ACE-> AMERICAN COUNCIL ON EXERCISE Medical Exercise Specialist Manual

Chapter Preview Medical Exercise Specialist Study Program

ACE

Thank you for your interest in the ACE Medical Exercise Specialist Certification! You've taken the first step toward a new career where you'll work as part of your client's healthcare team, helping them prevent or effectively manage chronic disease.

Enclosed is a free chapter preview from the ACE Medical Exercise Specialist Manual. With the ACE Medical Exercise Specialist Study Program, you'll receive the highest quality education, anchored in evidence-based exercise science. You'll gain the knowledge and skill to collaborate with the healthcare community to help individuals coping with one or more diseases or disorders, or following rehabilitation to reach their health and fitness goals. This in-depth study program for experienced exercise professionals will deliver everything you need to prepare for the ACE Medical Exercise Specialist Exam.

Whether you're looking to become a Medical Exercise Specialist—or you're part of an institution, health club or group and want to get your staff ACE Certified—we're here to help! Every member of our team is ACE Certified and excited to help you move closer to getting certified. Call us at the number below and get started today!

We hope to hear from you soon!



ACE Certification Experts

888.519.9841 Monday-Friday, 7:00 a.m. - 4:00 p.m. (PT)





AMERICAN COUNCIL ON EXERCISE

Medical Exercise Specialist Manual



The Definitive Resource for Health and Exercise Professionals Working with Special Populations

ACE® MEDICAL EXERCISE SPECIALIST MANUAL

THE DEFINITIVE RESOURCE FOR HEALTH AND EXERCISE PROFESSIONALS WORKING WITH SPECIAL POPULATIONS

AMERICAN COUNCIL ON EXERCISE®

EDITORS JAMES S. SKINNER, PH.D., FACSM CEDRIC X. BRYANT, PH.D., FACSM SABRENA JO, M.S. DANIEL J. GREEN



Library of Congress Catalog Card Number: 2015932186

ISBN 9781890720865 Copyright © 2021, 2015 American Council on Exercise (ACE) Printed in the United States of America

All rights reserved. Except for use in a review, the reproduction or utilization of this work in any form or by any electronic, mechanical, or other means, now known or hereafter invented, including xerography, photocopying, and recording, and in any information retrieval system, is forbidden without the written permission of the American Council on Exercise.

ACE, ACE IFT, ACE Integrated Fitness Training, and American Council on Exercise are registered trademarks of the American Council on Exercise. Other product names used herein are for identification purpose only and may be trademarks of their respective companies.

CDEF

Distributed by: American Council on Exercise 4851 Paramount Drive San Diego, CA 92123 (858) 576-6500 (858) 576-6564 FAX ACEfitness.org

Project Editor: Daniel J. Green
Technical Editors: James S. Skinner, Ph.D., FACSM, Cedric X. Bryant, Ph.D., FACSM, & Sabrena Jo, M.S.
Creative Direction: Ian Jensen
Art Direction & Cover Design: Karen McGuire
Internal Reviewers: Chris Gagliardi, B.S., & Belinda Thompson, B.S.
Production: Nancy Garcia
Photography: Dennis Covey, Rob Andrew, & Matt Gossman
Anatomical Illustrations: James Staunton
Index: Kathi Unger
Exercise models: Beth Baxter, Dana Cobb, Kris Fukuda, Tamra Herb, Aliya Lawson, Monika Lucs, Michael Marsh, Anthony Padilla, Jenn Samore, Jay Simon, Danielle Vojta, Renee West, & Richard Zuniga

Acknowledgments:

Thanks to the entire American Council on Exercise staff for their support and guidance through the process of creating this manual.

A special thanks to Rehab United for allowing us to photograph on location at their Sorrento Valley facility.

NOTICE

The fitness industry is ever-changing. As new research and clinical experience broaden our knowledge, changes in programming and standards are required. The authors and the publisher of this work have checked with sources believed to be reliable in their efforts to provide information that is complete and generally in accord with the standards accepted at the time of publication. However, in view of the possibility of human error or changes in industry standards, neither the authors nor the publisher nor any other party who has been involved in the preparation or publication of this work warrants that the information contained herein is in every respect accurate or complete, and they are not responsible for any errors or omissions or the results obtained from the use of such information. Readers are encouraged to confirm the information contained herein with other sources.

DISCLAIMER

Moving forward, ACE will use "they" and "their" in place of "he/she" and "his/her." This change eliminates gender biases associated with these pronouns and is more inclusive of all individuals across the gender spectrum. Note that all ACE content moving forward will reflect this update, and previous content will be updated as needed. It is ACE's goal to share our mission to Get People Moving with all people, regardless of race, gender, sexual orientation, physical or intellectual abilities, religious beliefs, ethnic background, or socioeconomic status.

MISSION

Get People Moving.

P21-007

TABLE OF CONTENTS

FOREWORD																	 	 			 	 		 	vii
INTRODUCTION																	 	 			 	 		 	ix
STUDYING FOR	THE	ACE	ME	DIC	AL	EX	ER	CIS	SE	SF	PE(CIA	LIS	T	EXA	M		 		 	 	 		 	xi

PART I: INTRODUCTION

 CHAPTER 1 Role and Scope of Practice for the Certified Medical Exercise Specialist Kelly Spivey
 2

 CHAPTER 2 Applying the ACE Integrated Fitness Training® Model in the Medical Exercise Setting Todd Galati . 20
 CHAPTER 3 Working With Clients With Health Challenges Nancey Trevanian Tsai

PART II: LEADERSHIP AND IMPLEMENTATION

CHAPTER 4 Behavioral Change	Tracie Rogers	94
CHAPTER 5 Communication Strat	tegies Barbara A. Brehm1	18
CHAPTER 6 Professional Relation	ships and Business Strategies Lisa Coors1	42

PART III: MAJOR CARDIOVASCULAR AND PULMONARY DISEASES AND DISORDERS

CHAPTER 7 Coronary Heart Disease Ralph La Forge	168
CHAPTER 8 Blood Lipid Disorders Ralph La Forge	208
CHAPTER 9 Hypertension W. Larry Kenney & Lacy M. Alexander	<u>244</u>
CHAPTER 10 Pulmonary Disease: Asthma and Chronic Obstructive Pulmonary Disease Natalie Digate Muth	276

PART IV: METABOLIC DISEASES AND DISORDERS

CHAPTER 11 Overweight and Obesity	Len Kravitz
CHAPTER 12 The Metabolic Syndrome	Barry A. Franklin, Wendy M. Miller, & Peter A. McCullough
CHAPTER 13 Diabetes Mellitus Larr	<i>y S. Verity</i>

PART V: MUSCULOSKELETAL DISORDERS

CHAPTER 14 Posture and Movement Fabio Comana & Chris McGrath	04
CHAPTER 15 Balance and Gait Michol Dalcourt & Fabio Comana	78
CHAPTER 16 Arthritis John G. Aronen & Kent A. Lorenz	26
CHAPTER 17 Osteoporosis and Osteopenia Kara A. Witzke	56
CHAPTER 18 Musculoskeletal Injuries of the Lower Extremity Scott Cheatham	86
CHAPTER 19 Musculoskeletal Injuries of the Upper Extremity Michael Levinson	32
CHAPTER 20 Low-back Pain Jennifer Solomon	60

PART VI: PERINATAL CONSIDERATIONS

CHAPTER 21 Prenatal and Postpartum Exercise Sabrena Jo	1
APPENDIX A ACE Code of Ethics 728 APPENDIX B Exam Content Outline 734	
APPENDIX C Nutrition for Health and Fitness Natalie Digate Muth744	1
GLOSSARY	



ABOUT THE AUTHOR

Ralph La Forge, M.S., is a physiologist and Accreditation Council on Clinical Lipidology-certified clinical lipid specialist and former managing director of the Cholesterol Disorder Physician Education Program at Duke University Medical Center, Endocrine Division in Durham, North Carolina. He was also managing director of preventive medicine and cardiac rehabilitation at Sharp Health Care in San Diego, where he also taught applied exercise physiology at the University of California at San Diego. Prior to that, La Forge was director of preventive cardiology and cardiac rehabilitation at the Lovelace Clinic in Albuquerque, New Mexico. He has helped more than 300 medical staff groups throughout North America organize and operate lipid disorder clinics and diabetes- and heartdisease-prevention programs. La Forge is the immediate past president of the Southeast Lipid Association and a clinical consultant to numerous healthcare institutions including the U.S. Indian Health Service Division of Diabetes Treatment and Prevention. He has published over 200 papers on clinical exercise science, lipidology, and preventive endocrinology.

IN THIS CHAPTER

EXERCISE TRAINING RECOMMENDATIONS FOR CLIENTS WITH STABLE CHD

APPROPRIATE PROGRAM CANDIDATES AND STABLE CHD

> FITNESS TESTING CHD PATIENTS

CONTRAINDICATIONS TO **EXERCISE TRAINING**

> **EXERCISE TRAINING** SUPERVISION CONSIDERATIONS

OVERALL WEEKLY EXERCISE ENERGY-EXPENDITURE GOALS

EXERCISE INTENSITY

EXERCISE FREQUENCY AND DURATION

> MODE OF EXERCISE FOR CHD

INTERVAL/INTERMITTENT AEROBIC EXERCISE TRAINING

RESISTANCE TRAINING

MINDFUL EXERCISE AND THE CHD CLIENT

EXERCISE GUIDELINES SUMMARY FOR CLIENTS WITH STABLE CHD

CASE STUDY

SUMMARY

HEART

HIGH-RISK PRIMARY **PREVENTION AND** STABLE CHD

EPIDEMIOLOGY

OVERVIEW

ANGINA PECTORIS

CARDIAC DYSRHYTHMIAS

MYOCARDIAL INFARCTION

HEART FAILURE

DIAGNOSTIC TESTING AND CRITERIA

ELECTROCARDIOGRAM

ECG EXERCISE TESTING

RADIONUCLIDE STRESS TEST

STRESS ECHOCARDIOGRAPHY

CORONARY ANGIOGRAPHY (CARDIAC CATHETERIZATION)

> VASCULAR IMAGING **TECHNIQUES**

CORONARY CALCIUM SCORING

TREATMENT OPTIONS

NUTRITIONAL **CONSIDERATIONS**

CARDIOVASCULAR DISEASE PREVENTION

CORONARY HEART DISEASE

CONGESTIVE HEART FAILURE

EATING PLANS TO SUPPORT OPTIMAL HEART HEALTH

DISEASE THE ROLE OF THE CMES

CORONARY

By Ralph La Forge

ADULTS WITH STABLE AND WELL-MANAGED coronary heart disease (CHD) represent a key opportunity for competent and experienced personal trainers or ACE[®] Certified Medical Exercise Specialists (CMES). CHD (also called coronary disease, coronary

artery disease, ischemic heart disease, and atherosclerotic heart disease) is a narrowing of the small blood vessels that supply blood and oxygen to the heart.

CHD is an important subset of the broader category of **cardiovascular disease (CVD)**, which refers to any disease that affects the cardiovascular system, principally cardiac disease, vascular diseases of the brain and kidney, and **peripheral arterial disease**. Most people with CHD can feel healthy for years before they start experiencing symptoms. The most common symptom is a **myocardial infarction (MI**) (also called a heart attack). If CHD is not treated, some of the plaques in the coronary arteries can break away and block the blood flow to the heart. Coronary disease is the most common cause of sudden death. It is also the most common cause of death in people over 65 years old. Men are 10 times more likely to get coronary disease than women [Centers for Disease Control and Prevention (CDC), 2013].

