of Cardiovascular and Pulmonary Rehabilitation Annual Meeting and resource materials AHA Annual Scientific Sessions AHA Guidelines and Statements on Exercise
17. Prediabetes, Metabolic Syndrome, Insulin Resistance, and Diabetes Management	American Diabetes Association (ADA) clinical practice guidelines ADA Professional Practice Resources (42) Diabetes (Personal Health Decisions) PHD risk assessment tool ADA for healthcare professionals American Association of Clinical Endocrinologists
18. Chronic Disease Management	(43)

AHA indicates American Heart Association.

izability of that evidence. In this endeavor, the related field of biostatistics provides important principles for appropriate design of clinical trials, interpretation of trial results, and the effective use of screening, diagnostic, and prognostic tools in the practice of preventive cardiology (56).

### 3.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. Terms used to describe the central tendency of population distributions (e.g., mean, median, and mode), and the terms used to describe the magnitude of dispersion around these measures (e.g., standard deviation, standard error, and percentiles) (51–56). Familiarity with terms that describe the frequency and burden of CVD as well as the importance of age adjustment.
2. Terms used to characterize screening and diagnostic tests including sensitivity, specificity, accuracy, and predictive values (positive and negative) (56,57).
3. Experimental study designs (randomized, nonrandomized, and noninferiority clinical trials) and nonexperimental designs (cohort, case-control, nested case-control, cross-sectional studies), as well as the principle of hypothesis testing that underlies these studies, and the number needed to treat and the number needed to harm.
4. Common analyses encountered in the medical literature such as the *t* test, chi-square test, multiple regression, Kaplan-Meier survival analysis, and the Cox proportional hazards analysis is necessary, including the types of errors that can be committed when inferences are made about data in studies.
5. Traditional risk factors (8–10) and nontraditional risk factors, such as calculation of non-high-density lipoprotein cholesterol (non-HDL-C) in persons with triglyceride levels above 200 mg/dL.
6. Inflammatory biomarkers, including high-sensitivity C-reactive protein, serum amyloid A, interleukin-6, lipoprotein-associated phospholipase A2, monocyte chemoattractant protein-1, soluble CD40 ligand, and myeloperoxidase and their possible utility in risk assessment (58,59).
7. The concepts of relative and absolute risk; short-term, long-term, and lifetime risk; and the population burden of CVD attributable to specific risk factors, including the Framingham Risk Assessment score in clinical practice and knowledge of its limitations (60).
8. Cost-benefit analyses of CVD interventions.

## 4. Cardiovascular Pharmacology (Complex Multipharmacologic Understanding)

### 4.1. Justification

Knowledge of cardiovascular pharmacology and the basic principles of pharmacokinetics, pharmacodynamics, and pharmacogenomics is critical to the targeted application of

drug therapy for individual patients. A basic knowledge of drug interactions, anticipated side effects, and dosing regimens in a heterogeneous mix of complex patients is necessary to integrate new research and new approaches for CVD prevention and treatment. The challenges posed by age, gender, reproductive hormones, and medical comorbidities, including the coexistence of disorders known to contribute to cardiac and vascular endothelial dysfunction, must be recognized. Interactions between medication and nutrition (e.g., grapefruit), over-the-counter supplements (e.g., antioxidant vitamins), nutraceuticals (e.g., stanol/sterol esters), and dietary alcohol intake are increasingly reported and of practical relevance. Knowledge that pharmacologic therapies may have differing impact based on underlying existence of disease and endogenous hormone status, including hormonal therapies, is important. A basic understanding of pharmacology will also be important as new therapies for myocardial salvage, regeneration, and remodeling become available.

### 4.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. Pharmacological approaches to lipids, hypertension, thrombosis, diabetes and insulin resistance, cigarette smoking cessation, and obesity (10). The important role of statins and other lipid-lowering medications, antiplatelet therapies, renin-angiotensin-aldosterone system agents, and antihypertensive medications should be considered (49).
2. Use of multiple drug combinations (coexistent conditions and risk factor clustering) (61) and drug-drug interactions (62), including the ever-increasing complexity of pharmacological regimens and potential and realization of drug-drug interactions.
3. Preventive cardiovascular strategies for comorbidities such as renal disease, autoimmune inflammatory disorders, diabetes mellitus, and cancer, which raise the risk for CVD due to the comorbidity itself as well as the treatment regimens used to treat these comorbidities.
4. Pharmacologic dosing adjustment in consideration of issues of aging (63), gender (64), ethnicity, and comorbidities, for example, renal disease and liver disease and ethnicity. Aggressive preventive cardiovascular regimens are optimally tolerated when body weight- and renal function-adjusted, including the elderly, women, and smaller-sized men. Knowledge of ethnic groups that have higher rates of toxicity to certain medications is also important.
5. Pharmacological interactions with over-the-counter supplements, nutraceuticals (soluble fiber, psyllium seed, stanol/sterol esters), and common dietary ingredients, such as grapefruit, which can interact with many common medications, including most statins, increasing blood levels of the medication when taken concurrently



- (65). A majority of patients are taking over-the-counter supplements that may interact with their medication, such as the antioxidant vitamins that adversely interact to reduce the antiatherosclerotic niacin benefit.
6. Pharmacogenomics, including the prospective role of patient testing for genetic polymorphisms that raise/lower the likelihood of adverse pharmacological side effects, or lack of metabolism/efficacy with a particular class of medications.

## 5. Genetics and Cardiovascular Disease in Individuals and Families

---

### 5.1. Justification

Genes and gene–environment interactions play important roles in the causation, pathogenesis, and prognosis of CVD (6,66). Thus, knowledge of the spectrum of inherited susceptibilities to CVD and elucidation of the patterns of inheritance for specific genetic abnormalities may provide improvements in early detection, risk stratification, and prevention of CVD in individual patients and their family members (6,66–68). Additionally, advances in pharmacogenomics provide an opportunity for improving disease treatment and response (69,70).

A wide spectrum of CVD with inherited genetic susceptibilities is now known, and the advances made over the last 25 years in understanding the genetic basis of these disorders provide a rationale for ensuring competence in genetics for experts in the prevention of CVD (71–73). The limitations of current genetic information in patient care and the gaps between knowledge of an apparently inherited susceptibility and the availability of, or access to, corresponding effective treatments must be explicitly acknowledged (74). Finally, the benefits, risks, and costs associated with knowledge of a patient's genetic susceptibility to CVD and the ethical implications of referral for genetic testing and counseling must be recognized (75,76).

### 5.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. Basic skills in eliciting a comprehensive family history during the patient encounter and familiarity with clinical tools and/or questionnaires for collecting genetic information.
2. Differences between genotype and phenotype and the concepts of dominance, recessiveness, X-linked inheritance, genetic heterogeneity, and penetrance.
3. Basic principles of clinical genetics including the types of family studies, linkage analyses, genetic association studies, and familiarity with recent advances in genome-wide association.
4. Chromosomal deletions, duplications, and rearrangements as a cause of clinical syndromes associated with genetic disorders.

5. Mendelian disorders and syndromes associated with congenital heart disease (e.g., DiGeorge, Noonan, and Williams syndromes) and those involving connective tissue (e.g., Marfan syndrome, Ehlers-Danlos syndrome).
6. Genetic basis of specific cardiovascular disorders such as cardiomyopathies, arrhythmias, and lipoprotein disorders and their potential role in diagnostic evaluation and treatment.
7. Teratogens including warfarin, hydantoin, retinoic acid, valproic acid, rubella, and alcohol.
8. The indications for, as well as benefits, risks, and ethical implications of, referral for genetic testing and counseling, and the limitations of available testing kits.

## 6. Behavioral and Psychosocial Programs (Financial and Socioeconomic Factors)

---

### 6.1. Justification

Psychosocial factors add a significant dimension to CVD development and outcomes (13,14,77–80). Psychosocial factors influence the pathophysiology of disease, access to healthcare services, and adherence to treatment (81,82). The best treatments are of no use to a patient if he or she cannot access the healthcare system, has inadequate services, or obtains health care too late to change the outcome (15,16,83–85).

Prospective cohort studies provide evidence for a role for depression, stress, psychosocial work characteristics, social isolation and support, and possibly hostility as factors in the etiology of CVD and prognosis after CVD diagnosis (13,14,77–80). Over 30% of all patients with diagnosed cardiovascular or cerebrovascular disease have either clinical depression, anxiety, or other psychologically adverse conditions (13,14). Depression is common overall, and risk is increased following a CVD event (14,78,80). Depression is a risk factor for coronary heart disease (CHD), recurrent CHD events, and heart failure (HF), and is associated with poor outcomes in CVD, postcoronary bypass, and HF. Socioeconomic factors such as education, occupation, income, and insurance status have a significant impact on risk factor development, CVD, and mortality (77,86–90).

All physicians and other healthcare providers should be able to diagnose anxiety and depression, and this should be routine after a CVD event or stroke (14). While psychological and medical interventions to treat depression and anxiety have not been shown to reduce future cardiac events to date, further research is underway to determine if outcomes after CVD events benefit from treatment (14,16,83,84,91). Cardiac rehabilitation programs that incorporate psychosocial screening and intervention can improve treatment outcomes, the quality of life, and adherence of patients with psychological disorders (15,92).

## 6.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. Psychosocial factors in the development of CVD and on CVD outcomes, particularly as barriers to treatment implementation.
2. The diagnosis of anxiety and depression disorders, recognition of suicidality, and the appropriate use of referrals.
3. Use of pharmacotherapies in the treatment of depression including patient selection, side effects, monitoring for efficacy, and impact on risk factors.
4. Recognition of safe treatments for anxiety and depression in patients with CVD.
5. Recognition of when referral for psychiatric or psychological care is needed and appropriate.
6. Cardiac rehabilitation for the assessment and management of psychosocial conditions related to CVD.
7. The role of healthcare systems and financing on psychosocial risks and outcomes for CVD, including the role of social workers and case managers in identifying and facilitating social services.

## 7. Advanced Risk Assessment (Renal, Inflammatory Diseases)

### 7.1. Justification

The assessment of both traditional and nontraditional risk factors underlies the primary and secondary prevention of CVD. Novel biomarkers are emerging as prognostic tools for CVD risk assessment. Persons with chronic kidney disease are considered to be a CHD risk equivalent by some guidelines (93,94). The doses of many cardiovascular medications need to be adjusted in persons with chronic kidney disease, especially as glomerular filtration rate declines and chronic kidney disease worsens.

Adults with inflammatory diseases such as lupus, psoriasis, or rheumatoid arthritis seem to be prone to accelerated atherothrombotic vascular disease (95,96). Healthcare providers need to be more aggressive in trying to motivate patients with chronic kidney disease or inflammatory disorders to optimize their lifestyle habits and to achieve optimal levels of blood pressure and lipids. A number of ongoing studies are trying to assess the role of chronically high levels of inflammation in the development of CVD. Persons with lupus may also need to be screened for a prothrombotic state.

Recent studies have also shown that acute myocardial infarction (MI) rates and cardiovascular risk factors are increased in persons with human immunodeficiency virus (HIV) infection as compared with non-HIV patients (97,98). Certain classes of antiretroviral drugs, especially protease inhibitors, appear to promote dyslipidemia and may independently increase risk via inflammatory pathways (99). Strategies to reduce risk for atherosclerotic vascular

disease should be incorporated into the standard care of HIV infection.

There is considerable ongoing research dealing with the prognostic role of biomarkers in persons with renal, inflammatory, or chronic infectious disease in both the primary and secondary prevention settings. In future years, we will have a better understanding of when measurement of biomarkers such as C-reactive protein, B-natriuretic peptide, and urinary microalbumin should change standard clinical management and the intensity of risk factor modification (100–102).

### 7.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. Measurement of urinary albumin creatinine ratio in changing pharmacologic management.
2. Inquiry regarding erectile difficulties on at least a yearly basis as a marker for early atherosclerotic vascular disease and generalized endothelial dysfunction.
3. Measurement of systolic blood pressures in each brachial artery and one of the pedal arteries in each foot to calculate an ankle-brachial index, and the therapeutic implications for diagnosis of peripheral vascular disease, a CHD risk equivalent.
4. Individuals with certain infectious diseases, chronic inflammatory conditions, and other collagen vascular diseases, as they are often at increased risk for atherosclerotic vascular disease and need to undergo comprehensive risk factor modification (97).
5. Aggressive management of all risk factors, as appropriate, in patients with a history of chest wall irradiation, as they are prone to premature atherosclerotic disease.

## 8. Subclinical Atherosclerosis Assessment (Imaging and Nonimaging)

### 8.1. Justification

The preclinical detection of atherosclerosis is an area of growing interest. The concept is to detect lesions in the cerebral, coronary, or peripheral vasculature before symptoms of end-organ ischemia occur (transient ischemic attack/stroke, angina/MI, claudication/limb ischemia), or before rupture and bleeding (aortic aneurysm) (19,20,103). Two recent studies from the MESA (Multi-Ethnic Study of Atherosclerosis) trial have clearly documented the prognostic power of elevated coronary calcium scores (104,105). Such patients could be targeted for intensive risk factor control, other medical interventions, and endovascular or surgical treatments if indicated.