The purpose of this chapter is to briefly acquaint the qualified CMES with CHD, describe its process, and provide appropriate exercise training recommendations. Hypertension, diabetes, obesity, and the metabolic syndrome, all of which impact the CHD process, are discussed elsewhere in this text. The CMES is strongly encouraged to partner the information in this chapter with ACSM's Guidelines for Exercise Testing and Prescription (11th edition) [American College of Sports Medicine (ACSM), 2022] and the American Association of Cardiovascular and Pulmonary Rehabilitation's (AACVPR) Guidelines for Cardiac Rehabilitation Programs (6th edition) (AACVPR, 2021). Both of these texts are good resources for the CMES who wishes to work with individuals with stable CHD. There is remarkable agreement between these and other guidelines (e.g., ACCF/AHA/ACP/AATS/PCNA/SCAI/STS, 2012), in terms of the recommended quantity and quality of exercise for those with CHD, although there are some minor differences. This chapter represents a diligent effort to maintain consistency to harmonize recommendations and eliminate discrepancies. Some recommendations from earlier guidelines have been updated as warranted by new evidence or a better understanding of earlier evidence. The CMES should see this chapter as a current synthesis of these guidelines, particularly those from ACSM and AACVPR.

THE ROLE OF THE CMES

Fundamentally, the role of the CMES who wishes to work with individuals with CHD is to work only with those clients who have stable CHD and are at low risk for exercise-related cardiovascular complications. The determination of stable CHD should only be made by the client's referring physician. Furthermore, the principal role of the CMES in this context is to design and allocate appropriate and

LEARNING OBJECTIVES:

- Explain the role of the CMES in the treatment of coronary heart disease (CHD).
- Describe the major forms of CHD (i.e., angina pectoris, cardiac dysrhythmias, myocardial infarction, and heart failure).
- Identify testing procedures and diagnostic criteria for the diagnosis of CHD.
- Describe how nutritional intake and physical activity influence a person's risk for, and management of, CHD.
- Identify treatment strategies for CHD, including medications and nutritional approaches.
- Explain how each major type of CHD affects a client's ability to perform physical activity and exercise.
- Design and implement appropriate exercise programs for clients with CHD, taking into account adherence to the clients' physicians' recommendations and limitations.

safe levels of physical activity to improve the client's functionality, favorably modify CHD risk factors, and further improve the function of the heart. It is strongly recommended that the clients with whom the CMES intends to work have successfully completed early phase cardiac rehabilitation (essentially phases I and II) by a formalized outpatient cardiac rehabilitation program when these programs are locally available. Fewer than 20% of eligible patients receive formalized cardiac rehabilitation exercise training, which would drastically narrow the population of individuals who could work with a CMES. Although there are no universally accepted guidelines for exercise specialists who work outside of the scope of formal cardiac rehabilitation programs, it is logical that these exercise professionals follow the same published standards of care (i.e., AACVPR and ACSM) as those working within cardiac rehabilitation programs. In cases where the client cannot participate in cardiac rehabilitation for various reasons, including program location and accessibility or work or personal schedules, the CMES can train the client with an appropriate physician referral and within the guidelines discussed in this chapter.

High-risk Primary Prevention and Stable CHD

An individual in need of a supervised exercise program may not have diagnosed CHD, but instead may merely be interested in preventing heart disease (primary prevention). Candidates for this program may also include those with multiple risk factors for CHD, but may never have had a cardiac event (secondary prevention). Those with documented CHD or unstable CHD are most appropriate for formalized and supervised cardiac rehabilitation. Ideally, the CHD client would have completed early phase cardiac rehabilitation prior to working with the CMES. An experienced CMES who wishes to work with CHD clients should work only with those who are under a physician's care and who have stable coronary disease.

For purposes of the CMES's **scope of practice**, stable CHD means that the individual's disease process is well managed [i.e., they do not have irregular, unpredictable symptoms, **unstable angina**, **heart failure (HF)**, or malignant ventricular **arrhythmias**]. Stable also means that the individual is currently under the care of a physician and on appropriate medical therapy for their level of CHD. The CMES is not expected to discriminate between stable and unstable CHD, but should rely on the client's personal physician's clinical evaluation and judgment—a physician who is currently caring for the client's disease. This physician can be a cardiologist, internist, or, in some cases, a primary care physician such as a family practitioner. One means of confirming stable CHD is periodic exercise electrocardiographic stress testing [exercise **electrocardiogram (ECG)**] by a physician.

This chapter presents several tables illustrating and describing specific cautions and riskstratification measures. Some of these overlap in intention, but it is imperative that the CMES keep in mind that CHD patients have a higher probability of cardiac symptoms and recurrent cardiovascular events (e.g., MI or **acute coronary syndrome**) and clearly require a more conservative approach to exercise training. This in no way should compromise the stable CHD patient's eventual ability to safely engage in higher levels of physical activity commensurate with those recommended for apparently healthy adults.

EPIDEMIOLOGY

In 2010, one in three deaths in American adults was caused by CVD, or approximately 2,150 deaths each day [American Heart Association (AHA), 2014]. To put these numbers in perspective, CVD took the life of one American every 40 seconds. However, the AHA (2014) report does provide some good news. From 2000 to 2010, the number of deaths attributable to CVD declined 31% and the number of CVD deaths per year declined 16.7%. In 2010, the number of deaths attributed to cardiovascular causes was 235.5 per 100,000 individuals. African-American males fared the worst, with 369.2 cardiovascular deaths per 100,000 individuals. The prevalence of CHD was greatest among persons aged \geq 65 years (19.8%),

followed by those aged 45–64 years (7.1%) and those aged 18–44 years (1.2%). CHD prevalence was greater among men (7.8%) than women (4.6%) (AHA, 2014). The decline in the mortality rate suggests that more persons are living with CHD, which should result in an increase in the prevalence of CHD. However, the decline in prevalence in this report was affected not only by CHD mortality but also by CHD incidence, which is decreased by the prevention and control of CHD risk factors, particularly a fall in **Iow-density lipoprotein (LDL)**, with a greater percentage of adults taking statin drugs. Given that CHD mortality is declining, the observed decline in prevalence of CHD in this study suggests that CHD incidence also has declined. However, CHD remains the number-one killer of American adults. Every year approximately 715,000 Americans have a heart attack. Of these, 525,000 are a first heart attack and 190,000 happen in people who have already had a heart attack (CDC, 2013). These statistics are sobering and point to the likelihood that most health and exercise professionals will work with clients who suffer from a heart-related ailment.

OVERVIEW

CHD is the end result of the accumulation of lipid-rich plaques within the walls of the arteries that supply the myocardium (the muscle of the heart). CHD results from the development of atherosclerosis in the coronary arteries. Atherosclerosis is a disease affecting both large and small arterial blood vessels. It is a chronic inflammatory response in the walls of arteries, in large part due to the deposition of lipoproteins (plasma proteins that carry **cholesterol** and **triglycerides**). Atherosclerosis is essentially caused by the formation of multiple plaques within the arteries. Today, atherosclerosis is seen not as a disease of the lumen of the artery, but a disease of the vessel wall. Atherogenesis is the process of the development of these plaques, which involves the infiltration, retention, and oxidation of LDL cholesterol in the arterial intima, inflammation, development of fatty streaks, and the calcification of atherosclerotic plaques. Under normal circumstances, the vascular endothelium does not bind leukocytes (white blood cells) well. However, injury to the endothelium (innermost layer of the artery wall) causes inflammation that results in the expression of adhesion molecules that facilitate atherosclerosis. It is now understood that an acute coronary event (e.g., MI) is more often caused by rupture of a complex vulnerable atherosclerotic plaque than by a gradual closure of the coronary blood vessel (Figure 7-1). The vulnerable plaque is essentially characterized by adhesions with thin fibrous caps that predispose the plaque to rupture. Rupture of the plaque releases numerous thrombotic substances into the blood that usually stimulate a rapid sequence of events that results in a clot or coronary thrombosis. The formation, progression, and rupture of the vulnerable plaque are viewed as a process related directly to inflammation.

In recent years, **vascular inflammation** in atherogenesis and CHD has gained considerable support for its role in accelerating arterial narrowing and thrombosis. Experimental work has elucidated molecular and cellular pathways of vascular inflammation that promote atherosclerosis (Libby, 2012). It is now believed that systemic and local inflammatory events mediate all phases of plaque development, progression, and degeneration. Inflammatory **cytokines** [e.g., tumor necrosis factor-alpha, interleukin (IL)-6, and IL-18] are thought to accelerate the rate of atherogenesis even in the absence of traditional CHD risk factors. Inflammation also plays an important role in the recruitment of leukocytes at the endothelium to the atherosclerotic plaque rupture, causing the clinical symptoms of CHD (Figure 7-2). This new and expanding understanding of the participation of the inflammatory process in atherosclerosis in no way challenges the importance of traditional risk factors, such as high LDL levels and hypertension. Indeed, inflammation provides a pathway that mechanistically links alterations in traditional risk factors and modifications in the biology of the artery wall that give rise to atherosclerosis and its complications.

171



a A blood-borne irritant injures the arterial wall, disrupting the endothelial layer and exposing the underlying connective tissue.



Blood platelets and circulating immune cells known as monocytes are then attracted to the site of the injury and adhere to the exposed connective tissue. The platelets release a substance referred to as platelet-derived growth factor (PDGF) that promotes migration of smooth muscle cells from the media to the intima.



• A plaque, which is basically composed of smooth muscle cells, connective tissue, and debris, forms at the site of injury.



 As the plaque grows, it narrows the arterial opening and impedes blood flow. Lipids in the blood, specifically low-density lipoprotein cholesterol (LDL-C), are deposited in the plaque.









Figure 7-1 Sequence of progression of atherosclerosis

Reprinted with permission from Kenney, W.L., Wilmore, J.H., & Costill, D.L. (2012). *Physiology of Sport and Exercise* (5th ed.). Champaign, Ill.: Human Kinetics.



Figure 7-2 The arterial inflammation process

Angina Pectoris

Angina pectoris is the discomfort (and sometimes pain) in the chest, arms, shoulders, and even jaw that results from inadequate blood flow, and more specifically oxygen, to the heart. Angina that occurs regularly with activity, upon awakening, or at other *predictable* times is termed **stable angina** and is associated with high-grade narrowing of the coronary arteries. The typical level of physical effort–related angina is proportionate to the exercise intensity but can also be influenced by temperature (e.g., cold weather conditions) and altitude. Angina can be easily graded by the "Functional Classification of Angina Pectoris" guidelines presented in Table 7-1. When appropriate, this angina classification is a useful tool for documenting client chest discomfort or pain. The symptoms of angina are often treated with nitrate medicines such as nitroglycerin, which come in short-acting and long-acting forms, and may be self-administered transdermally, sublingually (i.e., under the tongue), or orally as needed. Unstable angina is angina that changes in intensity, character, or frequency. Unstable angina may precede MI and requires urgent medical attention. Individuals who have unstable angina are not appropriate clients for the CMES and should always be referred back to their physicians.