While the concept of preclinical detection is appealing for several reasons, controversy exists about the usefulness and efficacy of some screening programs and paradigms. The preventive cardiovascular specialist should have the knowledge base and skills to 1) advise patients about the useful-

ness of such screening, including costs; 2) interpret the results of a screening test in terms of formulating a care plan; and 3) provide guidance about the need for subsequent testing and therapy.

Some screening approaches entail financial as well as potential medical risks, particularly if a positive test leads to further investigations and in some cases medical, surgical, or endovascular interventions. Thus, it is important to have some guidance about what competencies are needed in these areas.

## 8.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. General epidemiology and risk factors for asymptomatic disease in various vascular beds. Knowledge of these factors and associations will be helpful to the clinician in determining which patients are at higher versus lower risk to have asymptomatic disease in various vascular beds.
2. While controversial as to efficacy, the potential screening modalities for asymptomatic disease in the various vascular beds (Table 3) (18,20,106–118).
3. Technical limitations and overall sensitivity, specificity, accuracy, and risks of various screening tests (106,111, 113,118,119). Radiation exposure from coronary computed tomography angiogram may increase the risk of breast cancer, but the amount of ionizing radiation associated with the test is considerably less than that associated with a stress radionuclide examination (120). Knowledge of these factors is important for the clinician to weigh the results and risks of such screening tests and determine the need for further testing depending on the overall clinical scenario.
4. The need for further testing in patients with evidence of asymptomatic disease (Table 3). This would include the clinical indications for further testing, the methods used for subsequent testing, and their limitations (false positives, false negatives, overall accuracy) (103,108,118).
5. How to treat patients with the presence of asymptomatic disease in various vascular beds, the risks and benefits of these various treatments and interventions, and patient education and knowledge of sources for such educational information (i.e., Web sites, nonprofit organizations).
6. How to order and/or interpret the above screening tests, understand the test results, explain their meaning to the patient, and plan further testing and treatment, perhaps

in consultation with other medical specialists in related areas of vascular medicine.

## 9. Adherence and Disease Outcome Interdisciplinary Programs

### 9.1. Justification

Adherence is a measure of how consistently a patient follows the specific requirements of an intervention. Knowledge about how to achieve a superior level of adherence is crucial. While the true rate of patient adherence is difficult to measure without using sophisticated tools that in themselves may influence patient behaviors, research generally indicates that long-term adherence to behavioral and medical intervention may be as low as 50% (121). Nevertheless, in some settings and in some patients much higher rates of adherence occur, suggesting that low rates of adherence result from specific causal factors such as the cost of medications, depression, or low health literacy, and that adherence is amenable to change. Some personal factors such as personality traits do not consistently influence adherence while others such as self-efficacy do. Societal, healthcare system, and provider factors likely influence adherence rates (122–124). Successful prevention often requires lifelong actions by the patient, and therefore, a high level of long-term adherence is very important in effective prevention (125).

Disease outcome or management programs usually consist of at least 2 program elements: a patient monitoring component and a system to respond proactively to changes in the patient's symptoms or physical status. Effective disease management programs should reduce or delay the adverse consequences of chronic CVD events, such as preventing or reducing the number of HF hospitalizations in patients with HF, and reduce the episodic nature of health care based on the treatment of acute episodes (21). The long-term efficacy of most disease programs is uncertain. Since many patients have several chronic illnesses or complex prevention problems, the concepts underlying disease outcome interdisciplinary programs may in the future be applied to a wider set of prevention problems. Both adherence and disease outcome management programs are based on the integration of biologically derived scientific concepts with behavioral and social science concepts.

**Table 3. Testing Modalities**

Vascular Bed	Initial Testing	Follow-Up Tests
Carotid	Auscultation, carotid duplex ultrasound	MRA, CTA, carotid angiography, if indicated
Coronary	CAS, CTA	Stress test with or without imaging, if indicated; coronary angiography, if indicated
Aorta	Palpation, abdominal ultrasound	CTA, MRA
Noncerebral peripheral arteries	Ankle-brachial index	Treadmill exercise test, PVRs, duplex ultrasound, CTA, MRA

CAS indicates coronary artery calcium score; CTA, computed tomography angiogram; MRA, magnetic resonance angiography; and PVRs, pulse volume recordings.

## 9.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. How to measure adherence through a variety of approaches such as history and medication reconciliation.
2. How to determine the causal factors underlying nonadherence, including the important patient factors such as health literacy, depression, comorbid conditions, trust in the healthcare provider, adverse effects, and economic factors.
3. Patient-level interventions to improve adherence such as reducing the cost of medications, treating depression, and use of aids like weekly pill boxes.
4. Important provider factors such as interest in adherence and skill in identifying barriers to adherence, combined with understanding important healthcare system and societal factors such as ease of maintaining the intervention or medication, cost, systematic reminders, and number and frequency of patient contacts.
5. Components of a successful disease outcome program such as prompt evaluation and detection of new symptoms and adjustment of medications in response to changes in symptoms; frequency and duration of patient interactions with the disease management program; and mode of collecting information on patient status, including self-reports and automated devices.
6. Disease management programs, the role of feedback to the patient and provider beyond adjustment of therapeutic regimen, and the expected roles of the patient and their family, the provider, and other health personnel in a disease management program.
7. Common theoretical models for adherence and disease management such as: Stages of Change (22), Wagner's Chronic Care Model (23), and Self Efficacy (24).
8. Motivational interviewing and other patient empowerment techniques.

## 10. Nutrition Management

### 10.1. Justification

Many of the conditions and disease states that affect atherosclerotic risk can be prevented or at least modified by dietary interventions. These conditions and diseases include obesity and excess body weight, hypertension, lipid abnormalities, and diabetes. Even when conditions such as hypertension and hyperlipidemia are established and require pharmacologic therapy, dietary manipulations can reduce the dosage of medication required to achieve therapeutic goals. Similarly, some nutritional supplements (e.g., red rice yeast [126] and omega-3 fatty acids [127]) contain pharmacologically active substances that can be used therapeutically in selected patients. In contrast, some dietary and nutritional supplements may contain substances such as sympathomimetics that increase cardiac arrhythmias. Con-

sequently, healthcare providers practicing preventive cardiology must be required to have an understanding of nutrition and the principles of nutrition so that they are able to provide expert advice to patients and to reinforce expert advice given by nutritional professionals. In addition, credibility as a preventive specialist with patients is enhanced when the preventive cardiologist is conversant in the basics of diets and nutritional therapy.

### 10.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. Obesity, its role in producing insulin resistance and the metabolic syndrome, the healthcare provider's role in helping patients maintain body weight, the basic classifications of body weight, the contribution of overweight to hypertension and hyperlipidemia, the basic concepts of caloric balance, the caloric content of the major food groups, the role of exercise in helping to maintain body weight, and the action of the available prescription weight loss drugs and their possible cardiovascular side effects, including that recommendations may differ according to age (pediatric versus adult versus very elderly).
2. Surgical approaches to morbid obesity and the possible medical complications of this surgery.
3. Composition of diets used to assist in blood pressure control such as reduced sodium, calorie reduction for weight loss, moderation of alcohol consumption, increased potassium intake, and consumption of an overall healthy diet (128).
4. Composition of therapeutic diets used to manage hypercholesterolemia and hypertriglyceridemia and especially the effect of saturated fats, transfatty acids, and low dietary fat and cholesterol on serum lipids (129). In addition, the preventive cardiology specialist should be conversant in discussing the origin of dietary fats with regard to therapeutic potential (27,129).
5. Composition of diets used in patients with impaired glucose tolerance and diabetes (130).
6. Use of over-the-counter and nonprescription agents in lipid management including the risks and benefits of plant stanols and sterols (131), fish oils (127), and dietary fiber (127,132).
7. Potential cardiac dangers of too-rapid weight loss (133).
8. The limited role of dietary antioxidants in preventing atherosclerotic disease (126,134).
9. The role of alcohol intake in causing and preventing cardiac disease (135,136).

## 11. Lipid Management (Management of Dyslipidemia)

### 11.1. Justification

The management of dyslipidemia has emerged as a key therapeutic strategy to reduce both primary and secondary

cardiovascular events. Despite a plethora of large outcome trials (137–139) supporting the evidence for lipid-altering treatments to improve outcomes and national guidelines that have established specific goals of treatment, there remains a significant treatment gap in the achievement of low-density lipoprotein cholesterol (LDL-C), non-HDL-C, and HDL-C targets (140,141). The National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) Guidelines (142) updated in 2004 (143) mandate an LDL-C goal for high-risk patients of less than 100 mg/dL with an optional goal of less than 70 mg/dL for patients with CVD plus diabetes or other multiple risk factors. The AHA Secondary Prevention Guidelines also advocate an LDL-C goal and non-HDL-C goal of less than 70 mg/dL and less than 100 mg/dL, respectively (10). Yet, recent surveys demonstrate that less than one third of patients with CVD with additional risk factors are achieving these recommended targets (140). In addition, patients with low HDL-C and/or elevated triglycerides remain at elevated residual risk even at recommended LDL-C goals (144–146).

### 11.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. Basic lipid metabolism, including both exogenous and endogenous lipoprotein synthesis.
2. Synthetic process in which chylomicrons, very low-density lipoprotein, LDL-C, and HDL-C are developed, and the genetic disorders that are associated with dyslipidemia.
3. The diagnosis of familial hypercholesterolemia, familial combined hyperlipidemia, and familial hypoalphalipoproteinemia, which are associated with a significantly increased risk of premature atherosclerosis.
4. Familiarity with NCEP guidelines, including the recent updates that recommend optional LDL-C targets for very high-risk patients.
5. Matching the intensity of treatment to the risk of the patient. This is the cornerstone of national guidelines, and the preventive cardiovascular specialist should be familiar with the clinical trials that provided the evidence for more aggressive LDL-C reduction.
6. Ability to instruct a patient on appropriate therapeutic lifestyle changes, which includes an understanding of a low saturated fat and dietary cholesterol food management program and, if necessary, the incorporation of viscous soluble fiber and plant stanols/sterols to further lower LDL-C levels, and the importance of reducing trans fats, cholesterol, and simple carbohydrates in patients with high triglycerides and abdominal obesity.
7. Mechanism of action of statins, the expected efficacy of each dose of statin and, therefore, the ability to achieve the NCEP ATP III goals, plus the mechanism of action of niacin, ezetimibe, fibrates, bile acid sequestrants, and omega-3 fatty acids and their expected effects on

LDL-C, HDL-C, and triglycerides and risks and benefits of combination therapy.

8. Potential side effects of treatment to counsel patients and avoidance of drug interactions, which is imperative to improve the safety and compliance of long-term treatment.

## 12. Thrombosis Management

### 12.1. Justification

Atherothrombosis is a progressive process that includes atherosclerotic plaque formation, disruption, and thrombosis. These processes constitute the pathophysiology that underlies acute coronary syndrome, ischemic stroke or transient ischemic attack, and peripheral arterial disease (PAD). U.S. prevalence data lists 7.2 million people affected by MI, 6.5 million with angina pectoris, 5.5 million with stroke (147), and 8 to 12 million people affected by PAD (148). The NCEP ATP III considered PAD to be a CHD risk equivalent (142). The recently published REACH (Reduction of Atherothrombosis for Continued Health) registry showed that among patients with symptomatic atherothrombosis, 16% had symptomatic polyvascular disease (149). Approximately 56% of stroke patients 60 years of age and older have coexisting coronary artery disease (CAD), and evidence suggests the 20% to 40% of patients with ischemic stroke or transient ischemic attack concurrently have silent CAD (150,151).

### 12.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. The core set of risk factors, which includes hypertension, diabetes, hypercholesterolemia, tobacco use, and obesity, that contribute to the bulk of the risk for atherothrombosis (149), and novel parameters such as lipoprotein(a), apolipoprotein A-I and apolipoprotein B-100, fibrinogen, homocysteine, and high-sensitivity C-reactive protein (10,152).
2. Management of unstable angina and non-ST-segment elevation MI is extensive with multiple antithrombotic therapy combinations, and implement systems for antithrombotic therapy recommended in the ACC/AHA guidelines for unstable angina and non-ST-segment elevation MI (153).
3. Antithrombotic therapy options for preventing recurrent stroke and other cardiovascular events in patients with noncardioembolic ischemic stroke (32).
4. Risk factor identification and early assessment of PAD (149,152,154), and use of antithrombotic agents to prevent atherothrombotic events in this patient population (155–158), including their indications and contraindications, mechanism of action, efficacy, side effects, drug interactions, and costs (159).