TABLE	TABLE 7-1									
CANAD	CANADIAN CARDIOVASCULAR SOCIETY FUNCTIONAL CLASSIFICATION OF ANGINA PECTORIS									
Class	Definition	Specific Activity Scale								
I	Ordinary physical activity (e.g., walking and climbing stairs) does not cause angina; angina occurs with strenuous, rapid, or prolonged exertion at work or recreation	Ability to ski, play basketball, jog at 5 mph, or shovel snow without angina								
11	Slight limitation of ordinary activity. Angina occurs when walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, in cold, in wind, under emotional stress, or only during the few hours after awakening, when walking more than two blocks on level ground, or when climbing more than one flight of stairs at a normal pace and in normal conditions	Ability to garden, rake, roller skate, walk at 4 mph on level ground, or have sexual intercourse without stopping								
111	Marked limitation of ordinary physical activity. Angina occurs on walking one or two blocks on level ground or climbing one flight of stairs at a normal pace in normal conditions	Ability to shower or dress without stopping, walk 2.5 mph, bowl, make a bed, or play golf								
IV	Inability to perform any physical activity without discomfort	Anginal symptoms may be present at rest. Inability to perform activities requiring 2 or fewer metabolic equivalents without angina								

Source: Goldman, L. et al. (1981). Comparative reproducibility and validity of systems for assessing cardiovascular functional class: Advantages of a new specific activity scale. *Circulation*, 64, 1227–1234.

Cardiac Dysrhythmias

Cardiac **dysrhythmias** are cardiac rhythm disturbances that can be of atrial, **atrioventricular node (AV node)**, or ventricular origin. Many patients with CHD and/or who are post-MI or have had heart surgery have ventricular dysrhythmias. Some dysrhythmias are relatively benign, but some represent a high-risk state. For example, some rapid ventricular dysrhythmias can result in cardiac arrest. Cardiologists can prescribe several different classes of medicines or perform specific laboratory procedures (e.g., radiofrequency ablation or implantable defibrillators) that can reduce many types of cardiac dysrhythmias.

Cardiac dysrhythmias, especially ventricular arrhythmias, can be heart-rate and physicaleffort related, and thus can be elicited by physical exercise. For this reason, the CMES should be particularly conscious of symptoms that are induced by exercise-generated ventricular dysrhythmias. Such rhythm disturbances can transpire during or after exercise and occasionally a delayed onset of one to two hours after exercise, particularly intense exercise, can occur. These symptoms include dizziness, lightheadedness, **palpitations**, and, in rare occurrences, **syncope** (fainting). Any individual with a history of exercise-induced dysrhythmias should be thoroughly evaluated by a cardiologist. Without physician clearance, these individuals should not be considered stable, although many are or can be well managed by their cardiologist. The four primary cautions if such clients are referred to a CMES are as follows:

- Always graduate the workload slowly, with no sudden onset or cessation of moderate or vigorous exercise. Always have the client gradually warm up and cool down.
- Monitor the client for at least 10 to 15 minutes after an exercise session has concluded.
- Avoid heavy resistance exercise or any exercise in which the client is exerting against either an **isometric** load or very high resistance, particularly if the client also holds their breath or executes a **Valsalva maneuver** (expiration against a closed glottis).
- Inverted **hatha yoga** poses (head below the level of the heart) or rapid changes in body position are also not advised.

Myocardial Infarction

Acute MI is a medical condition that occurs when the blood supply to the heart muscle is interrupted, most commonly due to the rupture of a lipid-rich, vulnerable plaque. The resulting oxygen shortage, or **ischemia**, causes damage and potential death of some of the heart muscle cells below the blockage. MI symptoms may include various combinations of pain in the chest, upper extremity, or jaw, or epigastric discomfort with exertion or at rest (i.e., mid-back pain). The discomfort associated with acute MI usually lasts at least 20 minutes. Frequently, the discomfort is diffuse, not localized, not positional, not affected by movement of the region, and may be accompanied by shortness of breath, **diaphoresis** (profuse sweating), nausea, or



syncope. ECG and cardiac biomarkers (e.g., myocardial enzymes or cardiac troponin taken via blood test at the hospital) also help diagnose an MI. Patients frequently feel suddenly ill. Women often experience different symptoms than men. The most common symptoms of MI in women include shortness of breath, weakness, and fatigue. Approximately one-third of all MIs are "silent," without chest pain or other symptoms. Acute MI is a type of acute coronary syndrome, which is most frequently (but not always) a manifestation of CHD. In approximately half of all MIs, this is the individual's first indication of CHD. It is very important that the CMES be capable of recognizing the signs and symptoms of MI.

Training the post-MI client requires adherence to precautions very similar to those followed with the angina client. The CMES should never be in the position of training a client within four to six weeks of an MI and without direct written authorization and referral from the client's physician. Ideally, the client should have completed early phase cardiac rehabilitation or similarly supervised exercise therapy. It is important, however, to note that more than

70% of post-MI patients do not get referred to, or have access to, formal cardiac rehabilitation programs. In such cases, it is imperative that the CMES ensures that the individual has an appropriate physician evaluation prior to taking a referral. In all cases, clients should have a physician-supervised exercise ECG prior to working with the CMES. All post-MI clients, but especially those with an MI within the preceding eight to 12 weeks, should begin all exercise sessions with relatively low workloads [e.g., 3–5 **metabolic equivalents (METs)**] and progress very gradually. Most post-MI clients who have had symptom-free and negative exercise ECGs can progress to reasonably normal age- and gender-related aerobic and resistance work capacities over six to 12 months.

Heart Failure

One complication of CHD, particularly MI, is HF. More than 6.4 million Americans have HF, with more than 1 million new cases reported every year (AACVPR, 2021). An MI may compromise the function of the heart as a pump for the circulatory system, which can lead to HF. Essentially, HF is a condition characterized by a reduction in cardiac output sufficient to meet the body's metabolic demands, including many moderate-level physical activities. There are different types of HF. Left- or right-sided HF may occur depending on the affected part of the heart, which results in a low output. If one of the heart valves is affected, this may cause dysfunction, such as mitral valve regurgitation in the case of left-sided MI. The incidence of HF is particularly high in individuals with diabetes and requires special management strategies. These individuals, especially those who have poor **ventricular function** (i.e., the heart has a very poor pumping capacity), are at high risk for exercise-related complications. Some of the clinical manifestations of HF are listed in Table 7-2. Note that one key sign for the CMES is exercise intolerance (i.e., **dyspnea** at relatively low workloads).

The CMES should not train HF clients unless otherwise appropriately and specifically authorized by physician referral. Low-level progressive exercise (2) to 5 METs) can be helpful in early stages of training, but only with authorized supervision. Many restorative and easy yoga poses are also beneficial when appropriately taught by experienced and qualified yoga teachers. Higherintensity (50 to 70+% of VO, max) interval or continuous exercise has been used with reasonable success in cardiac rehabilitation programs but only after the patient has successfully completed lower levels of exercise training. Ismail et al. (2013) reviewed 74 HF exercise studies and reported that higher levels of exercise training can improve aerobic power by an average of 16% compared to less than half of that with low-level training. These findings indicate that the magnitude of gain in cardiorespiratory fitness is greater with increasing exercise intensity and appears to be unrelated to baseline fitness level or exercise volume. Moreover, high and vigorous exercise intensities do not appear to increase the risk of cardiac death, adverse events, and hospitalization. The CMES should use great caution when introducing higher-intensity exercise as a training stimulus in HF patients without direct clinical supervision.

DIAGNOSTIC TESTING AND CRITERIA

There are a number of reasons for a physician to order a battery of diagnostic tests to confirm or rule out CVD. In many cases, those with mild to moderate CHD have no major complaints other than fatigue. As the disease progresses, they may develop angina pectoris during physical activity. This type of pain typically subsides with rest. It is not uncommon for these symptoms to be ignored. If a patient presents with complaints of periodic chest pain, a physician will likely be aggressive in their diagnostics. In some cases, the patient does not have any complaints signifying CHD, but due to their multiple risk factors, warrants a closer look. In addition to standard blood work assessing blood **lipids** and lipoproteins, **glucose**, and inflammatory markers, noninvasive and/or invasive diagnostic tests will be ordered to evaluate the likelihood of any clinically significant blockage.

Electrocardiogram

An ECG is a graphic produced by an electrocardiograph machine, which records the electrical activity of the heart over time. Analysis of the various ECG waveforms and of electrical depolarization and repolarization yields important diagnostic information. A typical ECG tracing of the cardiac cycle (heartbeat) consists of a P wave, a QRS complex, a T wave, and a U wave. One of the most important diagnostic waveform lines in the ECG is the S-T segment, which connects

175

TABLE 7-2

CLINICAL MANIFESTATIONS OF HEART FAILURE

- Dyspnea (excessive shortness of breath) with physical effort
- Orthopnea (dyspnea at rest when lying flat)
- Elevated resting heart rate >100 bpm (tachycardia)
- Fluid retention, particularly in the extremities (peripheral edema)
- Weight gain
- Cold, pale, and possibly cyanotic extremities
- Exercise intolerance manifested by dyspnea and a rapid pulse

CHAPTER 7

the QRS complex and the T wave and represents the period when the ventricles are electrically depolarized. Most modern ECG monitors incorporate computerized electrocardiography, which records and measures ECG waveforms and time intervals.

ECG Exercise Testing

An exercise ECG test is a graded (gradual increase in speed and grade) exercise treadmill or stationary bicycle test with electrocardiographic recording. The test is considered "positive" if there is a specific standard level of change in the S-T segment component of the ECG, suggesting that there is a delay in the recovery after the contraction of the ventricles associated with narrowing of



the coronary arteries. The important diagnostic information that is recorded during a stress ECG is as follows: ECG response (S-T segment, S-T slope, and potential dysrhythmias), **heart rate (HR)** and **blood pressure (BP)** response, symptoms (angina, dyspnea, or dizziness), and the exercise level achieved (e.g., MET capacity). ECG stress testing can be employed for diagnostic or functional assessment. For functional assessment, the test is used primarily to evaluate the patient's symptomology, MET capacity, and training heart-rate response. The ECG stress test is not as effective of a diagnostic tool as a radionuclide (nuclear) stress test. It is important to note that standardized exercise ECG stress tests do have limitations in that they are not perfect for ruling in or out CHD. A well administered exercise ECG that stresses a patient to at least 90% of **maximal heart rate** will have a sensitivity of 73 to 90% and a specificity of 50 to 74% (Gibbons et al., 2002). Sensitivity is the percentage of sick people who are correctly identified as having the condition. Specificity indicates the percentage of healthy people who are correctly identified as not having the condition.

Medical clearance is strongly recommended for clients with CHD or with signs and symptoms of CHD, regardless of disease status, before beginning a moderate-intensity exercise program, or prior to progressing to vigorous-intensity exercise if they are already exercising regularly. If signs and symptoms of CHD are present in clients who are already exercising regularly, exercise should be discontinued until medical clearance is provided (ACSM, 2022).

Radionuclide Stress Test

Radionuclide stress testing involves injecting a radioactive isotope (typically thallium or cardiolyte) into the person's vein, after which an image of the heart becomes visible with a special camera. The radioactive isotopes are absorbed by a healthy heart muscle. Nuclear images are obtained in the resting condition and again immediately following exercise. The two sets of images are then compared. During exercise, if a significant blockage in a coronary artery or arteries results in diminished blood flow to a part of the cardiac muscle, this region of the heart will appear as a relative "cold spot" on the nuclear scan, signifying reduced or diminished blood flow. This cold spot may not be visible on the images that are taken while the patient is at rest (when coronary flow is adequate). Radionuclide stress testing, while more time-consuming and expensive than a simple exercise ECG, greatly enhances the accuracy in diagnosing CHD.

Stress Echocardiography

Another supplement to the routine exercise ECG is **stress echocardiography** (cardiac ultrasound). During stress echocardiography, the sound waves of an ultrasound are used to produce images of the heart at rest and at the peak of exercise. In a heart with normal blood supply, all segments of the left ventricle exhibit enhanced contractions of the heart muscle during peak exercise. Conversely, in the setting of CHD, if a segment of the left ventricle does not receive optimal blood flow during exercise, that segment will demonstrate reduced contractions of the heart muscle relative to the rest of the heart on the exercise echocardiogram. Stress echocardiography is very useful in enhancing the interpretation of the exercise ECG, and can be used to exclude the presence of significant CHD in patients suspected of having a "false positive" stress ECG.

Coronary Angiography (Cardiac Catheterization)

Coronary angiography involves inserting a catheter into an artery in the groin area and routing it into the coronary arteries of the heart. This procedure is done for both diagnostic and interventional purposes. A radio contrast agent is passed into the catheter and is visualized on a fluoroscope to evaluate coronary blood flow in the major arteries of the heart. The benefit of this procedure is that while the catheter is inside the heart, the cardiologist can perform a **percutaneous transluminal coronary angioplasty (PTCA)**, a procedure that uses a small balloon at the tip of the heart catheter to push open plaques. Coronary angiography has several goals:

- To confirm the presence of a suspected blockage in a coronary artery
- To quantify the severity of the disease and its effect on the heart
- To seek the cause of a symptom such as angina, shortness of breath, or other signs of cardiac insufficiency
- To make a patient assessment prior to heart surgery

Vascular Imaging Techniques

Several invasive and noninvasive imaging techniques have been evaluated for use in characterizing atherosclerosis. Invasive coronary angiography has traditionally been the standard clinical tool for visualizing coronary arteries. Since its introduction more than 30 years ago, more than 2 million coronary angiograms have been performed annually in North America. Although coronary angiography is extremely useful in diagnosing obstructive atherosclerosis, it does not effectively define the extent of atherosclerosis in the vessel wall. Intravascular ultrasound is a newer invasive technique that allows for the direct observation of a vessel's plaque volume. The development of noninvasive cardiovascular techniques, such as **computed tomography (CT)** imaging of coronary artery calcium, CT angiographic imaging, B-mode ultrasound of carotid intimamedia thickness (CIMT), and cardiovascular magnetic resonance imaging (CMRI), has enabled the more practical non-invasive evaluation of atherosclerosis at a preclinical stage.

Coronary Calcium Scoring

Coronary calcification is part of the pathogenesis of atherosclerosis and does not occur in normal vessels. Due to the association between coronary calcification and coronary plaque development, radiographically detected coronary artery calcium (CAC) can provide an estimate of total coronary plaque burden. Studies have reported that CAC scores are independently predictive of CHD outcomes, even after controlling for a variety of risk markers (Greenland et al., 2004). The primary methods for CAC measurement are electron-beam computed tomography (EBCT) and multi-detector computed tomography (MDCT).

CORONARY CALCIUM CT SCAN

A cardiac CT scan for coronary calcium is a non-invasive way of obtaining information about the presence, location, and extent of calcified **plaque** in the coronary arteries. Calcified plaque results when there is a build-up of fat and other substances under the inner layer of the artery. This material can calcify, which signals the presence of atherosclerosis. The goal of a cardiac CT scan for calcium scoring is to determine if CHD is present and to what extent, even if there are no symptoms. It is a screening study that may be recommended by a physician for patients with risk factors for CHD, but no clinical symptoms.

Calcified coronary plaque represents approximately 20% of the total coronary artery plaque burden, and thus, the more coronary calcium, the more atherosclerotic burden is present. The other 80% of the coronary plaque is fibrous plaque or lipid-rich (soft) plaque. Therefore, coronary calcium is a measure of a patient's atherosclerotic burden. After a coronary calcium scan, a calcium score called an **Agatston score** is generated. The score is based on the amount of calcium found in the coronary (heart) arteries. The client/patient may get an Agatston score for each

EXPAND YOUR KNOWLEDGE

CHAPTER 7

major artery and a total score. The test is negative if no calcium deposits (calcifications) are found in the arteries. This means the individual's chance of having a **heart attack** in the next two to five years is low. The test is positive if calcifications are found in the arteries. The higher the Agatston score is, the more severe the atherosclerosis. It is important to note that although this test is used frequently to help predict CHD risk, it is not perfect and the scan does emit a small amount of radiation. Its best use in predicting CHD is when the score is combined with other CHD risk factors, particularly LDL level. Table 7-3 presents a general guide to the range of calcium scores (Agatston units) and their relationship to severity of atherosclerosis.



TABLE 7-3	TABLE 7-3						
CALCIUM SCORES AND THE SEVERITY OF ATHEROSCLEROSIS							
0	No risk identified, negative test, low risk of CV event in next 5 years						
1–10	Minimal risk, low risk of CV event in next 5 years						
11–100	Mild risk, mild atherosclerosis is present, minimal coronary stenosis						
101–400	Moderate calcium is detected, moderate risk of a CV event in next 5 years						
>400	>400 High calcium score, significant risk of a CV event in next 5 years						

Note: CV = Cardiovascular

Source: National Cholesterol Education Program (2002). *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. National Heart, Lung, and Blood Institute National Institutes of Health NIH Publication No. 02-5215.

EXPAND YOUR KNOWLEDGE

OTHER VASCULAR IMAGING TECHNIQUES

Coronary CT Angiography

In CT angiography, computed tomography using a contrast material produces detailed pictures (a coronary CT angiogram). CT imaging uses special x-ray equipment to produce multiple images and a computer to join them together in cross-sectional views. This relatively new test is available to assess cholesterol plaque obstruction in the coronary arteries. Unlike a traditional coronary angiogram, CT angiograms do not use a catheter threaded through the blood vessels to the heart. Instead, a coronary CT angiogram relies on a powerful x-ray machine to produce images of the

heart and heart vessels. In the past, noninvasive functional tests of the heart were used, such as treadmill tests and nuclear studies, to indirectly assess if there were blockages in the coronary arteries. The only way to directly look at the coronary arteries was via a cardiac catheterization and coronary angiogram.

CT scans have been used to look at various anatomic regions, but have not been useful for the heart because the heart is continuously in motion. Today, a new generation of CT scanners that can take 64 pictures a minute is available; with the use of medication to slow the HR to less than 64, CT images of the coronary arteries are now possible (Figure 7-3). There is some concern about the amount of radiation dosage used in CT angiography, which has given rise to a "micro-dose" CT angiography that



Figure 7-3 Computed tomography (CT) scan

provides a simplified procedure with one-tenth of the radiation dose and at a reduced cost.

High-resolution Magnetic Resonance Imaging

High-resolution cardiovascular magnetic resonance imaging of the arterial wall is emerging as a powerful research technology for characterizing atherosclerotic lesions within carotid arteries and other large vessels. High-resolution **magnetic resonance imaging (MRI)** is able to noninvasively characterize three important aspects of atherosclerotic lesions: size, composition, and biologic activity. It can quantify not only wall and lumen areas and volumes, but also plaque composition. For example, high-resolution MRI can assess cap thickness and distinguish ruptured plaque caps from thick and stable caps. This technique can also be used to characterize the composition of a plaque by differentiating lipid-free regions from lipid-rich and calcified regions. In addition, high-resolution MRI can identify recent intra-plaque hemorrhages using multi-contrast-weighted studies.

Intravascular Ultrasound

Intravascular ultrasound (IVUS) is a valuable adjunct to coronary angiography. While angiography provides only a two-dimensional assessment of the lumen of the target vessel, IVUS allows the tomographic measurement (the recording of internal body images at a predetermined plane) of artery lumen area, plaque size, plaque distribution, and to some extent, plaque composition.

IVUS involves invasively placing a specialized catheter with a miniaturized ultrasound probe attached to the distal end of the catheter. The proximal end of the catheter is attached to computerized ultrasound equipment. It allows the application of ultrasound technology to see from inside blood vessels out through the surrounding blood column, visualizing the endothelium (inner wall) of blood vessels. Because the arterial remodeling and plaque deposition that characterize the early stages of atherosclerotic progression occur without decreases in lumen area, IVUS may be able to detect atherosclerotic disease at an earlier state than coronary angiography. In many cases, IVUS may provide the ability to detect some "angiographically silent" **atheromas**.

B-mode Ultrasound Assessment

B-mode ultrasound is a noninvasive imaging modality that employs ultrasound to accurately image the walls of arteries and is a useful tool for evaluating carotid intima-media wall thickness (CIMT). The normal arterial wall consists of three layers: the tunica intima, tunica media, and tunica adventitia. The thickness of the two innermost layers in the carotid artery (the intima and media), or the CIMT, is increasingly used as a surrogate marker for early atherosclerosis. Carotid ultrasound measurements correlate well with histology, and increased CIMT is associated with the presence of vascular risk factors and more advanced atherosclerosis, including CHD. Large observational studies have established that CIMT is an independent marker of risk for cardiovascular events. A meta-analysis of eight studies reported that the relative risk per one 0.10-mm difference in common carotid artery CIMT was 1.15 for MI and 1.18 for **stroke**, adjusted for age and sex (Lorenz et al., 2007).

TREATMENT OPTIONS

Pharmacological therapy is usually first-step therapy for CHD and often used in conjunction with surgical therapy. The CMES should be familiar with each of these classes of medications and their principle indications. The principle classes of drug therapy for CHD are beta blockers, angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), calcium channel blockers (CCBs), **diuretics, vasodilators,** cardiac glycosides, antiarrhythmic agents, blood modifiers (e.g., anticoagulants), and antilipemic agents (e.g., statins).

Aside from pharmacological therapy, the two interventions most often employed in CHD patients are **coronary artery bypass grafting (CABG)** and PTCA, also called percutaneous coronary intervention (PCI). PTCA will usually include intracoronary stenting (using a wire mesh device to open the artery). The CMES can work with all of these clients, as long as they have no complications and are stable from symptom, ventricular function, and ECG abnormality perspectives. Most often, these individuals require aggressive risk-factor control, especially blood lipid and BP management. The CMES should follow the exercise guidelines and

179

precautions in this chapter for all CHD clients. The most important rule to remember for CABG clients, however, is to avoid traditional resistance-training programs with moderate to heavy weights for the first six weeks post-surgery. This will give the sternum sufficient time



to heal from the CABG sternotomy (surgical opening of the sternum). Graduated upper-extremity range-of-motion exercises and many hatha yoga poses that do not place undue strain on the sternum or upper back are recommended for clients who have had CABG within the previous four to eight weeks. As with the post-MI client, the CMES should see these individuals only after physician evaluation and referral.

The success rate for CABG can reach beyond 10 years, but those with multiple risk factors may see coronary blockages in as little as six years post-CABG. Unfortunately, the success rates for angioplasty are not as promising. Up to 30% of post-PTCA clients will experience **restenosis** within the first six months of the procedure. PTCA is more effective than medical therapy in relieving angina, but it confers no greater survival benefit. Aggressive lipid-lowering therapy appears to be as effective as percutaneous coronary intervention plus usual medical care for preventing ischemic events (Boden et al., 2007).

A CMES working with clients who are post-PTCA or post-CABG should take notice of any of the following signs and symptoms of restenosis or worsening of the coronary atherosclerotic process:

- Complaints of general fatigue
- · Reduced exercise tolerance or accelerated HR at customary workloads
- Any symptoms of chest discomfort or pain

Since CHD is common among the American population, an astute CMES may pick up on these subtle complaints in any of the populations they serve.