## 13. Hypertension Management

### 13.1. Justification

Hypertension is a major contributor to the global disease burden and is one of the leading preventable causes of premature death worldwide (160). In the United States, a disproportionate burden of hypertension and its associated complications, including CHD, HF, stroke, and end-stage renal disease and CVD mortality, affect African Americans (also referred to as U.S. blacks) (161). Preventive cardiovascular specialists should have knowledge of hypertension and mechanisms of elevated blood pressure, and an understanding of the therapeutic lifestyle changes and pharmacologic interventions that are crucial for controlling hypertension in clinical practice. A basic knowledge of the potential mechanisms of elevated blood pressure and associated risk factors is necessary to understand the ongoing research into new approaches for prevention, identification, and therapy. A basic understanding of the use of therapeutic lifestyle changes and appropriate drug therapies for patients with compelling indications is necessary to reduce cardiovascular morbidity and mortality.

### 13.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. Major risk factors for hypertension as described by the Seventh Report of the Joint National Committee (35), and the impact of hypertension on CVD morbidity and mortality specifically related to MI, HF, stroke, and renal failure, and the diagnostic categories and treatment goals.
2. Therapeutic lifestyle and pharmacologic interventions in hypertension and CVD including weight reduction, adopting Dietary Approaches to Stop Hypertension (DASH) eating plan, sodium reduction, physical activity, moderation of alcohol consumption, and medication regimens.
3. Indications for and interpretation of ambulatory blood pressure and home blood pressure monitoring.
4. Definition and approach to white coat hypertension and masked hypertension.
5. Evaluation and treatment of *identifiable causes of hypertension* (secondary hypertension), including chronic kidney disease, coarctation of the aorta, glucocorticoid excess states, pheochromocytoma, primary aldosteronism, renovascular hypertension, sleep apnea, thyroid/parathyroid disease, and drug induced or drug related.
6. Definition, differential diagnosis, and treatment of resistant hypertension.
7. The findings of clinical trials in hypertension with evidence supporting compelling indications and contraindications for antihypertensive drug classes.

## 14. Smoking Cessation

### 14.1. Justification

Smoking remains the most important risk factor for CVD in the world (162,163). It is estimated that 40% of all heart disease is related to smoking (164,165). The biochemical and physiological consequences of smoking on CVD are well defined (164,166,167). Compelling evidence exists demonstrating that smoking cessation is associated with significant reversal of risk for CAD, stroke, and cancer-related deaths (162). In addition, there are multiple societal consequences from cigarette smoking, including enormous economic costs. In the United States alone, it is estimated that smoking costs \$167 billion each year (162). Since 1965, smoking in the United States has declined by 47% among people over the age of 18 years. However, it is estimated that more than 23% of adult men and 19% of adult women continue to smoke, and this number is rising in the young (162). In addition, exposure to secondhand smoke places significantly more persons at risk for heart disease and stroke (168,169).

Clinical competency in smoking cessation treatment is critical for those whose expertise encompasses primary and secondary prevention of CVD and stroke. Clinical competency includes skills in patient education, counseling, and behavioral change, and knowledge of important pharmacotherapies, including risks and benefits. Clinical competency relies on the identification of smoking status in all patients, prompt and definitive advice to quit, and the implementation of smoking cessation counseling and pharmacotherapies. Systematic follow-up of all smokers at subsequent visits and the involvement of healthcare professionals with smoking cessation expertise improves lifetime smoking cessation.

### 14.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. Short- and long-term pathophysiological consequences related to tobacco use, which include pulmonary and cardiovascular diseases (36,166,167,170–176).
2. Pathophysiology of smoking addiction and be knowledgeable of the methods of assessing the physiologic levels of nicotine addiction (e.g., Fagerstrom Questionnaire) (176).
3. Use of behavioral skills that facilitate smoking cessation, including interpersonal communication skills, behavioral change techniques such as self-monitoring and self-care, stress management counseling, patient contracting and goal setting, self-efficacy, motivational interviewing, and relapse prevention (170,174,176–178).
4. Use of the 5 As (Ask, Advise, Assess, Assist, and Arrange) treatment intervention during office visits.

Each visit should include preparation, relapse prevention, and counseling (170,174,176).

5. Use of pharmacologic therapies to support smoking cessation based on age, pregnancy status, medical history, and comorbidities of the patient (167,170,171,175, 179–183).

## 15. Obesity Management (Behavioral Programs)

### 15.1. Justification

Obesity is a disease that is reaching epidemic proportions, not only in the United States, but also elsewhere throughout the world. At the present time, more than 60% of the U.S. adult population is classified as either overweight or obese (39,184). In addition, childhood obesity is growing in an alarming fashion. Overweight and obesity are particularly prevalent in certain minority groups and in individuals of lower socioeconomic status. Overweight is defined as a body mass index of 25 to 29.9 kg/m<sup>2</sup>, and obesity as a body mass index greater than 30 kg/m<sup>2</sup> (40). Higher body weight is associated with an increased risk of hypertension, hyperlipidemia, stroke, CAD, insulin resistance, and type 2 diabetes mellitus. Moderate weight loss has been shown to decrease the severity of these comorbidities, and data from observational studies suggest a concomitant decrease in mortality (41,185).

Overweight and obesity are felt to result from an imbalance between energy intake and expenditure. Less than 20% of American adults regularly engage in moderate physical activity. The AHA identified an “epidemiological triad” in Prevention Conference VII (November 2004), which includes *host factors* (genetic makeup, age/gender, attitudes, and behavior), *vectors for increased energy consumption or decreased energy expenditure* (i.e., automobile travel instead of walking or biking, large portion sizes, and high-fat and high-calorie foods), and *environmental factors* (i.e., cost of goods, government policy, as well as sociocultural forces). They suggest that all components need to be addressed in order for successful prevention to occur (186).

A variety of behavioral options exist to manage overweight and obesity effectively. These include dietary therapy, physical activity, and behavioral techniques. To be successful in achieving long-term weight maintenance, however, these methods have to be individually applied to each patient in the context of regular and consistent medical supervision. Reduction of initial body weight by only 5% to 10% has been shown to result in significant cardiovascular risk factor reduction (187), as well as a variety of other health benefits. Presently, training on overweight/obesity in specialty and subspecialty medical education is woefully inadequate.

### 15.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. Assessment of the overweight/obese patient involves calculation of body fatness, which can be estimated by body mass index, waist circumference, overall cardiovascular risk status, as well as the patient’s motivation to lose weight, including the methods for measurement of these. Importance of documenting previous history of weight loss attempts, social supports available, the patient’s attitude toward and ability to perform physical activity, as well as financial considerations that may impact his or her attempts at weight loss.
2. Initial goal of weight loss therapy is to reduce body weight by 7% to 10% over a 6-month period of time, after which the rate typically declines. Knowledge that reassessment should occur to determine if further weight loss is desirable and that efforts at maintaining weight loss must be put into place.
3. Caloric content and caloric balance of the major food groups, the physiologic and pathologic effects of excess body weight, and the principles of weight loss via reduced caloric intake and increased caloric expenditure.
4. Combination of individually planned dietary, exercise, and behavioral techniques, and when to use a low-calorie diet versus a very-low-calorie dietary strategy (188), as well as an understanding of exercise physiology and guidelines for activity prescription, including aerobic exercise and strength training (189).
5. Behavioral therapy, with the use of other healthcare professionals, to include management/problem-solving techniques for stress, enhancing social support, cognitive restructuring, and promoting the ability to self-monitor, as well as setting realistic goals.
6. Recognition that maintenance of weight loss is often more difficult than the initial loss; thus, the practitioner must be skilled at understanding the utility of a multifaceted program that consists of ongoing dietary management, physical activity, as well as behavioral management.
7. Challenges of treating special populations, such as children/adolescents, the elderly, and smokers.
8. Guidelines for pursuing pharmacologic or surgical means of weight loss therapy if behavioral programs are not successful.
9. Pharmacology, use, and side effects of weight loss medications, both prescription and nonprescription.
10. Counseling of patients regarding the nutritional and medical risks associated with rapid weight loss (particularly that mediated by surgical intervention) that may impact CVD, including iron and B12 deficiency anemia, folate deficiency, as well as the potential for other deficiencies, including vitamin D and other fat-soluble vitamins. Knowledge that physical fitness

should be optimized prior to any drastic means of weight loss.

## 16. Exercise Physiology, Physical Activity Management, and Cardiac Rehabilitation (Secondary Prevention)

### 16.1. Justification

Important goals of exercise-based cardiac rehabilitation are to stabilize existing atherosclerotic plaques, improve endothelial function and lessen arterial inflammation by modulating lipid/lipoprotein levels and blood pressure, and achieve smoking cessation, if appropriate (190,191). Additional objectives are to: increase functional capacity, decrease symptoms, reduce body weight and fat stores, promote psychosocial well-being, and improve the ability of the patient to return to work (192,193). Recent meta-analyses indicate that exercise-based cardiac rehabilitation improves the cardiovascular risk factor profile and reduces all-cause and cardiovascular mortality, and that these benefits persist in the current era of cardiovascular therapeutics (194).

In the past, only post-MI patients were considered candidates for exercise-based cardiac rehabilitation. However, the proven benefits and safety of this intervention have expanded to include patients with angina, diabetes or metabolic syndrome, cardiomyopathy, pacemakers, heart valve replacement, concomitant pulmonary disease, cardiac transplant, and HF, as well as patients who have undergone percutaneous coronary intervention or coronary artery bypass graft surgery (191,192,195), yet these diagnoses are not all covered by health insurance.

Moderate-to-vigorous physical activity and improved cardiorespiratory fitness reduce cardiovascular-associated morbidity and mortality by multiple mechanisms (189,195), including antiatherosclerotic, anti-ischemic, antiarrhythmic, and antithrombotic effects. Each 1 metabolic equivalent (MET) (1 MET = 3.5 mL O<sub>2</sub>/kg/min) increase in exercise capacity appears to confer an 8% to 17% reduction in mortality (196). Alternatively, an approximate 1,000-kcal/week increase in activity confers the equivalent survival benefit that would accrue by increasing cardiorespiratory fitness by 1 MET (197). Exercise testing may be helpful in quantifying aerobic capacity and in establishing a safe and effective exercise prescription (198,199).

### 16.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. Prescription of exercise in primary prevention populations.
2. Evaluation, appropriate interventions, and expected outcomes for each of the core components of contemporary cardiac rehabilitation/secondary prevention programs, including exercise training, risk factor modification, medical surveillance/emergency support, and psychosocial (e.g., stress management)/vocational

- counseling (200), as well as alternative delivery sources (e.g., site-supervised versus home-based [telephone, Internet, completion of exercise logs] programs) (191).
3. ACC/AHA current guidelines for secondary prevention for patients with coronary and other atherosclerotic vascular disease, including smoking cessation, blood pressure control, lipid management, physical activity, weight management, diabetes management, antiplatelet agents/anticoagulants, renin-angiotensin-aldosterone system blockers, beta blockers, and influenza vaccination (10).
4. Multidisciplinary team approach to implementing an effective cardiac rehabilitation/secondary prevention program (200,201).
5. Traits, emotional states, and life situations that have been linked to CVD; for example, depression, anxiety, anger/hostility, social isolation, vital exhaustion, and chronic life stresses.
6. Stages of behavior change and the concept of motivational interviewing to enable patients to favorably modify long-standing, deleterious lifestyle habits.
7. Administration and interpretation of exercise tests as per the ACC/AHA 2000 Clinical Competence Statement on Stress Testing (199), with specific reference to the following variables: indications and contraindications; test end points; peak or maximal oxygen uptake; hemodynamics; rating of perceived exertion; recognition of supraventricular and ventricular arrhythmias; and interpretation of ST-segment displacement (5,196,198,199). Competence includes the knowledge and skills required for certification in AHA Basic and Advanced Life Support.
8. Use of exercise test results for activity counseling and exercise prescription (5,196,198).
9. Inverse relationship between physical activity and/or cardiorespiratory fitness, expressed as mL O<sub>2</sub>/kg/min or METs, and cardiovascular and all-cause mortality in persons with and without known CAD (189,196,197).
10. Safety, benefits, rationale for and contraindications to endurance training and resistance exercise in primary and secondary prevention programs (5,196,198,202).
11. Basic terminology and fundamentals of exercise physiology, with specific reference to the interpretation of clinical exercise testing and the prescription of exercise in health and disease (5,196,198).
12. Spectrum of cardiovascular conditions among patients who require physical activity counseling and/or might benefit from exercise training, including pathophysiology, signs and symptoms of stable and unstable disease, and medical and surgical treatments for the following conditions: CAD, including recent MI, postpercutaneous coronary intervention and postcoronary artery bypass grafting; cardiomyopathy and HF; valvular heart disease; peripheral arterial disease; hypertension; cardiac arrhythmia; and cardiac transplantation.