NUTRITIONAL CONSIDERATIONS

A foundational component of working with clients with CHD is knowledge of general nutrition recommendations for optimal heart health and how to best share them with clients to help facilitate adoption of the recommendations. It is also helpful to know when and how to refer to a **registered dietitian (RD)** for **medical nutrition therapy (MNT)** and to have a general understanding of the nutrition considerations the RD is likely to recommend.

This section will first outline foundational principles for nutrition and behavior change for CVD prevention and then provide an overview of nutrition considerations for individuals who have been diagnosed with CHD and who may have experienced complications from a cardiovascular event, such as **congestive heart failure (CHF)**.

Cardiovascular Disease Prevention

While the CMES may work with clients who have already been diagnosed with CVD or are at very high risk of disease, knowledge and implementation of the guidelines to help prevent CVD are as valuable for secondary and tertiary prevention. In fact, the AHA states that preventive recommendations can be applied to the management of individuals with CVD, though in these cases the recommendations may have to be intensified (Eckel et al., 2013).

A critical component to the recommendations is consideration of the practicalities of implementing the guidelines, including behavior-change principles and the value of client-focused guidance, rather than simply sharing recommendations and adhering to the unrealistic expectation that the client will seamlessly convert recommendations into action. Table 7-4 highlights the AHA's major recommendations for lifestyle management.

In a follow-up to the release of these guidelines, the AHA published a paper on the best evidence to help implement the guidelines (Gidding et al., 2009). This paper placed heavy emphasis

on recognizing the barriers and opportunities for implementation of change on multiple levels including the individual, family, community, and policy (the **socio-ecological model**) (Figure 7-4). The AHA statement reinforces ACE's strongly held belief that principles of behavioral change—including client-centered focus, readiness to change, goal setting, and repeated review and evaluation of the success and feasibility of the intervention—drive exercise and nutrition interventions. Communication strategies such as **motivational interviewing** (see Chapter 5) are helpful to facilitate this process. During lifestyle coaching sessions, the CMES may have an opportunity to share tips to help support healthy nutrition choices. When that opportunity occurs, the AHA advises targeting the behaviors shown in Table 7-5.

TABLE 7-4

SUMMARY OF RECOMMENDATIONS FOR LIFESTYLE MANAGEMENT

DIET

LDL-C—Advise adults who would benefit from LDL-C lowering to:

- Consume a dietary pattern that emphasizes intake of vegetables, fruits, and whole grains; includes low-fat dairy products, poultry, fish, legumes, nontropical vegetable oils, and nuts; and limit intake of sweets, sugar-sweetened beverages, and red meats.
 - ✓ Adapt this dietary pattern to appropriate calorie requirements, personal and cultural food preferences, and nutrition therapy for other medical conditions (including diabetes mellitus).
 - ✓ Achieve this pattern by following plans such as the DASH eating plan, the Healthy U.S.-Style Dietary Pattern, or the AHA diet.
- Aim for a dietary pattern that achieves 5 to 6% of calories from saturated fat.
- Reduce percent of calories from saturated fat.
- Reduce percent of calories from trans fat.

BP-Advise adults who would benefit from BP lowering to:

• Consume a dietary pattern that emphasizes intake of vegetables, fruits, and whole grains; includes low-fat dairy products, poultry, fish, legumes, nontropical vegetable oils, and nuts; and limit intake of sweets, sugar-sweetened beverages, and red meats.

✓ Adapt this dietary pattern to appropriate calorie requirements, personal and cultural food preferences, and nutrition therapy for other medical conditions (including diabetes mellitus).

- ✓ Achieve this pattern by following plans such as the DASH eating plan, the Healthy U.S.-Style Dietary Pattern, or the AHA diet.
- Lower sodium intake.
 - ✓ Consume no more than 2,300 mg of sodium/day.
 - ✓ Further reduction of sodium intake to 1,500 mg/day is desirable since it is associated with an even greater reduction in BP.
 - ✓ Reduce intake by at least 1,000 mg/day, since that will lower BP, even if the desired daily sodium intake is not yet achieved.
- · Combine the DASH eating plan with lower sodium intake.

PHYSICAL ACTIVITY

Lipids

• In general, advise adults to engage in aerobic physical activity to reduce LDL-C and non-HDL-C: 3 to 4 sessions a week, lasting on average 40 minutes per session and involving moderate-to-vigorous intensity physical activity.

BP

• In general, advise adults to engage in aerobic physical activity to lower BP: 3 to 4 sessions a week, lasting on average 40 minutes per session and involving moderate-to-vigorous intensity physical activity.

Note: LDL-C = Low-density lipoprotein cholesterol; DASH = Dietary Approaches to Stop Hypertension; UDSA = U.S. Department of Agriculture; AHA = American Heart Association; BP = Blood pressure; non-HDL-C = non-high-density lipoprotein cholesterol

Source: Eckel, R.H. et al. (2013). 2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Journal of the American College of Cardiology*, 63, 25. DOI: 10.1016/j.jacc.2013.11.003

Figure 7-4

Influencing food choice: A multilevel framework for indentifying facilitors or barriers to attaining AHA Dietary Recommendations



Reprinted with permission from Gidding, S. et al. (2009). Implementing American Heart Association Pediatric and Adult Nutrition Guidelines: A Scientific Statement from the American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity and Metabolism, Council on Cardiovascular Disease in the Young, Council on Arteriosclerosis, Thrombosis and Vascular Biology, Council on Cardiovascular Nursing, Council on Epidemiology and Prevention, and Council for High Blood Pressure Research. *Circulation*, 119, 1161–1175.

TABLE 7-5

EXAMPLES OF EATING BEHAVIORS TO TARGET IN COACHING								
 Food selection Limiting sugar-containing beverages Use of a simply structured diet table that categorizes foods into three easily identifiable groups (e.g., "go" for good foods and "slow" and "whoa" for poor foods) 	 Food presentation Eat more meals as a family Reduce portion size Choose healthy alternatives to poor food choices Repeat presentation of foods not well liked 							
 Food acquisition Make healthier choices on foods prepared and/or purchased outside the home Shop for healthier foods Control food availability in the home 	 Self-monitoring Routine weighing so that caloric intake adjustments can be made Scheduled physical activity Record of caloric intake 							

Additional strategies

- Praise for meeting goals from peers and others in the home must be provided
- Behavioral contracting with nonfood rewards; reinforcement should be social and not related to food, money, or gifts
- Removal of stimuli for undesired or inappropriate food choices
- · Parents must model desired or appropriate behaviors

Reprinted with permission from Gidding, S. et al. (2009). Implementing American Heart Association Pediatric and Adult Nutrition Guidelines: A Scientific Statement from the American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity and Metabolism, Council on Cardiovascular Disease in the Young, Council on Arteriosclerosis, Thrombosis and Vascular Biology, Council on Cardiovascular Nursing, Council on Epidemiology and Prevention, and Council for High Blood Pressure Research. *Circulation*, 119, 1161–1175.

Coronary Heart Disease

CHD results from atherosclerotic narrowing of the coronary arteries likely to produce angina pectoris or MI. Thus, a major objective of nutrition therapy for individuals with CHD is to minimize atherosclerotic narrowing, or at least decrease the risk of an atherosclerotic lesion causing an MI.

Individuals with CHD are well served by an evaluation from an RD for MNT. Together the RD and client will develop an eating plan and lifestyle changes that are best suited to help prevent and treat CHD.

The Academy of Nutrition and Dietetics (A.N.D.) has established guidelines for optimal nutrition management for several cardiovascular conditions, including lipid disorders, hypertension, and CHF through its Evidence Analysis Library, a tool for RDs that synthesizes the nutrition research and offers evidence-based recommendations to help guide clinical decisions. The components of those guidelines that are most specific to CHD are included here. Further details for lipid disorders and hypertension are included within their respective chapters in this text (Chapters 8 and 9).

REGISTERED DIETITIANS AND MEDICAL NUTRITION THERAPY

A critical partner in the nutrition management of an individual with CHD is the RD, ideally one with specialized training in nutrition therapy for CVD.

The A.N.D Evidence Analysis Library reports that patients who attend several visits with an RD over a period of six to 12 weeks effectively decrease dietary fat, **saturated fat**, and energy intake. Research suggests that these nutrition changes result in a 7 to 21% reduction in total cholesterol, a 7 to 22% reduction in LDL cholesterol, and an 11 to 31% reduction in triglycerides. The best results occur with at least three to six visits to an RD (A.N.D., 2013a).

In these visits, the RD implements MNT, which includes:

- A comprehensive nutrition assessment
- Planning and implementation of an evidence-based nutrition intervention
- Monitoring and evaluation of a client's progress

Specifically, during the MNT visits, the RD assesses the client's current food and nutrition intake; related health history including medication, supplement use, labs, and anthropometrics; physical activity and sleep history; and current knowledge, attitudes, and beliefs about food and behavioral change. From there, the RD will determine the client's ideal energy and **macronutrient** needs, and compare needs with current intake. During the initial visit, the RD will recommend a meal plan based on the best evidence for optimal nutrition management of CHD, as well as the client's individual factors. Follow-up visits will gauge the client's adherence to recommendations, troubleshoot challenges, and acknowledge successes.

In many cases for insured individuals, MNT is a reimbursable service for individuals diagnosed with CHD with referral from a physician.

Macronutrient Composition

Individuals with CHD are advised to consume 25 to 35% of calories from fat (with <7% of calories from saturated fat and as little **trans fatty acids** as possible). Total **protein** should comprise 15 to 20% of calories and 45 to 60% of calories should be from total **carbohydrates**, with emphasis on high-fiber sources and avoidance of refined carbohydrates. Individuals with baseline elevated saturated fat intake may benefit from replacing saturated fat calories with **unsaturated fats**, protein, or **complex carbohydrates** (fruits, vegetables, and whole grains). This substitution can decrease LDL cholesterol by up to 16%, subsequently decreasing the risk for a cardiovascular event (A.N.D., 2013a).

Specific Cardioprotective Nutrients

The A.N.D.'s Evidence Analysis Library (2013a; 2013b) also discusses nutrients thought to be cardioprotective. The main findings are highlighted here.

EXPAND YOUR KNOWLEDGE

CHAPTER 7

Omega-3 Fatty Acids

Knowledge of specific dietary components that help to optimize heart health continues to evolve. A fair body of evidence supports that individuals with CHD should consume two or more 4-oz servings of fatty fish per week, due to their high content of **omega-3 fatty acids**. These fatty acids, in particular docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA), have been shown to decrease risk of arrhythmias and fatal heart disease and decrease progression of coronary atherosclerosis in individuals with diagnosed CHD. High intake of plant-derived alpha-linolenic acid (ALA) (about 1.4 grams per day) is associated with decreased risk of cardiac death, non-fatal MI, and recurrent MI. **Adequate Intake (AI)** of ALA is 1.6 grams per day for men and 1.1 grams per day for women. No AI has been established for DHA or EPA, although the Institute of Medicine (IOM, 2005) suggests that 10% of the needed ALA could come from EPA or DHA, which suggests a daily intake of about 100 mg per day. Some expert panels have recommended much higher intakes of 250 and 500 mg per day due to the significant health benefits attributed to these fatty acids and the low risk of complications such as bleeding, even at this higher range (Harris, 2010).

Some RDs may advise clients with CHD but without angina or implantable cardioverter defibrillators (ICD) to supplement their diet with 850 mg per day of EPA and/or DHA to reduce risk of sudden death by 45%. (This is in contrast to individuals *without* CHD or those who have angina or ICD in which supplementation may be contraindicated.) Consumption of more than 3 grams per day of omega-3 fatty acids may cause gastrointestinal distress.