13. The role of orthostatic/gravitational stress (e.g., intermittent sitting/standing) in preventing deconditioning during the hospital confinement period and early convalescence after an acute coronary event or intervention (203).
14. Prescription of exercise (5,196,198), with respect to the intensity (e.g., threshold or minimal effective dosage) (204), frequency, duration, and modes of endurance exercise training, as well as lifestyle physical activity (205) and appropriate tracking modalities (e.g., pedometers) with specific reference to those patients with cardiovascular conditions as listed above.
15. Comorbidities and how they might affect exercise prescription and performance. This is especially important in diabetes mellitus, chronic pulmonary disease, and dialysis-dependent chronic kidney disease.
16. Application of the elements of risk stratification (e.g., ejection fraction, signs/symptoms of myocardial ischemia, cardiorespiratory fitness) in triaging coronary patients to varied degrees of monitored/supervised exercise-based rehabilitation programs (191–193).
17. Variables that influence a patient's participation in and adherence to an exercise-based cardiac rehabilitation program (191), including the importance of physician support and encouragement (201).
18. Selected high-intensity, anaerobic-type domestic, occupational, or recreational activities (e.g., running, racquet sports, snow removal) may pose an increased risk of acute cardiovascular events, especially in habitually sedentary persons with known or occult CAD (206).
19. The role that physical activity plays as primary prevention for CVD as a public health measure.

## **17. Prediabetes, Metabolic Syndrome, Insulin Resistance, and Diabetes Management**

### **17.1. Justification**

There are very strong epidemiological data available to support the concept that type 2 diabetes is associated with greatly increased risk of cardiovascular events. In addition, patients with type 1 diabetes of long duration, with other complications, are also at risk for cardiovascular events. Several clinical trials have assessed the effect of glycemic control on long-term microvascular complications, including CVD. Good glycemic control utilizing effective insulin regimens in type 1 diabetes may prevent CVD, and trials of the effect of intensive therapy on CVD in type 2 diabetes are in progress. The metabolic syndrome, prediabetes, and insulin resistance also portend an elevated risk of CVD.

### **17.2. Minimal Knowledge**

The expert in the prevention of CVD should demonstrate knowledge and competence in:

1. The understanding of molecular and cellular mechanisms of impaired glucose tolerance, hyperinsulinemia, metabolic syndrome, and overt diabetes.
2. Methods of cardiovascular risk assessment in diabetic patients, including risk engines and other tools developed to assess cardiovascular risk in patients with type 2 diabetes.
3. Adherence and disease outcome interdisciplinary programs that meet the educational standards recommended by the ADA. Such programs may be effectively integrated with weight management programs and other risk factor management education strategies.
4. Principles of diabetes management, both behavioral and pharmacological, and multiple resources that are available from organizations such as the ADA and American Association of Clinical Endocrinologists that are available on Web sites for both patients and healthcare professionals.
5. Criteria for effective screening and diagnosis of prediabetes, insulin resistance, and metabolic syndrome (207), including the several definitions for the metabolic syndrome that have been recommended.
6. Identification of patients at risk for type 2 diabetes, understanding treatment and when to refer to a specialist. Management of prediabetes consists of lifestyle changes (208).
7. Management of overt diabetes, including the goals for metabolic control standards of care for people with diabetes, essential clinical and laboratory tests and requirements for screening for complications, and individualized pharmacological therapy that are regularly published and updated by the ADA and other organizations (209).
8. Implementation of guidelines regarding maintenance of normoglycemia in the intensive care unit setting (210).

## **18. Chronic Disease Management**

### **18.1. Justification**

Patient-specific and systems approaches to the primary and secondary prevention of CVD are essential for optimal atherosclerosis disease management (211–213). This type of approach must encompass a simple and meaningful strategy for both patient and provider throughout the process of risk assessment and intervention (medication management and lifestyle modification) (214). Guidelines are based on the results of randomized trials (10).

Too often, providers forget to implement the standard guidelines that deal with antiplatelet therapy, antihypertensive therapy, lipid-lowering therapy, and lifestyle changes; all of these items are important in improving the quality of medical care. As a profession, we need to improve the frequency with which evidence-based guidelines are applied in clinical practice. Many guidelines have been published, but there has been a disappointing lack of standard implementation.

Basic physician education and passive dissemination of guidelines alone are generally insufficient to sustain quality improvement. Chart audit and feedback of results, reminder systems to consider use of specific medicines or tests, and the use of local opinion leaders have had variable results. Multifactorial interventions that simultaneously attack different barriers to change tend to be more successful than isolated efforts. Dissemination of practice guidelines and knowledge of cardiovascular prevention must be accompanied by more intensive educational and behavioral interventions to maximize the chances of improving physician practice patterns.

The AHA has endorsed the Get With The Guidelines approach, and the ACCF has endorsed an approach to the management of chronic stable angina and the prevention of CVD in general (212,215). These approaches should help medical practices and the individual patient better understand the various pharmacologic therapies available for a given individual condition.

The ACCF Guidelines Applied in Practice (GAP) program is also a well-conceived systems approach that has been focused on patients presenting with acute coronary syndromes (216–218). The same principles of prompt initiation of antiplatelet therapy, beta blockade, inhibition of the renin-angiotensin-aldosterone system, cholesterol-lowering therapy, and better dietary and exercise habits can also be modified for use in chronic management of persons with atherosclerotic vascular disease (219–221).

### 18.2. Minimal Knowledge

The expert in the prevention of CVD should demonstrate knowledge and competence in:

- Existing patient-specific, systems approaches to prevention of CVD, including:
  - The implementation of the AHA's Get With The Guidelines.
  - Chronic disease management programs specific to CVD.
  - The basics of the chronic care model applied to CVD prevention (222–224).
  - Understanding of current performance measures for primary and secondary prevention of CVD in all practice settings.
  - Implementation of performance improvement measures.

### Staff

#### *American College of Cardiology Foundation*

John C. Lewin, MD, Chief Executive Officer  
Charlene May, Senior Director, Science and Clinical Policy  
Tanja Kharlamova, Associate Director, Science and Clinical Policy  
Fareen Pourhamidi, MS, MPH, Senior Specialist, Evidence-Based Medicine  
María Velásquez, Specialist, Science and Clinical Policy  
Erin A. Barrett, Senior Specialist, Science and Clinical Policy

#### *American Heart Association*

Nancy Brown, Chief Executive Officer  
Rose Marie Robertson, MD, FAHA, FACC, FESC, Chief Science Officer  
Gayle R. Whitman, PhD, RN, FAHA, FAAN, Senior Vice President, Office of Science Operations  
Kathryn A. Taubert, PhD, FAHA, Senior Scientist

### REFERENCES

- Hirsh J, Guyatt G, Albers GW, et al. Antithrombotic and thrombolytic therapy: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th edition). *Chest*. 2008;133:1105–25.
- National Cancer Institute. Prevention and Cessation of Cigarette Smoking: Control of Tobacco Use (PDQ), February 2008. Available at: <http://www.cancer.gov/cancertopics/pdq/prevention/control-of-tobacco-use/Patient/page2>. Accessed July 1, 2009.
- National Library of Medicine. Quitting smoking. Available at: <http://www.nlm.nih.gov/medlineplus/quittingsmoking.html>. Accessed July 2, 2009.
- American Heart Association. Smoking and cardiovascular disease. Available at: <http://www.americanheart.org/smokingcessation>. Accessed July 2, 2009.
- American College of Sports Medicine. ACSM's Guidelines for Exercise Testing and Prescription. 7th edition. Baltimore, Md: Lippincott Williams & Wilkins, 2006.
- Arnett DK, Baird AE, Barkley RA, et al. Relevance of genetics and genomics for prevention and treatment of cardiovascular disease: a scientific statement from the American Heart Association Council on Epidemiology and Prevention, the Stroke Council, and the Functional Genomics and Translational Biology Interdisciplinary Working Group. *Circulation*. 2007;115:2878–901.
- Pearson TA, Bazzarre TL, Daniels SR, et al. American Heart Association guide for improving cardiovascular health at the community level: a statement for public health practitioners, healthcare providers, and health policy makers from the American Heart Association Expert Panel on Population and Prevention Science. *Circulation*. 2003;107:645–51.
- Mosca L, Banka CL, Benjamin EJ, et al. Evidence-based guidelines for cardiovascular disease prevention in women: 2007 update. *J Am Coll Cardiol*. 2007;49:1230–50.
- Pearson TA, Blair SN, Daniels SR, et al. AHA guidelines for primary prevention of cardiovascular disease and stroke: 2002 update: Consensus Panel Guide to Comprehensive Risk Reduction for Adult Patients Without Coronary or Other Atherosclerotic Vascular Diseases. American Heart Association Science Advisory and Coordinating Committee. *Circulation*. 2002;106:388–91.
- Smith SC Jr., Allen J, Blair SN, et al. AHA/ACC guidelines for secondary prevention for patients with coronary and other atherosclerotic vascular disease: 2006 update endorsed by the National Heart, Lung, and Blood Institute. *J Am Coll Cardiol*. 2006;47:2130–9.
- U.S. Department of Health and Human Services. U.S. Surgeon General's family history initiative: resource packet for health professionals. Available at <http://www.hhs.gov/familyhistory/respachealth.html>. Accessed May 11, 2009.
- American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th edition, text revision. American Psychiatric Publishing, Inc., 2000.
- Bunker SJ, Colquhoun DM, Esler MD, et al. "Stress" and coronary heart disease: psychosocial risk factors. *Med J Aust*. 2003;178:272–6.
- Huffman JC, Smith FA, Blais MA, et al. Recognition and treatment of depression and anxiety in patients with acute myocardial infarction. *Am J Cardiol*. 2006;98:319–24.
- Milani RV, Lavie CJ, Cassidy MM. Effects of cardiac rehabilitation and exercise training programs on depression in patients after major coronary events. *Am Heart J*. 1996;132:726–32.

16. Taylor CB, Youngblood ME, Catellier D, et al. Effects of antidepressant medication on morbidity and mortality in depressed patients after myocardial infarction. *Arch Gen Psychiatry*. 2005;62:792–8.
17. Goldstein LB, Adams R, Albers MJ, et al. Primary prevention of ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council. *Stroke*. 2006;37:1583–633.
18. Greenland P, Bonow RO, Brundage BH, et al. ACCF/AHA 2007 clinical expert consensus document on coronary artery calcium scoring by computed tomography in global cardiovascular risk assessment and in evaluation of patients with chest pain: a report of the American College of Cardiology Foundation Clinical Expert Consensus Task Force (ACCF/AHA Writing Committee to Update the 2000 Expert Consensus Document on Electron Beam Computed Tomography). *J Am Coll Cardiol*. 2007;49:378–402.
19. Budoff MJ, Achenbach S, Blumenthal RS, et al. Assessment of coronary artery disease by cardiac computed tomography: a scientific statement from the American Heart Association Committee on Cardiovascular Imaging and Intervention, Council on Cardiovascular Radiology and Intervention, and Committee on Cardiac Imaging, Council on Clinical Cardiology. *Circulation*. 2006;114:1761–91.
20. Fleming C, Whitlock EP, Beil TL, et al. Screening for abdominal aortic aneurysm: a best-evidence systematic review for the U.S. Preventive Services Task Force. *Ann Intern Med*. 2005;142:203–11.
21. Krumholz HM, Currie PM, Riegel B, et al. A taxonomy for disease management: a scientific statement from the American Heart Association Disease Management Taxonomy Writing Group. *Circulation*. 2006;114:1432–45.
22. Prochaska JO, DiClemente CC. Stages and processes of self-change of smoking: toward an integrative model of change. *J Consult Clin Psychol*. 1983;51:390–5.
23. Wagner EH. Chronic disease management: what will it take to improve care for chronic illness? *Eff Clin Pract*. 1998;1:2–4.
24. Bandura A. Health promotion by social cognitive means. *Health Educ Behav*. 2004;31:143–64.
25. Lichtenstein AH, Appel LJ, Brands M, et al. Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee. *Circulation*. 2006;114:82–96.
26. Sacks FM, Lichtenstein A, Van HL, et al. Soy protein, isoflavones, and cardiovascular health: an American Heart Association Science Advisory for professionals from the Nutrition Committee. *Circulation*. 2006;113:1034–44.
27. Eckel RH, Borra S, Lichtenstein AH, et al. Understanding the complexity of trans fatty acid reduction in the American diet: American Heart Association Trans Fat Conference 2006: report of the Trans Fat Conference Planning Group. *Circulation*. 2007;115:2231–46.
28. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA*. 2001;285:2486–97.
29. Grundy SM, Pasternak R, Greenland P, et al. AHA/ACC scientific statement: assessment of cardiovascular risk by use of multiple-risk-factor assessment equations: a statement for healthcare professionals from the American Heart Association and the American College of Cardiology. *J Am Coll Cardiol*. 1999;34:1348–59.
30. Krumholz HM, Anderson JL, Brooks NH, et al. ACC/AHA clinical performance measures for adults with ST-elevation and non-ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Performance Measures (Writing Committee to Develop Performance Measures on ST-Elevation and Non-ST-Elevation Myocardial Infarction). *J Am Coll Cardiol*. 2006;47:236–65.
31. Hirsch AT, Haskal ZJ, Hertzner NR, et al. ACC/AHA 2005 guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): executive summary: a collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease). *J Am Coll Cardiol*. 2006;47:1239–312.
32. Sacco RL, Adams R, Albers G, et al. Guidelines for prevention of stroke in patients with ischemic stroke or transient ischemic attack: a statement for healthcare professionals from the American Heart Association/American Stroke Association Council on Stroke: co-sponsored by the Council on Cardiovascular Radiology and Intervention: the American Academy of Neurology affirms the value of this guideline. *Stroke*. 2006;37:577–617.
33. Calhoun DA, Jones D, Textor S, et al. Resistant hypertension: diagnosis, evaluation, and treatment: a scientific statement from the American Heart Association Professional Education Committee of the Council for High Blood Pressure Research. *Hypertension*. 2008;51:1403–19.
34. Rosendorff C, Black HR, Cannon CP, et al. Treatment of hypertension in the prevention and management of ischemic heart disease: a scientific statement from the American Heart Association Council for High Blood Pressure Research and the Councils on Clinical Cardiology and Epidemiology and Prevention. *Circulation*. 2007;115:2761–88.
35. Chobanian AV, Bakris GL, Black HR, et al. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension*. 2003;42:1206–52.
36. Ockene IS, Miller NH. Cigarette smoking, cardiovascular disease, and stroke: a statement for healthcare professionals from the American Heart Association. American Heart Association Task Force on Risk Reduction. *Circulation*. 1997;96:3243–7.
37. Agency for Healthcare Research and Quality. Helping smokers quit: a guide for clinicians. Available at: <http://www.ahrq.gov/clinic/tobacco/clinhlpsmksqt.htm>. Accessed July 1, 2009.
38. Fiore MC, Jaén CR, Baker TB, et al. Treating Tobacco Use and Dependence: 2008 Update. Clinical Practice Guideline. Rockville, Md: U.S. Department of Health and Human Services. Public Health Service. May 2008.
39. The National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report. *Obes Res*. 1998;6(suppl 2):51S–209S.
40. American Gastroenterological Association medical position statement on obesity. *Gastroenterology*. 2002;123:879–81.
41. World Health Organization. Obesity: preventing and managing the global epidemic. Report on a WHO consultation. 2000. Technical Report No. 894.
42. Stevens RJ, Kothari V, Adler AI, et al. The UKPDS risk engine: a model for the risk of coronary heart disease in Type II diabetes (UKPDS 56). *Clin Sci (Lond)*. 2001;101:671–9.
43. Nicholls SJ, Tuzcu EM, Sipahi I, et al. Intravascular ultrasound in cardiovascular medicine. *Circulation*. 2006;114:e55–9.
44. Libby P. Vascular biology of atherosclerosis: overview and state of the art. *Am J Cardiol*. 2003;91:3A–6A.
45. Mensah GA. Healthy endothelium: the scientific basis for cardiovascular health promotion and chronic disease prevention. *Vascul Pharmacol*. 2007;46:310–4.
46. McGill HC Jr., McMahan CA, Malcom GT, et al. Effects of serum lipoproteins and smoking on atherosclerosis in young men and women. The PDAY Research Group. Pathobiological Determinants of Atherosclerosis in Youth. *Arterioscler Thromb Vasc Biol*. 1997;17:95–106.
47. Libby P, Theroux P. Pathophysiology of coronary artery disease. *Circulation*. 2005;111:3481–8.
48. Miller DT, Ridker PM, Libby P, et al. Atherosclerosis: the path from genomics to therapeutics. *J Am Coll Cardiol*. 2007;49:1589–99.
49. Forrester JS, Libby P. The inflammation hypothesis and its potential relevance to statin therapy. *Am J Cardiol*. 2007;99:732–8.
50. Deleted in proof.
51. Anderson GM, Bronskill SE, Mustard CA, et al. Both clinical epidemiology and population health perspectives can define the role of health care in reducing health disparities. *J Clin Epidemiol*. 2005;58:757–62.
52. Hlatky MA. Economics and cost-effectiveness in evaluating the value of cardiovascular therapies. Role of economic models in randomized clinical trials. *Am Heart J*. 1999;137:S41–6.
53. Krumholz HM, Brindis RG, Brush JE, et al. Standards for statistical models used for public reporting of health outcomes: an American Heart Association Scientific Statement from the Quality of Care and Outcomes Research Interdisciplinary Writing Group: cosponsored by

- the Council on Epidemiology and Prevention and the Stroke Council. *Circulation*. 2006;113:456–62.
54. Russell LB, Gold MR, Siegel JE, et al. The role of cost-effectiveness analysis in health and medicine. Panel on Cost-Effectiveness in Health and Medicine. *JAMA*. 1996;276:1172–7.
55. Weinstein MC, Siegel JE, Gold MR, et al. Recommendations of the Panel on Cost-effectiveness in Health and Medicine. *JAMA*. 1996;276:1253–8.
56. Larson M, Sullivan L. Editor's note: statistical primer for cardiovascular research. *Circulation*. 2006;114:2.
57. Diamond GA, Forrester JS. Analysis of probability as an aid in the clinical diagnosis of coronary-artery disease. *N Engl J Med*. 1979;300:1350–8.
58. Pearson TA, Mensah GA, Alexander RW, et al. Markers of inflammation and cardiovascular disease: application to clinical and public health practice: a statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. *Circulation*. 2003;107:499–511.
59. Vasan RS. Biomarkers of cardiovascular disease: molecular basis and practical considerations. *Circulation*. 2006;113:2335–62.
60. Lloyd-Jones DM, Wilson PW, Larson MG, et al. Framingham risk score and prediction of lifetime risk for coronary heart disease. *Am J Cardiol*. 2004;94:20–4.
61. Fonarow GC, Heywood JT. The confounding issue of comorbid renal insufficiency. *Am J Med*. 2006;119:S17–S25.
62. Cooke GE. Pharmacogenetics of multigenic disease: heart disease as an example. *Vasc Pharmacol*. 2006;44:66–74.
63. Ali RJ, Movahed A. Use of cardiovascular medications in the elderly. *Int J Cardiol*. 2002;85:203–15.
64. Anthony M, Berg MJ. Biologic and molecular mechanisms for sex differences in pharmacokinetics, pharmacodynamics, and pharmacogenetics: part I. *J Womens Health Gen Genet*. 2002;11:601–15.
65. Antman EM. Ischemic heart disease. In: *Cardiovascular Therapeutics: A Companion to Braunwald's Heart Disease*. 3rd edition. Philadelphia, Pa: Elsevier, 2007.
66. Kullo IJ, Ding K. Mechanisms of disease: the genetic basis of coronary heart disease. *Nat Clin Pract Cardiovasc Med*. 2007;4:558–69.
67. Frezzo TM, Rubinstein WS, Dunham D, et al. The genetic family history as a risk assessment tool in internal medicine. *Genet Med*. 2003;5:84–91.
68. Qureshi N, Bethea J, Modell B, et al. Collecting genetic information in primary care: evaluating a new family history tool. *Fam Pract*. 2005;22:663–9.
69. Ginsburg GS, Donahue MP, Newby LK. Prospects for personalized cardiovascular medicine: the impact of genomics. *J Am Coll Cardiol*. 2005;46:1615–27.
70. Ouzounian M, Lee DS, Gramolini AO, et al. Predict, prevent and personalize: genomic and proteomic approaches to cardiovascular medicine. *Can J Cardiol*. 2007;23(suppl A):28A–33A.
71. Crouch MA, Gramling R. Family history of coronary heart disease: evidence-based applications. *Prim Care*. 2005;32:995–1010.
72. Hamosh A, Scott AF, Amberger JS, et al. Online Mendelian Inheritance in Man (OMIM), a knowledgebase of human genes and genetic disorders. *Nucleic Acids Res*. 2005;33:D514–7.
73. Pagon R. Internet resources in medical genetics. *Curr Protoc Hum Genet*. 2007;62:9.12.1–9.12.8.
74. Humphries SE, Ridker PM, Talmud PJ. Genetic testing for cardiovascular disease susceptibility: a useful clinical management tool or possible misinformation? *Arterioscler Thromb Vasc Biol*. 2004;24:628–36.
75. McPherson E. Genetic diagnosis and testing in clinical practice. *Clin Med Res*. 2006;4:123–9.
76. Wade CH, Wilfond BS. Ethical and clinical practice considerations for genetic counselors related to direct-to-consumer marketing of genetic tests. *Am J Med Genet C Semin Med Genet*. 2006;142:284–92.
77. Albert MA, Glynn RJ, Buring J, et al. Impact of traditional and novel risk factors on the relationship between socioeconomic status and incident cardiovascular events. *Circulation*. 2006;114:2619–26.
78. Barth J, Schumacher M, Herrmann-Lingen C. Depression as a risk factor for mortality in patients with coronary heart disease: a meta-analysis. *Psychosom Med*. 2004;66:802–13.
79. Krantz DS, Sheps DS, Carney RM, et al. Effects of mental stress in patients with coronary artery disease: evidence and clinical implications. *JAMA*. 2000;283:1800–2.
80. Rugulies R. Depression as a predictor for coronary heart disease. A review and meta-analysis. *Am J Prev Med*. 2002;23:51–61.
81. DiMatteo MR, Lepper HS, Croghan TW. Depression is a risk factor for noncompliance with medical treatment: meta-analysis of the effects of anxiety and depression on patient adherence. *Arch Intern Med*. 2000;160:2101–7.
82. Rasmussen JN, Chong A, Alter DA. Relationship between adherence to evidence-based pharmacotherapy and long-term mortality after acute myocardial infarction. *JAMA*. 2007;297:177–86.
83. Berkman LF, Blumenthal J, Burg M, et al. Effects of treating depression and low perceived social support on clinical events after myocardial infarction: the Enhancing Recovery in Coronary Heart Disease Patients (ENRICH) Randomized Trial. *JAMA*. 2003;289:3106–16.
84. Glassman AH, O'Connor CM, Califf RM, et al. Sertraline treatment of major depression in patients with acute MI or unstable angina. *JAMA*. 2002;288:701–9.
85. Lesperance F, Frasure-Smith N, Koszycki D, et al. Effects of citalopram and interpersonal psychotherapy on depression in patients with coronary artery disease: the Canadian Cardiac Randomized Evaluation of Antidepressant and Psychotherapy Efficacy (CREATE) trial. *JAMA*. 2007;297:367–79.
86. Alter DA, Chong A, Austin PC, et al. Socioeconomic status and mortality after acute myocardial infarction. *Ann Intern Med*. 2006;144:82–93.
87. Hemingway H, Marmot M. Evidence based cardiology: psychosocial factors in the aetiology and prognosis of coronary heart disease. Systematic review of prospective cohort studies. *BMJ*. 1999;318:1460–7.
88. Kanjilal S, Gregg EW, Cheng YJ, et al. Socioeconomic status and trends in disparities in 4 major risk factors for cardiovascular disease among US adults, 1971–2002. *Arch Intern Med*. 2006;166:2348–55.
89. Kivimaki M, Lawlor DA, Davey SG, et al. Socioeconomic position, co-occurrence of behavior-related risk factors, and coronary heart disease: the Finnish Public Sector study. *Am J Public Health*. 2007;97:874–9.
90. Rasmussen JN, Rasmussen S, Gislason GH, et al. Mortality after acute myocardial infarction according to income and education. *J Epidemiol Community Health*. 2006;60:351–6.
91. Schwartz AR, Gerin W, Davidson KW, et al. Toward a causal model of cardiovascular responses to stress and the development of cardiovascular disease. *Psychosom Med*. 2003;65:22–35.
92. Caulin-Glaser T, Maciejewski PK, Snow R, et al. Depressive symptoms and sex affect completion rates and clinical outcomes in cardiac rehabilitation. *Prev Cardiol*. 2007;10:15–21.
93. Sandhu S, Wiebe N, Fried LF, et al. Statins for improving renal outcomes: a meta-analysis. *J Am Soc Nephrol*. 2006;17:2006–16.
94. Shlipak MG, Fried LF, Cushman M, et al. Cardiovascular mortality risk in chronic kidney disease: comparison of traditional and novel risk factors. *JAMA*. 2005;293:1737–45.
95. Doria A, Iaccarino L, Sarzi-Puttini P, et al. Cardiac involvement in systemic lupus erythematosus. *Lupus*. 2005;14:683–6.
96. Giles JT, Post W, Blumenthal RS, et al. Therapy insight: managing cardiovascular risk in patients with rheumatoid arthritis. *Nat Clin Pract Rheumatol*. 2006;2:320–9.
97. Law MG, Friis-Moller N, El-Sadr WM, et al. The use of the Framingham equation to predict myocardial infarctions in HIV-infected patients: comparison with observed events in the D:A:D Study. *HIV Med*. 2006;7:218–30.
98. Triant VA, Lee H, Hadigan C, et al. Increased acute myocardial infarction rates and cardiovascular risk factors among patients with human immunodeficiency virus disease. *J Clin Endocrinol Metab*. 2007;92:2506–12.
99. D:A:D Study Group, Friis-Moller N, Reiss P, et al. Class of antiretroviral drugs and the risk of myocardial infarction. *N Engl J Med*. 2007;356:1723–35.
100. Ndrepepa G, Braun S, Niemoller K, et al. Prognostic value of N-terminal pro-brain natriuretic peptide in patients with chronic stable angina. *Circulation*. 2005;112:2102–7.