Antioxidants and Plant Stanols and Sterols

Antioxidant-rich whole foods, including fruits, vegetables, whole grains, and nuts high in vitamins E, C, and β -carotenoids (a form of vitamin A), appear to provide cardiovascular protection. However, supplementation of these antioxidants does not appear to provide benefit (Sesso et al., 2008; Cook et al., 2007). Plant **stanols** and **sterols** are cholesterol-mimicking substances that occur naturally in many whole grains, vegetables, fruits, legumes, nuts, and seeds. They also have been added to many common foods such as cereals and bars, margarine, and orange juice. Plant stanols and sterols so closely resemble cholesterol that they can bind to cholesterol receptors and prevent cholesterol from being absorbed into the bloodstream. Then, instead of eventually depositing into plaques in the arteries, they are excreted with the waste. Consumption of 2 to 3 grams per day of plant stanols and sterols appears to decrease total cholesterol with no documented adverse effects (see Chapter 8).

Nuts

Consumption of 5 ounces of unsalted and lower-saturated fat tree nuts (e.g., walnuts, almonds, pecans, and pistachios) per week decreases risk of CHD, mostly due to their omega-3-rich fatty acid profile, which helps to decrease total and LDL cholesterol. However, nuts have high caloric density (5 ounces of nuts is equivalent to approximately 900 kcal). It is essential that they serve as an isocaloric substitute, rather than an addition, to the diet.

Alcohol

Individuals with CHD may experience decreased risk of CVD from alcohol consumption (a maximum of one drink per day for women and up to two drinks per day for men) due to its effects on cholesterol composition. With that said, nondrinkers should never be encouraged to begin drinking in an effort to decrease CVD risk.

B Vitamins

High consumption of whole-food sources of B vitamins including **folate**, vitamin B6, and vitamin B12 provides cardioprotective benefit. However, supplementation of these vitamins does not appear to provide cardiovascular benefit.

Fiber

Diets high in fiber (at least 25 to 30 grams per day), especially soluble fiber, provide substantial cardiovascular benefit, including a reduction in total and LDL cholesterol. Rich sources of soluble fiber include fruits, vegetables, and whole grains, especially high-fiber cereals, oatmeal, and legumes.

Congestive Heart Failure

As with other chronic conditions, referral to an RD for MNT of congestive heart failure is important. The RD will work with client to determine caloric, macronutrient, and **micronutrient** needs. RDs take several unique nutrition variables into consideration when developing a nutrition plan for a client with CHF, including protein to replace losses, fluid intake, specific micronutrient needs, and supplementation. A summary of recommendations from A.N.D.'s Evidence Analysis Library on Congestive Heart Failure (A.N.D., 2013b) are outlined here:

• *Protein:* Evidence suggests that individuals with CHF have higher protein needs than those without CHF. Protein needs vary considerably based on the extent of protein depletion as assessed by clinical and lab measures. On average, stable depleted clients need about

1.37 g/kg of protein per day, and adequately nourished need about 1.12 g/kg of protein per day.

- *Fluid intake:* Recommended fluid intake ranges from 1.4 to 1.9 liters per day (48 to 64 oz), with the actual advised amount within that range depending on clinical symptoms including **edema**, fatigue, and shortness of breath. Evidence supports that this fluid restriction helps to lessen symptoms and improve quality of life.
- *Sodium:* Recommended intake is less than 2 grams per day. As with fluid restriction, sodium restriction helps to lessen symptoms and improve quality of life.
- Folate and Vitamin B12: Consuming at least the **Dietary Reference Intake (DRI)** of folate and getting at least 200 to 500 mcg of vitamin B12 (whether through diet or supplementation) can improve clinical symptoms.
- *Thiamine:* Diuretic use is the standard of care for individuals with CHF. In some cases, diuretics can cause thiamine deficiency. For this reason, clients should be sure to consume (either through food or supplementation) at least the AI of 1.2 mg/day for men and 1.1 mg/day for women of thiamine (IOM, 2005).
- *Magnesium:* Individuals with CHF should be sure to consume at least the RDA of 320 mg/ day for women >30 years and 420 mg/day for men >30 years, as low levels of magnesium can cause heart arrhythmias in those with CHF.
- *L-Arginine, Carnitine, Coenzyme Q10, and Hawthorn:* Insufficient evidence exists to confirm or deny the potential benefit of these supplements for individuals with CHF.

Eating Plans to Support Optimal Heart Health

An overall healthy, balanced eating plan to meet calorie needs is helpful in preventing and treating CHD. Many different eating plans and eating patterns have been studied, including very low-fat diets such as the Ornish plan to more moderate eating plans such as the Therapeutic Lifestyle Changes (TLC), *Dietary Guidelines for Americans* (U.S. Department of Agriculture, 2020), **Dietary Approaches to Stop Hypertension (DASH) eating plan**, and the **Healthy Mediterranean-Style Dietary Pattern**. (More information about the TLC eating plan is discussed in Chapter 8 and the DASH plan is included in the nutrition section of Chapter 9. The Ornish and Mediterranean eating plans are described here.)



CHAPTER 7

Each of these dietary approaches has demonstrated cardiovascular health benefits. In fact, these eating plans contain common elements, including: high in vegetables and fruits, high in whole grains, moderate amounts of protein-rich foods, limited added sugars, and emphasis on oils over solid fats. The plans are generally lower in sodium, saturated fat, and caloric density than the typical American diet and higher in fiber and potassium. In fact, the typical American diet is far from the recommendations in many of the dietary variables that best predict (or protect from) CVD. Figure 7-5 highlights the typical American diet compared to recommended intakes.



Data Source: Analysis of What We Eat in America, NHANES 2013-2016, ages 1 and older, 2 days dietary intake data, weighted. *Recommended Intake Ranges*: Healthy U.S.-Style Dietary Patterns (see *Appendix 3*).

Figure 7-5

Dietary intakes compared to recommendations: Percent of the U.S. population ages 1 and older who are below and at or above each dietary goal Reprinted from United States Department of Agriculture (2020). 2020-2025 Dietary Guidelines for Americans (9th ed.). www.dietaryguidelines.gov

Ornish Diet and Lifestyle Spectrum Plan

Dean Ornish, a physician-researcher at the University of California-San Francisco and founder of the Preventive Medicine Research Institute was the first to demonstrate that nutrition, exercise, and stress management can *reverse* heart blockages. Ornish published a ground-breaking randomized-controlled study that first showed regression of disease after a one-year intensive lifestyle intervention (Ornish et al., 1990). A follow-up study found regression at both one and five years. In this intervention, 48 patients with moderate to severe CHD who were randomized to an intensive lifestyle change (consumption of a 10% fat, whole foods vegetarian diet, aerobic exercise, stress-management training, smoking

cessation, and group psycho-social support) experienced reversal of their CVD (evidenced by decreased percent stenosis by 1.75 absolute percentage points by 1 year and 3.1 absolute percentage points after 5 years). The control group experienced a significant increase of stenosis and 45 cardiovascular events (compared to 28 in the experimental group) (Ornish et al., 1998). Numerous subsequent studies as part of this trial, coined the Lifestyle Heart Trial, have continued to validate these findings. The major challenge for the average person is adherence to such an eating plan. Recognizing this, Ornish has evolved his program to be more personalized through his Ornish Lifestyle Spectrum program (www.ornishspectrum.com).

Healthy Mediterranean-Style Dietary Pattern

Another particularly effective eating plan for individuals with, or at high risk of, CHD is the Healthy Mediterranean-Style Dietary Pattern. While there are many variations of the Mediterranean dietary pattern, it generally is high in vegetables, fruits, nuts, olive oils, and whole grains. Wine is included at most meals. Meats and full-fat milk products are limited. Overall, this eating plan is relatively high in **monounsaturated fat** and low in saturated fat. Many research studies evaluating Mediterranean dietary patterns use the following standards in determining how well a participant follows this type of dietary pattern (Trichopoulou et al., 2003):

- Vegetables (other than potatoes): 4 or more servings per day
- Fruits: 4 or more servings per day
- Whole grains: 2 or more servings per day
- Beans (legumes): 2 or more servings per week
- Nuts: 2 or more servings per week
- Fish: 2 or more servings per week
- Red and processed meat: 1 or fewer servings per day
- Dairy foods: 1 or fewer servings per day
- Alcohol: 1/2 to 1 drink per day for women, 1 to 2 for men

The body of research on the Healthy Mediterranean-Style Dietary Pattern continues to grow, with many studies demonstrating its efficacy in improving CVD markers such as weight, BP, fasting glucose, total cholesterol, C-reactive protein, stroke, and MI (Hoevenaar-Blom et al., 2012; Nordmann et al., 2011). Another study found a 73% lower risk of cardiac death and non-fatal MI compared to a prudent diet in study participants who had experienced a previous MI (de Lorgeril et al., 1999). Overall, greater adherence to a Mediterranean-style eating plan is associated with improved cardiovascular health (Sofi et al., 2010).

How familiar are you with the Ornish eating plan and the Healthy Mediterranean-Style Dietary Pattern? Although it is outside the scope of practice for a CMES to provide eating plans to clients, it is important to understand the principles of eating a heart-healthy diet for those with CHD so that you can recognize when a client may need a referral to an RD for further support. Take the time to read the references provided for the Ornish and Mediterranean plans so that you have a good foundational understanding of how a supportive diet can influence heart health.

EXERCISE TRAINING RECOMMENDATIONS FOR CLIENTS WITH STABLE CHD

The last three decades have seen numerous published studies demonstrating the many benefits of cardiac rehabilitation, but particularly the exercise-training benefits in CHD clients (Table 7-6) (Lavie et al., 2009). These are essentially the same benefits realized by apparently healthy individuals without CHD, but are clearly more of a priority in those with CHD.





TABLE 7-6

KEY BENEFITS OF SYSTEMATIC EXERCISE TRAINING IN THOSE WITH CORONARY HEART DISEASE							
 Improved exercise capacity and VO₂max (aerobic power) Increased muscular fitness (e.g., strength, endurance, and flexibility) 	 Increased myocardial ischemia tolerance Increased myocardial capillary density Reduced adiposity and metabolic syndrome risk factors 						
 Lessening of angina symptoms/raising of the ischemic threshold 	Reduced stress						
 Modest decreases in body fat, blood pressure, total and LDL cholesterol, non-HDL cholesterol, triglycerides, and high-sensitivity c-reactive protein (hsCRP) 	 Control of diabetes mellitus Improved well-being and self-efficacy Improvement in behavioral characteristics (depression, anxiety, and hostility) 						
 Increased HDL cholesterol Improved endothelial (arterial) function 	Reduction in overall morbidity and mortality (especially that associated with depression and psychological distress)						

Note: LDL = low-density lipoprotein; HDL = high-density lipoprotein

Appropriate Program Candidates and Stable CHD

Generally speaking, a CMES who wishes to work with individuals with CHD or those who are prone to CHD may see clients in one of two categories: those who do not have clinically documented CHD but who have elevated risk for CHD because of their CHD risk factors and those who have documented and stable CHD. Both groups require consideration for when a



pre-program exercise ECG test is recommended. ACSM, along with the AACVPR, has published and updated guidelines for when a medical examination, exercise testing with ECG, and supervision of exercise testing is recommended. Refer to Chapter 3 for information on how to perform a pre-exercise health-risk assessment.

Note that exercise testing here should not be confused with "fitness testing," but instead refers to clinical exercise testing with ECG (i.e., exercise ECG) performed by a physician and/or a clinical exercise physiologist. It is imperative that the CMES understand the difference between pre-program clinical exercise ECG testing administered by clinical personnel and supervised by a physician and periodic submaximal fitness testing.