101. Omland T, Sabatine MS, Jablonski KA, et al. Prognostic value of B-type natriuretic peptides in patients with stable coronary artery disease: the PEACE Trial. *J Am Coll Cardiol*. 2007;50:205–14.
102. Sakai H, Tsutamoto T, Ishikawa C, et al. Direct comparison of brain natriuretic peptide (BNP) and N-terminal pro-BNP secretion and extent of coronary artery stenosis in patients with stable coronary artery disease. *Circ J*. 2007;71:499–505.
103. Lavenson GS Jr., Pantera RL, Garza RM, et al. Development and implementation of a rapid, accurate, and cost-effective protocol for national stroke prevention screening. *Am J Surg*. 2004;188:638–43.
104. Detrano R, Guerci AD, Carr JJ, et al. Coronary calcium as a predictor of coronary events in four racial or ethnic groups. *N Engl J Med*. 2008;358:1336–45.
105. Lakoski SG, Greenland P, Wong ND, et al. Coronary artery calcium scores and risk for cardiovascular events in women classified as “low risk” based on Framingham risk score: the multi-ethnic study of atherosclerosis (MESA). *Arch Intern Med*. 2007;167:2437–42.
106. Lewis RF, Abrahamowicz M, Cote R, et al. Predictive power of duplex ultrasonography in asymptomatic carotid disease. *Ann Intern Med*. 1997;127:13–20.
107. Rockman CB, Jacobowitz GR, Gagne PJ, et al. Focused screening for occult carotid artery disease: patients with known heart disease are at high risk. *J Vasc Surg*. 2004;39:44–51.
108. Ardehali R, Nasir K, Kollandavelu A, et al. Screening patients for subclinical atherosclerosis with non-contrast cardiac CT. *Atherosclerosis*. 2007;192:235–42.
109. Chien C, Feng YF, Arora RR. Advances in computed tomography-based evaluation of coronary arteries: a review of coronary artery imaging with multidetector spiral computed tomography. *Rev Cardiovasc Med*. 2007;8:53–60.
110. Dowe DA. The case in favor of screening for coronary artery disease with coronary CT angiography. *J Am Coll Radiol*. 2007;4:289–94.
111. Ehara M, Surmely JF, Kawai M, et al. Diagnostic accuracy of 64-slice computed tomography for detecting angiographically significant coronary artery stenosis in an unselected consecutive patient population: comparison with conventional invasive angiography. *Circ J*. 2006;70:564–71.
112. Nasir K, Shaw LJ, Liu ST, et al. Ethnic differences in the prognostic value of coronary artery calcification for all-cause mortality. *J Am Coll Cardiol*. 2007;50:953–60.
113. Patel MR, Hurwitz LM, Orlando L, et al. Noninvasive imaging for coronary artery disease: a technology assessment for the Medicare Coverage Advisory Commission. *Am Heart J*. 2007;153:161–74.
114. Reant P, Brunot S, Lafitte S, et al. Predictive value of noninvasive coronary angiography with multidetector computed tomography to detect significant coronary stenosis before valve surgery. *Am J Cardiol*. 2006;97:1506–10.
115. Ashton HA, Buxton MJ, Day NE, et al. The Multicentre Aneurysm Screening Study (MASS) into the effect of abdominal aortic aneurysm screening on mortality in men: a randomised controlled trial. *Lancet*. 2002;360:1531–9.
116. Lederle FA, Walker JM, Reinke DB. Selective screening for abdominal aortic aneurysms with physical examination and ultrasound. *Arch Intern Med*. 1988;148:1753–6.
117. Bendermacher BL, Teijink JA, Willigendael EM, et al. A clinical prediction model for the presence of peripheral arterial disease—the benefit of screening individuals before initiation of measurement of the ankle-brachial index: an observational study. *Vasc Med*. 2007;12:5–11.
118. Holland-Letz T, Endres HG, Biedermann S, et al. Reproducibility and reliability of the ankle-brachial index as assessed by vascular experts, family physicians and nurses. *Vasc Med*. 2007;12:105–12.
119. Wardlaw JM, Chappell FM, Best JJ, et al. Non-invasive imaging compared with intra-arterial angiography in the diagnosis of symptomatic carotid stenosis: a meta-analysis. *Lancet*. 2006;367:1503–12.
120. Einstein AJ, Henzlova MJ, Rajagopalan S. Estimating risk of cancer associated with radiation exposure from 64-slice computed tomography coronary angiography. *JAMA*. 2007;298:317–23.
121. Osterberg L, Blaschke T. Adherence to medication. *N Engl J Med*. 2005;353:487–97.
122. Chapman RH, Benner JS, Petrilla AA, et al. Predictors of adherence with antihypertensive and lipid-lowering therapy. *Arch Intern Med*. 2005;165:1147–52.
123. Eagle KA, Kline-Rogers E, Goodman SG, et al. Adherence to evidence-based therapies after discharge for acute coronary syndromes: an ongoing prospective, observational study. *Am J Med*. 2004;117:73–81.
124. Lee JK, Grace KA, Taylor AJ. Effect of a pharmacy care program on medication adherence and persistence, blood pressure, and low-density lipoprotein cholesterol: a randomized controlled trial. *JAMA*. 2006;296:2563–71.
125. Burnier M. Medication adherence and persistence as the cornerstone of effective antihypertensive therapy. *Am J Hypertens*. 2006;19:1190–6.
126. Liu J, Zhang J, Shi Y, et al. Chinese red yeast rice (*Monascus purpureus*) for primary hyperlipidemia: a meta-analysis of randomized controlled trials. *Chin Med*. 2006;1:4.
127. Jacobson TA. Beyond lipids: the role of omega-3 fatty acids from fish oil in the prevention of coronary heart disease. *Curr Atheroscler Rep*. 2007;9:145–53.
128. Appel LJ, Brands MW, Daniels SR, et al. Dietary approaches to prevent and treat hypertension: a scientific statement from the American Heart Association. *Hypertension*. 2006;47:296–308.
129. Sacks FM, Katan M. Randomized clinical trials on the effects of dietary fat and carbohydrate on plasma lipoproteins and cardiovascular disease. *Am J Med*. 2002;113(suppl 9B):13S–24S.
130. Cernea S, Hancu N, Raz I. Diet and coronary heart disease in diabetes. *Acta Diabetol*. 2003;40(suppl 2):S389–400.
131. John S, Sorokin AV, Thompson PD. Phytosterols and vascular disease. *Curr Opin Lipidol*. 2007;18:35–40.
132. Tucker KL. Dietary intake and coronary heart disease: a variety of nutrients and phytochemicals are important. *Curr Treat Options Cardiovasc Med*. 2004;6:291–302.
133. Van Itallie TB, Yang MU. Cardiac dysfunction in obese dieters: a potentially lethal complication of rapid, massive weight loss. *Am J Clin Nutr*. 1984;39:695–702.
134. Kris-Etherton PM, Lichtenstein AH, Howard BV, et al. Antioxidant vitamin supplements and cardiovascular disease. *Circulation*. 2004;110:637–41.
135. Fuchs FD, Chambless LE. Is the cardioprotective effect of alcohol real? *Alcohol*. 2007;41:399–402.
136. Tolstrup J, Gronbaek M. Alcohol and atherosclerosis: recent insights. *Curr Atheroscler Rep*. 2007;9:116–24.
137. Cannon CP, Braunwald E, McCabe CH, et al. Intensive versus moderate lipid lowering with statins after acute coronary syndromes. *N Engl J Med*. 2004;350:1495–504.
138. Nissen SE, Tuzcu EM, Schoenhagen P, et al. Effect of intensive compared with moderate lipid-lowering therapy on progression of coronary atherosclerosis: a randomized controlled trial. *JAMA*. 2004;291:1071–80.
139. Nissen SE, Nicholls SJ, Sipahi I, et al. Effect of very high-intensity statin therapy on regression of coronary atherosclerosis: the ASTEROID trial. *JAMA*. 2006;295:1556–65.
140. Davidson MH, Maki KC, Pearson TA, et al. Results of the National Cholesterol Education (NCEP) Program Evaluation Project Utilizing Novel E-Technology (NEPTUNE) II survey and implications for treatment under the recent NCEP Writing Group recommendations. *Am J Cardiol*. 2005;96:556–63.
141. Fonarow GC. Treating to goal: new strategies for initiating and optimizing lipid-lowering therapy in patients with atherosclerosis. *Vasc Med*. 2002;7:187–94.
142. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106:3143–421.
143. Grundy SM, Cleeman JI, Merz CN, et al. Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines. *Circulation*. 2004;110:227–39.
144. Caro J, O'Brien J, Hollenbeck C, et al. Economic burden and risk of cardiovascular disease and diabetes in patients with different cardiometabolic risk profiles. *Value in Health*. 2007;10:S12–20. Abstract.
145. Grundy SM, Cleeman JI, Daniels SR, et al. Diagnosis and management of the metabolic syndrome. An American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. Executive summary. *Cardiol Rev*. 2005;13:322–7.
146. Taylor AJ, Lee HJ, Sullenberger LE. The effect of 24 months of combination statin and extended-release niacin on carotid intima-