Although cardiovascular disease risk factors are no longer used to determine if medical clearance is required prior to initiating an exercise program, they should still be considered, as recognizing and controlling these risk factors is imperative for cardiovascular disease prevention and management (ACSM, 2022). The intended use for identifying risk factors is to aid in the identification of occult coronary artery disease and to

then work together with clients on lifestyle behavioral change. Assessing CVD risk factors as part of a comprehensive health screening affords relevant information to be used not only for personalized exercise program design, but also for providing client education and collaborating on goals and next steps for lifestyle modification.

Fitness Testing CHD Patients

The CMES can administer most fitness tests to clients with stable CHD, provided that the client meets the overall considerations and recommendations in Table 7-7 and has no contraindications to resistance exercise. Appropriate fitness tests include flexibility, muscular endurance, and strength tests in which the client does not exert to muscular contraction "failure." Good cueing will prevent breath-holding (Valsalva maneuver). Inappropriate tests are those that push the client to a near-maximal perceived exertion, predicted HR, or \dot{VO}_2 max. Submaximal aerobic endurance tests may be performed, but only when the client has had a recent physician-supervised negative exercise ECG and is free from exercise-related cardiac symptoms (e.g., angina and dysrhythmias).

TABLE 7-7

OVERALL RECOMMENDATIONS FOR TRAINING STABLE CORONARY HEART DISEASE (CHD) CLIENTS

- Perform a 5- to 10-minute warm-up and cool-down with each exercise session that includes static and dynamic stretching and light to very light aerobic activities.
- The aerobic portion of exercise sessions should emphasize increased caloric expenditure by using rhythmic largemuscle-group activities.
- Have clients perform conditioning for the upper and lower body using a variety of aerobic activities and exercise equipment.
- Consider all safety factors, including clinical status, risk stratification category, exercise capacity, adverse event/ ischemic/angina threshold, cognitive/psychological impairment, and musculoskeletal limitations.
- Observe the presence of classic angina pectoris that is induced with exercise and relieved with rest or nitroglycerin, as this is evidence of myocardial ischemia.
- If an adverse event threshold has been established, exercise intensity should remain 10 bpm below that threshold HR.
- If peak HR is unknown, teach individuals to use RPE to guide exercise intensity.
- HIIT may be beneficial for this population.
- Individuals should use medications based on recommendations from their healthcare providers.
- If a change to beta-blocker medication has occurred without a new exercise test, monitor for signs and symptoms and note the RPE and HR most recently used during training as the new THR.
- If using diuretic therapy, emphasize and educate clients on proper hydration.
- Any amount of exercise is better than none, and multiple shorter daily bouts may be appropriate initially, with a gradual progression as tolerated.
- Perform an exercise test when there are clinical changes or symptoms that may indicate a compromised ability to exercise.
- Consider premorbid activity levels, the client's vocational and avocational goals and requirements, and personal health and fitness goals.

Note: bpm = Beats per minute; HR = Heart rate; RPE = Rating of perceived exertion; HIIT = High-intensity interval training; THR = Target heart rate

Source: American College of Sports Medicine (2022). *ACSM's Guidelines for Exercise Testing and Prescription* (11th ed.). Philadelphia: Wolters Kluwer.

Fitness testing protocols are discussed in more detail in *The Exercise Professional's Guide to Personal Training* (ACE, 2020) and will not be included here. The CMES must follow four rules when performing fitness assessments in patients with CHD:

- Avoid testing to maximal aerobic power (i.e., VO₂max) or maximal heart-rate levels when assessing cardiorespiratory endurance.
- Always warm up prior to the fitness test with lower-level activity similar in nature to the fitness test protocol (i.e., do not allow the client to engage in sudden-onset intense exercise of any type), and always cool down after the test with low-level aerobic activity commensurate with the intensity of the primary conditioning activity.
- Ensure that the client is currently taking all of their prescription medicines, especially if they are on beta blockers, ACE inhibitors, and/or ARBs.

• Clearly understand and recognize signs and symptoms of inappropriate response to exercise (e.g., chest discomfort, dyspnea, unusual fatigue, confusion, dizziness or lightheadedness, and/or the reporting of palpitations prior to exercise).

The CMES should adhere to the general procedures for submaximal testing of cardiorespiratory fitness (Table 7-8).

TABLE 7-8

GENERAL PROCEDURES FOR SUBMAXIMAL TESTING OF CARDIORESPIRATORY FITNESS

- Obtain resting heart rate and blood pressure immediately prior to exercise in the exercise posture.
- The individual should be familiarized with the ergometer or treadmill. If using a cycle ergometer, properly position the individual on the ergometer (i.e., upright posture, ~25-degree bend in the knee at maximal leg extension, and hands in proper position on the handlebars).
- The exercise test should begin with a two- to three-minute warm-up to acquaint the individual with the cycle ergometer or treadmill and prepare him or her for the exercise intensity in the first stage of the test.
- A specific protocol should consist of two- or three-minute stages with appropriate increments in work rate.
- Heart rate should be monitored at least two times during each stage, near the end of the second and third minutes of each stage. If heart rate is >110 beats per minute (bpm), steady-state heart rate (i.e., two heart rates within 5 bpm) should be reached before the workload is increased.
- Blood pressure should be monitored in the last minute of each stage and repeated (verified) in the event of a hypotensive or hypertensive response.
- Rating of perceived exertion (using either the Borg category or category-ratio scale) and additional rating scales should be monitored near the end of the last minute of each stage.
- Individual's appearance and symptoms should be monitored and recorded regularly.
- The test should be terminated when the individual reaches 70% of heart-rate reserve (85% of agepredicted maximal heart rate), fails to conform to the exercise test protocol, experiences adverse signs or symptoms, requests to stop, or experiences an emergency situation.
- An appropriate cool-down/recovery period should be initiated consisting of either:
 - ✓Continued exercise at a work rate equivalent to that of the first stage of the exercise test protocol or lower or
 - ✓A passive cool-down if the individual experiences signs of discomfort or an emergency situation occurs
- All physiologic observations (e.g., heart rate, blood pressures, signs, symptoms) should be continued for at least five minutes of recovery unless abnormal responses occur, which would warrant a longer post-test surveillance period. Continue low-level exercise until heart rate and blood pressure stabilize, but not necessarily until they reach pre-exercise levels.

Reprinted with permission from American College of Sports Medicine (2022). ACSM's Guidelines for Exercise Testing and Prescription (11th ed.). Philadelphia: Wolters Kluwer.

Contraindications to Exercise Training

Similar to other clinical populations who present high risk for exercise-related complications, there are specific instances when the CMES should not train a CHD client. Table 7-9 includes standardized contraindications to exercise training that are consistent across nearly all of the professional organizations addressing cardiac rehabilitation physical activity. Exceptions should be considered based on the clinical judgment of the physician and qualified clinical exercise physiologists experienced in cardiac rehabilitation.

Exercise Training Supervision Considerations

Most patients/clients who have successfully completed 24 to 36 sessions of formalized early phase cardiac rehabilitation will not require direct supervision of every exercise session. This does not mean that they will not require specific supervised instruction by the CMES on exercise techniques and familiarization with new modes of exercise training. The AACVPR (2021) has

190

TABLE 7-9

INDICATIONS AND CONTRAINDICATIONS FOR INPATIENT AND OUTPATIENT CARDIAC REHABILITATION

Indications	Contraindications					
Medically stable postmyocardial infarction	Unstable angina					
Stable angina	Uncontrolled hypertension (resting systolic blood					
Coronary artery bypass graft (surgery)	pressure >180 mmHg and/or resting diastolic blood pressure >110 mmHg)					
 Percutaneous transluminal coronary angioplasty 	 Orthostatic blood pressure drop of >20 mmHg with symptoms 					
• Stable heart failure caused by either systolic or diastolic dysfunction (cardiomyopathy)	 Significant aortic stenosis (aortic valve area <1.0 cm²) 					
Heart transplantation	Uncontrolled atrial or ventricular arrhythmias					
Valvular heart disease/surgery	Uncontrolled sinus tachycardia (>120 bpm)					
Peripheral arterial disease	Uncompensated heart failure					
 At risk for coronary artery disease with diagnoses of diabetes mellitus, dyslipidemia, hypertension, or obesity 	Third-degree atrioventricular block without pacemaker					
Other patients who may benefit from	Active pericarditis or myocarditis					
structured exercise and/or patient education	Recent embolism (pulmonary or systemic)					
based on physician referral and consensus of the rehabilitation team	Acute thrombophlebitis					
	Aortic dissection					
	Acute systemic illness or fever					
	Uncontrolled diabetes mellitus					
	Severe orthopedic conditions that would prohibit exercise					
	 Other metabolic conditions, such as acute thyroiditis, hypokalemia, hyperkalemia, or hypovolemia (until adequately treated) 					
	Severe psychological disorder					

Source: Balady, G.J. et al. (2007). 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*, 115, 2675–2682.

Reprinted with permission from American College of Sports Medicine (2022). ACSM's Guidelines for Exercise Testing and Prescription (11th ed.). Philadelphia: Wolters Kluwer.

published recommendations on exercise supervision requirements based on the individual's risk of exercise-related cardiovascular complications. Those at the lowest, moderate, and highest risk for complications all require various levels of supervision (Table 7-10). The CMES can minimize risk by ensuring appropriate physician referral, requiring pre-program exercise ECG testing when necessary, and conservative application of progressive exercise training stimulus, particularly in the early stages of training. The CMES is also required to be currently certified in **cardiopulmonary resuscitation (CPR)** and proper use of an **automated external defibrillator (AED)** and must be fully knowledgeable of the 2020 AHA CPR guidelines (Merchant et al., 2020).

STRATIFICATION OF RISK FOR C	ARDIAC EVENTS DURING EXER	CISE PARTICIPATION
Characteristics of Patients at Lowest Risk	Characteristics of Patients at Moderate Risk	Characteristics of Patients at Highest Risk
 Absence of complex ventricular dysrhythmias during exercise testing and recovery Absence of angina or other significant symptoms (e.g., unusual shortness of breath, lightheadedness, or dizziness during exercise testing and recovery) Normal heart rate and blood pressure response to exercise and recovery Functional capacity ≥7 METs 	 Presence of stable angina or other significant symptoms (e.g., unusual shortness of breath, lightheadedness, or dizziness) occurring only at high levels of exertion (≥7 METs) Functional capacity <5 METs 	 Presence of complex ventricular arrhythmias during exercise testing or recovery Presence of angina or other significant symptoms [e.g., unusual shortness of breath, lightheadedness, or dizziness at low levels of exertion (e.g., <5 METs) or during recovery] Abnormal clinical exercise ECG (e.g., ST-segment abnormalities) during exercise testing or recovery Abnormal heart rate or blood pressure response to exercise and recovery Functional capacity ≤3 METs

TABLE 7-10

Note: MET = Metabolic equivalent; ECG = Electrocardiogram

Sources: American Association of Cardiovascular and Pulmonary Rehabilitation (2021). *Guidelines for Cardiac Rehabilitation Programs* (6th ed.). Champaign, III.: Human Kinetics; Williams, M.A. (2001). Exercise testing in cardiac rehabilitation: Exercise prescription and beyond. *Cardiology Clinics*, 19, 3, 415–431.