- media thickness: ARBITER 3. *Curr Med Res Opin.* 2006;22:2243–50.
147. Thom T, Haase N, Rosamond W, et al. Heart disease and stroke statistics—2006 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation.* 2006;113:e85–e151.
148. Hirsch AT, Criqui MH, Treat-Jacobson D, et al. Peripheral arterial disease detection, awareness, and treatment in primary care. *JAMA.* 2001;286:1317–24.
149. Bhatt DL, Steg PG, Ohman EM, et al. International prevalence, recognition, and treatment of cardiovascular risk factors in outpatients with atherothrombosis. *JAMA.* 2006;295:180–9.
150. Adams RJ, Chimowitz MI, Alpert JS, et al. Coronary risk evaluation in patients with transient ischemic attack and ischemic stroke: a scientific statement for healthcare professionals from the Stroke Council and the Council on Clinical Cardiology of the American Heart Association/American Stroke Association. *Stroke.* 2003;34:2310–22.
151. Ness J, Aronow WS. Prevalence of coexistence of coronary artery disease, ischemic stroke, and peripheral arterial disease in older persons, mean age 80 years, in an academic hospital-based geriatrics practice. *J Am Geriatr Soc.* 1999;47:1255–6.
152. Ridker PM, Stampfer MJ, Rifai N. Novel risk factors for systemic atherosclerosis: a comparison of C-reactive protein, fibrinogen, homocysteine, lipoprotein(a), and standard cholesterol screening as predictors of peripheral arterial disease. *JAMA.* 2001;285:2481–5.
153. Anderson JL, Adams CD, Antman EM, et al. ACC/AHA 2007 guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients With Unstable Angina/Non-ST-Elevation Myocardial Infarction). *J Am Coll Cardiol.* 2007;50:e1–157.
154. Selvin E, Erlinger TP. Prevalence of and risk factors for peripheral arterial disease in the United States: results from the National Health and Nutrition Examination Survey, 1999–2000. *Circulation.* 2004;110:738–43.
155. Antithrombotic Trialists' Collaboration. Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. *BMJ.* 2002;324:71–86.
156. Peripheral Arterial Diseases Antiplatelet Consensus Group. Antiplatelet therapy in peripheral arterial disease. Consensus statement. *Eur J Vasc Endovasc Surg.* 2003;26:1–16.
157. American Diabetes Association. Peripheral arterial disease in people with diabetes. *Diabetes Care.* 2003;26:3333–41.
158. Hirsch AT, Haskal ZJ, Hertzner NR, et al. ACC/AHA 2005 practice guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): a collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease). *J Am Coll Cardiol.* 2006;47:e1–192.
159. Proceedings of the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy: evidence-based guidelines. *Chest.* 2004;126(suppl 3):172S–696S.
160. Kearney PM, Whelton M, Reynolds K, et al. Global burden of hypertension: analysis of worldwide data. *Lancet.* 2005;365:217–23.
161. Rosamond W, Flegal K, Friday G, et al. Heart disease and stroke statistics—2007 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation.* 2007;115:e69–171.
162. Rosamond W, Flegal K, Furie K, et al. Heart disease and stroke statistics—2008 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation.* 2008;117:e25–146.
163. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet.* 2004;364:937–52.
164. Critchley J, Capewell S. Smoking cessation for the secondary prevention of coronary heart disease. *Cochrane Database Syst Rev.* 2003;CD003041.
165. Isles CG, Hole DJ, Hawthorne VM, et al. Relation between coronary risk and coronary mortality in women of the Renfrew and Paisley survey: comparison with men. *Lancet.* 1992;339:702–6.
166. Dale LC, Ebbert JO, Hays JT, et al. Treatment of nicotine dependence. *Mayo Clin Proc.* 2000;75:1311–6.
167. Fiore M, Bailey W, Cohen S. Treating tobacco use and dependence: clinical practice guideline. Rockville, Md: U.S. Department of Health and Human Services, Public Health Service, 2000.
168. U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General. The health consequences of involuntary exposure to tobacco smoke: a report of the Surgeon General 2006. Available at: <http://www.surgeongeneral.gov/library/secondhandsmoke/>. Accessed March 5, 2008.
169. Teo KK, Ounpuu S, Hawken S, et al. Tobacco use and risk of myocardial infarction in 52 countries in the INTERHEART study: a case-control study. *Lancet.* 2006;368:647–58.
170. Ebbert JO, Sood A, Hays JT, et al. Treating tobacco dependence: review of the best and latest treatment options. *J Thorac Oncol.* 2007;2:249–56.
171. Jorenby DE, Leischow SJ, Nides MA, et al. A controlled trial of sustained-release bupropion, a nicotine patch, or both for smoking cessation. *N Engl J Med.* 1999;340:685–91.
172. Jorenby DE. Smoking cessation strategies for the 21st century. *Circulation.* 2001;104:E51–E52.
173. Lancaster T, Stead L. Physician advice for smoking cessation. *Cochrane Database Syst Rev.* 2004;CD000165.
174. Miller NH, Taylor CB. Lifestyle Management for Patients With Coronary Heart Disease. Champaign, IL: Human Kinetics, 1995.
175. Silagy C, Lancaster T, Stead L, et al. Nicotine replacement therapy for smoking cessation. *Cochrane Database Syst Rev.* 2004; CD000146.
176. Smith PM, Taylor CB. Implementing an Inpatient Smoking Cessation Program. London: Taylor & Francis, 2006.
177. Prochaska JO, Velicer WF, Rossi JS, et al. Stages of change and decisional balance for 12 problem behaviors. *Health Psychol.* 1994;13:39–46.
178. Rollnick S, Mason P, Butler C. Health Behavior Change: A Guide for Practitioners. New York, NY: Churchill Livingstone, 1999.
179. Gonzales D, Rennard SI, Nides M, et al. Varenicline, an alpha4beta2 nicotinic acetylcholine receptor partial agonist, vs sustained-release bupropion and placebo for smoking cessation: a randomized controlled trial. *JAMA.* 2006;296:47–55.
180. Gourlay SG, Stead LF, Benowitz NL. Clonidine for smoking cessation. *Cochrane Database Syst Rev.* 2004;CD000058.
181. Silagy C, Lancaster T, Stead L, et al. Nicotine replacement therapy for smoking cessation. *Cochrane Database Syst Rev.* 2002; CD000146.
182. Tonstad S, Tonnesen P, Hajek P, et al. Effect of maintenance therapy with varenicline on smoking cessation: a randomized controlled trial. *JAMA.* 2006;296:64–71.
183. Wagena EJ, Knipschild P, Zeegers MP. Should nortriptyline be used as a first-line aid to help smokers quit? Results from a systematic review and meta-analysis. *Addiction.* 2005;100:317–26.
184. National Center for Healthcare Statistics. Prevalence of overweight and obesity among adults: United States, 1999–2002.
185. U.S. Preventive Services Task Force. Screening for obesity in adults: recommendations and rationale. *Ann Intern Med.* 2003;139:930–2.
186. Mullis RM, Blair SN, Aronne LJ, et al. Prevention Conference VII: obesity, a worldwide epidemic related to heart disease and stroke: group IV: prevention/treatment. *Circulation.* 2004;110:e484–8.
187. Goldstein DJ. Beneficial health effects of modest weight loss. *Int J Obes Relat Metab Disord.* 1992;16:397–415.
188. Wadden TA. Treatment of obesity by moderate and severe caloric restriction. Results of clinical research trials. *Ann Intern Med.* 1993;119:688–93.
189. Thompson PD, Buchner D, Pina IL, et al. Exercise and physical activity in the prevention and treatment of atherosclerotic cardiovascular disease: a statement from the Council on Clinical Cardiology (Subcommittee on Exercise, Rehabilitation, and Prevention) and the Council on Nutrition, Physical Activity, and Metabolism (Subcommittee on Physical Activity). *Circulation.* 2003;107:3109–16.

190. Ades PA. Cardiac rehabilitation and secondary prevention of coronary heart disease. *N Engl J Med*. 2001;345:892–902.
191. Wenger N, Froelicher E, Smith L, et al. Cardiac rehabilitation as secondary prevention. Agency for Health Care Policy and Research and National Heart, Lung, and Blood Institute. *Clin Pract Guidel Quick Ref Guide Clin*. 1995; Oct(17):1–23
192. Dalal H, Evans PH, Campbell JL. Recent developments in secondary prevention and cardiac rehabilitation after acute myocardial infarction. *BMJ*. 2004;328:693–7.
193. LaBresh KA, Ellrodt AG, Gliklich R, et al. Get with the guidelines for cardiovascular secondary prevention: pilot results. *Arch Intern Med*. 2004;164:203–9.
194. Taylor RS, Brown A, Ebrahim S, et al. Exercise-based rehabilitation for patients with coronary heart disease: systematic review and meta-analysis of randomized controlled trials. *Am J Med*. 2004;116:682–92.
195. Leon AS, Franklin BA, Costa F, et al. Cardiac rehabilitation and secondary prevention of coronary heart disease: an American Heart Association scientific statement from the Council on Clinical Cardiology (Subcommittee on Exercise, Cardiac Rehabilitation, and Prevention) and the Council on Nutrition, Physical Activity, and Metabolism (Subcommittee on Physical Activity). *Circulation*. 2005;111:369–76.
196. Franklin B, Gordon N. Contemporary Diagnosis and Management in Cardiovascular Exercise. Newtown, Pa: Handbooks in Healthcare Company; 2005.
197. Myers J, Kaykha A, George S, et al. Fitness versus physical activity patterns in predicting mortality in men. *Am J Med*. 2004;117:912–8.
198. Fletcher GF, Balady GJ, Amsterdam EA, et al. Exercise standards for testing and training: a statement for healthcare professionals from the American Heart Association. *Circulation*. 2001;104:1694–740.
199. Rodgers GP, Ayanian JZ, Balady G, et al. American College of Cardiology/American Heart Association Clinical Competence statement on stress testing: a report of the American College of Cardiology/American Heart Association/American College of Physicians—American Society of Internal Medicine Task Force on Clinical Competence. *J Am Coll Cardiol*. 2000;36:1441–53.
200. Balady GJ, Williams MA, Ades PA, et al. Core components of cardiac rehabilitation/secondary prevention programs: 2007 update: a scientific statement from the American Heart Association Exercise, Cardiac Rehabilitation, and Prevention Committee, the Council on Clinical Cardiology; the Councils on Cardiovascular Nursing, Epidemiology and Prevention, and Nutrition, Physical Activity, and Metabolism; and the American Association of Cardiovascular and Pulmonary Rehabilitation. *Circulation*. 2007;115:2675–82.
201. King ML, Williams MA, Fletcher GF, et al. Medical director responsibilities for outpatient cardiac rehabilitation/secondary prevention programs: a scientific statement from the American Heart Association/American Association for Cardiovascular and Pulmonary Rehabilitation. *Circulation*. 2005;112:3354–60.
202. Williams MA, Haskell WL, Ades PA, et al. Resistance exercise in individuals with and without cardiovascular disease: 2007 update: a scientific statement from the American Heart Association Council on Clinical Cardiology and Council on Nutrition, Physical Activity, and Metabolism. *Circulation*. 2007;116:572–84.
203. Convertino VA. Effect of orthostatic stress on exercise performance after bed rest: relation to in-hospital rehabilitation. *J Cardiac Rehabil*. 1983;3:660–3.
204. Swain DP, Franklin BA. Is there a threshold intensity for aerobic training in cardiac patients? *Med Sci Sports Exerc*. 2002;34:1071–5.
205. Dunn AL, Marcus BH, Kampert JB, et al. Comparison of lifestyle and structured interventions to increase physical activity and cardiorespiratory fitness: a randomized trial. *JAMA*. 1999;281:327–34.
206. Thompson PD, Franklin BA, Balady GJ, et al. Exercise and acute cardiovascular events placing the risks into perspective: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism and the Council on Clinical Cardiology. *Circulation*. 2007;115:2358–68.
207. Reaven G. Metabolic syndrome: pathophysiology and implications for management of cardiovascular disease. *Circulation*. 2002;106:286–8.
208. Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med*. 2002;346:393–403.
209. American Diabetes Association. Standards of medical care in diabetes—2006. *Diabetes Care*. 2006;29(suppl 1):S4–42.
210. American College of Endocrinology and American Diabetes Association consensus statement on inpatient diabetes and glycemic control. *Diabetes Care*. 2006;29:1955–62.
211. Douglas PS, Blumenthal RS. President's page: prevention's place in cardiology: what the future holds. *J Am Coll Cardiol*. 2005;46:1777–8.
212. Gluckman TJ, Baranowski B, Ashen MD, et al. A practical and evidence-based approach to cardiovascular disease risk reduction. *Arch Intern Med*. 2004;164:1490–500.
213. Gluckman TJ, Sachdev M, Schulman SP, et al. A simplified approach to the management of non-ST-segment elevation acute coronary syndromes. *JAMA*. 2005;293:349–57.
214. Fonarow GC, Abraham WT, Albert NM, et al. Association between performance measures and clinical outcomes for patients hospitalized with heart failure. *JAMA*. 2007;297:61–70.
215. Vasaiwala S, Nolan E, Ramanath VS, et al. A quality guarantee in acute coronary syndromes: the American College of Cardiology's Guidelines Applied in Practice program taken real-time. *Am Heart J*. 2007;153:16–21.
216. Eagle KA, Koelling TM, Montoye CK. Primer: implementation of guideline-based programs for coronary care. *Nat Clin Pract Cardiovasc Med*. 2006;3:163–71.
217. Jani SM, Montoye C, Mehta R, et al. Sex differences in the application of evidence-based therapies for the treatment of acute myocardial infarction: the American College of Cardiology's Guidelines Applied in Practice projects in Michigan. *Arch Intern Med*. 2006;166:1164–70.
218. Montoye CK, Eagle KA. An organizational framework for the AMI ACC-GAP Project. *J Am Coll Cardiol*. 2005;46:1–29.
219. Blumenthal RS, Gluckman TJ. New insights into the relationship between myocardial perfusion and lipid-lowering. *J Am Coll Cardiol*. 2003;42:611–3.
220. Parikh P, McDaniel MC, Ashen MD, et al. Diets and cardiovascular disease: an evidence-based assessment. *J Am Coll Cardiol*. 2005;45:1379–87.
221. Wilson PW, Smith SC Jr., Blumenthal RS, et al. 34th Bethesda Conference: task force #4—how do we select patients for atherosclerosis imaging? *J Am Coll Cardiol*. 2003;41:1898–906.
222. Block RC, Pearson TA. Organizing services for cardiovascular prevention. *Curr Treat Options Cardiovasc Med*. 2007;9:278–86.
223. Epping-Jordan JE, Pruitt SD, Bengoa R, et al. Improving the quality of health care for chronic conditions. *Qual Saf Health Care*. 2004;13:299–305.
224. Kirsh S, Watts S, Pascuzzi K, et al. Shared medical appointments based on the chronic care model: a quality improvement project to address the challenges of patients with diabetes with high cardiovascular risk. *Qual Saf Health Care*. 2007;16:349–53.

---

**Key Words:** ACCF/AHA Competence and Training Statements ■ competency ■ prevention ■ cardiovascular ■ training ■ vascular ■ cardiac ■ cardiac rehabilitation.

## APPENDIX 1. AUTHOR RELATIONSHIPS WITH INDUSTRY AND OTHER ENTITIES—ACCF/AHA/ACP 2009 COMPETENCE AND TRAINING STATEMENT: A CURRICULUM ON PREVENTION OF CARDIOVASCULAR DISEASE

Committee Member	Consultant	Speaker	Ownership/ Partnership/ Principal	Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness
Dr. C. Noel Bairey Merz ( <i>Chair</i> )	<ul style="list-style-type: none"> <li>• Access Communications Inc. (lecture)</li> <li>• Adventist Health (expert witness consulting)</li> <li>• American College of Physicians (lecture)</li> <li>• American Psychosomatic Society (award lecture)</li> <li>• AstraZeneca (lecture)</li> <li>• Bayer (consulting; advisory board; lecture Argentine Cardiology Congress)</li> <li>• Berlin Heart Institute (lecture)</li> <li>• CDC (grant reviewing)</li> <li>• CMP Media (consulting)</li> <li>• Genesis Associates (consulting)</li> <li>• Hospicom (advisory boards)</li> <li>• Hunt and Associates (expert witness consulting)</li> <li>• KOS (lectures)</li> <li>• Madera Medical Society (lectures)</li> <li>• Mayo Clinic (lecture)</li> <li>• MedReviews/CVT (lectures)</li> <li>• Merck &amp; Co. (lectures)</li> <li>• Navigant</li> <li>• Novartis (advisory boards at ACC and AHA Scientific Sessions)</li> <li>• NHLBI (council)</li> <li>• New York University (lecture)</li> <li>• Pfizer (consulting)</li> <li>• QD Healthcare Group (consulting)</li> <li>• Remillard and Associates (expert witness consulting)</li> <li>• Rodale Press (health advisor)</li> <li>• Stanford University (lectures)</li> <li>• Thomson Physician's World (satellite lecture, China)</li> <li>• Thornton (lecture)</li> <li>• University of California, San Francisco (invited lecture at ACC)</li> <li>• University of Michigan (invited named lectureship)</li> <li>• University of New Mexico (lectures)</li> <li>• University of Pittsburgh (NHLBI DSMB chair)</li> <li>• Wolfson Communications (consulting)</li> </ul>	None	<ul style="list-style-type: none"> <li>• ATS Medical</li> <li>• Boston Scientific</li> <li>• Eli Lilly*</li> <li>• Johnson &amp; Johnson*</li> <li>• Medtronic*</li> <li>• Teva Pharmaceuticals</li> </ul>	<ul style="list-style-type: none"> <li>• CV Therapeutics</li> <li>• Parke-Davis/Pfizer</li> </ul>	None	None
Dr. Mark J. Alberts	<ul style="list-style-type: none"> <li>• AGA Medical</li> <li>• AstraZeneca</li> <li>• Bayer</li> <li>• Boehringer Ingelheim</li> <li>• Bristol-Myers Squibb</li> <li>• diaDexus</li> <li>• Eli Lilly &amp; Co.</li> <li>• Genentech</li> <li>• KOS</li> <li>• Medicines Company</li> <li>• Merck</li> <li>• Novo Nordisk</li> <li>• PDL BioPharma, Inc.</li> <li>• Pfizer*</li> <li>• Sanofi</li> <li>• Schering-Plough</li> <li>• Tap Pharmaceuticals-DSMB</li> </ul>	<ul style="list-style-type: none"> <li>• AstraZeneca</li> <li>• Boehringer Ingelheim</li> <li>• Bristol-Myers Squibb</li> <li>• diaDexus</li> <li>• Genentech</li> <li>• Medicines Company</li> <li>• Novo Nordisk</li> <li>• PDL BioPharma, Inc.</li> <li>• Sanofi</li> </ul>	None	<ul style="list-style-type: none"> <li>• AGA Medical</li> <li>• AstraZeneca</li> <li>• Boehringer Ingelheim</li> <li>• Bristol-Myers Squibb</li> <li>• Novo Nordisk</li> <li>• Photo Thera</li> <li>• Sanofi</li> <li>• Schering-Plough</li> </ul>	None	None





Committee Member	Consultant	Speaker	Ownership/ Partnership/ Principal	Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness
Dr. Vivian Fonseca	<ul style="list-style-type: none"> <li>• Daiichi Sankyo</li> <li>• Eli Lilly</li> <li>• GlaxoSmithKline</li> <li>• Novartis</li> <li>• Novo Nordisk</li> <li>• Pfizer</li> <li>• Sanofi-Aventis</li> <li>• Takeda</li> </ul>	<ul style="list-style-type: none"> <li>• Daiichi Sankyo</li> <li>• Eli Lilly</li> <li>• GlaxoSmithKline</li> <li>• Novartis</li> <li>• Novo Nordisk</li> <li>• Pfizer</li> <li>• Sanofi-Aventis</li> <li>• Takeda</li> </ul>	None	<ul style="list-style-type: none"> <li>• American Diabetes Association</li> <li>• AstraZeneca</li> <li>• Daiichi Sankyo</li> <li>• Eli Lilly</li> <li>• GlaxoSmithKline</li> <li>• National Institutes of Health</li> <li>• Novartis</li> <li>• Pfizer</li> <li>• Sanofi-Aventis</li> <li>• Takeda</li> </ul>	None	None
Dr. Barry A. Franklin	None	None	None	None	None	None
Dr. Patrick E. McBride	<ul style="list-style-type: none"> <li>• Johnson &amp; Johnson</li> <li>• Reliant</li> </ul>	None	None	None	None	None
Dr. George A. Mensah	None	None	None	None	None	None
Dr. Geno J. Merli	<ul style="list-style-type: none"> <li>• Bacchus</li> <li>• Bayer</li> <li>• Bristol-Myers Squibb</li> <li>• Sanofi-Aventis</li> </ul>	<ul style="list-style-type: none"> <li>• Sanofi-Aventis</li> </ul>	None	<ul style="list-style-type: none"> <li>• Bayer</li> <li>• Bristol-Myers Squibb</li> <li>• Boehringer Ingelheim</li> <li>• Sanofi-Aventis</li> </ul>	None	None
Dr. Patrick T. O'Gara	None	None	None	None	None	None
Dr. Paul D. Thompson	<ul style="list-style-type: none"> <li>• Abbott</li> <li>• AstraZeneca</li> <li>• KOS</li> <li>• Pfizer</li> <li>• Reliant</li> <li>• Schering-Plough</li> </ul>	<ul style="list-style-type: none"> <li>• AstraZeneca</li> <li>• KOS</li> <li>• Merck</li> <li>• Pfizer</li> <li>• Schering-Plough</li> </ul>	<ul style="list-style-type: none"> <li>• CryoLife</li> <li>• Illumina</li> <li>• Merck</li> <li>• Pfizer</li> <li>• Schering-Plough</li> <li>• ZOLL</li> </ul>	<ul style="list-style-type: none"> <li>• AstraZeneca</li> <li>• Merck</li> <li>• Pfizer</li> </ul>	None	None
Dr. James A. Underberg	<ul style="list-style-type: none"> <li>• Liposcience Inc.*</li> </ul>	<ul style="list-style-type: none"> <li>• Abbott</li> <li>• AstraZeneca</li> <li>• Daichii-Sankyo*</li> <li>• Forest</li> <li>• GlaxoSmithKline</li> <li>• KOS</li> <li>• Merck/Schering-Plough</li> <li>• Novartis</li> <li>• Pfizer</li> <li>• Reliant</li> </ul>	None	<ul style="list-style-type: none"> <li>• AstraZeneca</li> <li>• Bayer</li> <li>• GlaxoSmithKline*</li> <li>• Merck</li> <li>• Novartis*</li> </ul>	None	None

This table represents the relevant relationships of committee members with industry and other entities that were disclosed orally at the initial writing committee meeting and updated in conjunction with all meetings and conference calls of the writing committee during the document development process. It does not necessarily reflect relationships with industry at the time of publication. A person is deemed to have a significant interest in a business if the interest represents ownership of 5% or more of the voting stock or share of the business entity, or ownership of \$10 000 or more of the fair market value of the business entity; or if funds received by the person from the business entity exceed 5% of the person's gross income for the previous year. A relationship is considered to be modest if it is less than significant under the preceding definition. Relationships noted in this table are modest unless otherwise noted. \*Significant (greater than \$10 000) relationship. ACC indicates American College of Cardiology; AHA, American Heart Association; CDC, Centers for Disease Control and Prevention; DSMB, Data Safety Monitoring Board; and NHLBI, National Heart, Lung, and Blood Institute.



Peer Reviewer	Representation	Consultant	Speaker	Ownership/ Partnership/ Principal	Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness
Dr. George L. Bakris	Organizational Reviewer— American Society of Hypertension	<ul style="list-style-type: none"> <li>• Abbott Laboratories</li> <li>• Boehringer Ingelheim Corp.</li> <li>• Bristol-Myers Squibb/SanofiPharma</li> <li>• Daiichi/Sankyo, Inc.</li> <li>• Forest Laboratories</li> <li>• Gilead</li> <li>• GlaxoSmithKline</li> <li>• Merck &amp; Co., Inc.</li> <li>• Novartis Pharma.</li> <li>• Walgreens</li> </ul>	<ul style="list-style-type: none"> <li>• Abbott Laboratories</li> <li>• Boehringer Ingelheim Corp.</li> <li>• Bristol-Myers Squibb/SanofiPharma</li> <li>• Daiichi/Sankyo, Inc.</li> <li>• Forest Laboratories</li> <li>• Gilead</li> <li>• GlaxoSmithKline</li> <li>• Merck &amp; Co., Inc.</li> <li>• Novartis Pharma.</li> <li>• Walgreens</li> </ul>	None	<ul style="list-style-type: none"> <li>• GlaxoSmithKline*</li> <li>• Forest Laboratories*</li> <li>• National Institutes of Health (NIDDK/NHLBI)*</li> </ul>	None	None
Dr. Michael S. Lauer	Organizational Reviewer— National Heart, Lung, and Blood Institute	None	None	None	None	None	None
Ms. Janet B. Long	Organizational—National Lipid Association	None	• AstraZeneca	None	None	None	None
Dr. Peter Wilson	Organizational—National Lipid Association	None	None	None	<ul style="list-style-type: none"> <li>• GlaxoSmithKline*</li> <li>• Sanofi-Aventis*</li> </ul>	None	None
Dr. Lynne T. Braun	Organizational—Preventive Cardiovascular Nurses Association	<ul style="list-style-type: none"> <li>• AstraZeneca</li> <li>• diaDexus</li> </ul>	None	None	None	None	None
Ms. Joanna Sikkema	Organizational—Preventive Cardiovascular Nurses Association	None	None	None	None	None	None
Dr. Vera Bittner	Content Reviewer—ACCF Prevention Committee	<ul style="list-style-type: none"> <li>• Abbott Laboratories</li> <li>• CV Therapeutics, Inc.</li> <li>• Pfizer, Inc.</li> <li>• Reliant Pharma., LLP</li> </ul>	None	None	<ul style="list-style-type: none"> <li>• Atherogenics*</li> <li>• CV Therapeutics, Inc.*</li> <li>• Merck Research Labs*</li> <li>• NIH/Abbott*</li> <li>• Pfizer, Inc.*</li> </ul>	None	None
Dr. Gregg C. Fonarow	Content Reviewer—ACCF Prevention Committee	<ul style="list-style-type: none"> <li>• AstraZeneca</li> <li>• Bristol-Myers Squibb</li> <li>• GlaxoSmithKline*</li> <li>• Merck/Schering-Plough*</li> <li>• Novartis Pharma*</li> <li>• Pfizer, Inc.*</li> </ul>	None	None	<ul style="list-style-type: none"> <li>• GlaxoSmithKline*</li> <li>• Pfizer, Inc.*</li> </ul>	None	None
Dr. Pamela B. Morris	Content Reviewer—ACCF Prevention Committee	• Actelion†	None	None	None	None	None
Dr. Ileana L. Piña	Content Reviewer—ACCF/ AHA/ACP Task Force on Competence and Training	• GlaxoSmithKline	<ul style="list-style-type: none"> <li>• AstraZeneca</li> <li>• Novartis</li> </ul>	None	None	None	None
Dr. Howard H. Weitz	Content Reviewer—ACCF/ AHA/ACP Task Force on Competence and Training	None	None	None	None	None	None

This table represents the relevant relationships with industry and other entities that were disclosed at the time of peer review. It does not necessarily reflect relationships with industry at the time of publication. A person is deemed to have a significant interest in a business if the interest represents ownership of 5% or more of the voting stock or share of the business entity, or ownership of \$10 000 or more of the fair market value of the business entity; or if funds received by the person from the business entity exceed 5% of the person's gross income for the previous year. A relationship is considered to be modest if it is less than significant under the preceding definition. Relationships noted in this table are modest unless otherwise noted. \*Significant (greater than \$10 000) relationship. ACCF indicates American College of Cardiology Foundation; NHLBI, National Heart, Lung, and Blood Institute; and NIDDK, National Institute of Diabetes and Digestive Kidney Diseases.