All Americans, including those with stable and symptom-free CHD, should participate in an amount of energy expenditure equivalent to 150 minutes per week of moderate-intensity aerobic physical activity or 75 minutes per week of vigorous-intensity aerobic physical activity or a combination of both that generates energy equivalency for substantial overall health benefits including cardiovascular health (U.S. Department of Health & Human Services, 2018). This seems to be an acceptable goal for most CHD patients who successfully complete graduated cardiac rehabilitation physical-activity programs. This volume includes systematic workouts, recreational activities, and activities of daily living (ADL). Considering that most CHD patients have one or more CHD and/or metabolic syndrome risk factors that are related to increased adiposity, it seems reasonable to consider at least 2,000 kcal per week of moderate physical activity as an optimal goal, which is the overall ACSM recommendation for individuals who have overweight or obesity (Donnelly et al., 2009). This volume of activity approximates 250 to 300 minutes per week of moderate-level exercise. The CMES must understand how to estimate the energy cost of various physical activities in terms of kcal per session, per day, and per week (Howley, 2012; Ainsworth et al., 2011). The CMES should adhere to the overall recommendations for exercise in clients with stable CHD (see Table 7-7).

CLINICAL PEDOMETRY AND THE CHD PATIENT

Clinical pedometry [the process of employing well-engineered reliable step-counters (pedometers) in clinical disease prevention and management programs] can be helpful, particularly in the maintenance stages of CHD exercise programming. Note that walking for 30 minutes equates to 3,000 to 4,000 steps, whereas a 1-mile walk equates to approximately 2,000 steps.

Research has indicated that approximately 8,000 steps a day approximates 30 minutes of moderate to vigorous physical activity (i.e., approximately 8,000 steps a day of walking is an acceptable proxy for 30 minutes of moderate to vigorous physical activity) (Tudor-Locke et al., 2011). This does not mean that 8,000 walking steps (approximately 3 to 4 miles of walking) can or should be done in 30 minutes; however, keeping up a moderate pace (rather than a more leisurely cadence) is required to derive the benefits of higher-intensity aerobic activities. This level of daily walking appears to be what would be required for CHD patients who wish to maintain at least a minimum level of cardiovascular health. There is evidence that targeting 7,500 to 8,000 steps daily may be efficient to maintain waist circumference and to improve lipid profile during the year following a MI or acute coronary syndrome (Houle et al., 2013). This could be considered a starting target point to initiate changes in physical-activity behavior. Considerably more steps per day are generally required for weight loss. It is important to note that the CMES should use the step count (daily, weekly, or monthly) as the principal outcomes measure versus projected estimates of caloric expenditure or distance.

There is also evidence that the walking speed of ≥ 100 steps/minute represents the lower boundary of moderate-intensity walking for most adults. Indeed, to meet current U.S. public health physical-activity guidelines, individuals are encouraged to walk a minimum of 3,000 steps in 30 minutes (approximately 3 mph for most adults) on five days each week (Marshall et al., 2009). Three bouts of 1,000 steps in 10 minutes each day can also be used to meet the recommended goal. This walking speed, when maintained, is sufficient to stimulate insulin sensitization among other cardiometabolic mechanisms that are important in diabetes- and CHD-prevention programs. Does this mean that anything slower than 3,000 steps in 30 minutes is not clinically effective? Absolutely not, but at higher muscle contraction frequencies (i.e., walking speeds) there is greater regulation of lipid and glucose metabolism, which are key mechanisms that underscore cardiometabolic risk reduction in both diabetes- and CHD-prevention programs. It is also important to note that the 3 mph speed threshold would be faster than necessary for shorter individuals (e.g., <5'7", or perhaps slower than optimal for those taller than 6'2"), so height and gait mechanics should be taken into consideration.

New Lifestyles, Yamax, and Accusplit are three companies selling reliable pedometers. The CMES should choose pedometers that have been validated to accurately measure steps, are inexpensive, and have step filters, such as the Accusplit AX 2720 and 120 XLM series pedometers. Step filters are built into the pedometer's electronics and reduce

the recording of spontaneous and fidgety movements. These Yamax-Digiwalker and Accusplit pedometers have been used in many studies and are generally inexpensive and well-engineered. Most reliable pedometers have relatively long-term memories (i.e., \geq 99,999 step memory does not need resetting more than once a week or even once a month in some models). The 2720 and XLE models also record the number of minutes over the course of a day that the individual is actively moving. The interested CMESs can obtain a practical detailed instruction document with references (LaForge, 2013).

Figure 7-6 illustrates a formal method of creating pedometry programs for those with CHD by using Rx forms to both instruct and demonstrate the formality of the systematic clinical use of pedometers. It is important to note that this is an example of a form that a client would receive from a physician or clinician, not from the CMES.

EXPAND YOUR KNOWLEDGE

Figure 7-6

Sample outpatient pedometry prescription form for CHD clients. It is important to note that this is an example of a form that a client would receive from a physician or clinician, not from the CMES.

RX for Outpatient Pedometry

Patient name:	Date:
Therapuetic code:	
Order for following patient physica	al activity pedometer:
Dedometer: e.g., AX 2720 pedo	meter
Rx: steps/day steps/week	/month:/
Other Rx:	
Patient instructions: See attached physical activity and pedometer guidelines	
Referring Certified Medical Exercise Specialist	

Exercise Intensity

Exercise intensity or exercise workload is perhaps the most important and modifiable component of the exercise program. Work intensity most directly relates to the workload placed on the heart and the coronary arteries. Exercise speed, movement velocity, and resistance load all increase the workload of the heart, primarily through increased HR and BP. The two most practical intensity-monitoring strategies for the CMES are the client's volitional response to the exercise workload [e.g., **rating of perceived exertion (RPE)**] and exercise HR. In some cases, HR response may be blunted due to medications (e.g., beta blockers). Recognizing signs and symptoms of excessive physical effort is essential whether the individual is performing yard work or engaging in structured fitness activity (Table 7-11).

TABLE 7-11

SIGNS AND SYMPTOMS OF EXCESSIVE EFFORT IN CORONARY HEART DISEASE (CHD) PATIENTS

- · Chest discomfort or chest pain that intensifies with increasing physical effort
- Dyspnea (excessive shortness of breath) at relatively low work levels
- Palpitations (e.g., palpable ventricular arrhythmias)
- A drop in systolic blood pressure with increasing work loads
- Inappropriate heart-rate response to exercise (chronotropic incompetence)
- Sudden onset fatigue at rest or low-levels of physical activity

The CMES should understand the principles of cardiorespiratory endurance exercise programming, particularly the relationship between **heart-rate reserve (HRR)** and $\mathbf{\dot{V}O_2}$ reserve ($\mathbf{\dot{V}O_2}\mathbf{R}$) (i.e., HRR is more closely aligned with percent $\mathbf{\dot{V}O_2}R$ versus percent $\mathbf{\dot{V}O_2}$ max and this is true for nearly all conventional forms of aerobic exercise, including stationary exercise machine work) (ACSM, 2022; Dalleck & Kravitz, 2006).

ACSM (2022) recommends that the *initial* stages of aerobic-conditioning cardiac rehabilitation programs for low-risk and stable CHD clients have an exercise intensity of 20 (for patients with myocardial infarction) to 30 (for patients recovering from heart surgery) beats per minute above seated or standing resting heart rate to a maximum of ≤ 120 beats per minute. This corresponds to an RPE of \leq 13 on the 6 to 20 scale. However, it should be assumed that in most cases the CMES will be working with clients who are not in the early phases of cardiac rehabilitation (e.g., two to four weeks post-MI or -hospitalization), but who are in the improvement or maintenance stage of conditioning, in which case a moderate to vigorous intensity is used, ranging from 40 to 80% of HRR and $\dot{V}O_{a}R$ is more appropriate. The majority of work in this stage should be in the 45 to 65% VO₂R range, with higher intensities reserved for those who have safely and asymptomatically performed at the lower range. The majority of CHD clients will do well with 20 to 60 minutes of exercise per session. For durations of 45 minutes or longer, exercise intensity should be in the moderate range (i.e., 40 to 59% of HRR and $\dot{V}O_2R$). It is important to understand that some CHD clients fail to achieve predicted maximal heart rates even in the absence of medications that reduce HR (e.g., beta-blocking agents). This phenomenon is known as **chronotropic incompetence**. These individuals are at higher risk for CVD complications and are not within the CMES scope of practice.

A good but simple estimate of cardiac workload intensity can be determined using the following formula:

The Double Product

Myocardial (heart muscle) work = Heart rate (in beats per minute) x Systolic blood pressure (mmHg) / 100 For example: 150 beats/minute x 150 mmHg/100 = 225 This expression is known as the **double product**, but is also sometimes referred to as the **rate-pressure product** and often corresponds to the threshold for cardiac-related symptoms (e.g., angina). Intensive aerobic activities significantly increase HR but moderately increase **systolic blood pressure (SBP)**, whereas intensive resistance workloads (e.g., resistance training) moderately increase HR but cause a more significant rise in SBP. Both forms of exercise can dramatically raise the double product and therefore increase cardiac workload. Cardiac symptoms and heart-muscle dysfunction are directly related to exertional HR and BP. It is not practical for the CMES to calculate the double product for each exercise session, but they should thoroughly understand the consequences of various aerobic, resistance, and even mindful exercise modalities (e.g., hatha yoga styles and **Pilates**) and how they influence cardiac work.

It is important to understand which physical activities and exercises can rapidly increase SBP. For example, during heavy resistance exercise where an individual is exerting at \geq 80% of maximal voluntary contraction (MVC) levels, SBP increases quickly along with the **diastolic blood pressure (DBP)**. Additionally, when a person exerts to muscular failure during resistance exercise (which is not recommended), the CMES can assume a peak or near-peak BP response. Even for individuals with relatively stable CHD, this level of arterial pressure (also called "afterload"), which the heart has to work against, can be dramatic and deserves serious caution.

Exercise Frequency and Duration

Exercise should be performed at least every other day, but preferably on most days of the week. Frequency of exercise depends on several factors including disease status, baseline exercise tolerance, exercise intensity, musculoskeletal health, and other health goals. For clients with very limited exercise capacities, multiple short (1- to 10-minute) daily sessions may be appropriate.

Exercise duration for most CHD clients in the maintenance stage of conditioning is usually set between 30 and 60 minutes per day. However, as mentioned previously, many clients will require 60 or more minutes per day to adequately manage body weight, **dyslipidemia**, and associated risk factors. CHD, diabetes, and metabolic syndrome clients should have exercise programming dosed by daily or weekly energy expenditure rather than separately quantifying only frequency, intensity, and duration. The total accumulated energy expenditure of the exercise sessions is perhaps the single most important program feature associated with risk-factor reduction.

In most cases, the CHD client will require an activity program that uses *at least* 1,000 kcal/week but preferably (at least eventually) 2,000 kcal or more per week. In the case of a CHD client with the metabolic syndrome, **atherogenic dyslipidemia**, and obesity, the physical-activity program's energy expenditure should be \geq 2,000 kcal per week (gross kcal cost) to meaningfully alter these risk factors. Of course, these energy expenditures are a function of activity mode, frequency, duration, and intensity, and therein lies an opportunity for the CMES and the client to work together to design a creative and productive activity program. Once again, to constructively do this, the CMES will need a good working knowledge of the energy costs of a broad range of physical activities (Ainsworth et al., 2011).

Mode of Exercise for CHD

As with apparently healthy adults, cardiorespiratory endurance exercise is the principal focus of exercise programming for those with CHD, and should include rhythmic, large muscle group activities with an emphasis on increased caloric expenditure for maintenance of a healthy body weight and its many other associated health benefits. Cardiorespiratory endurance exercise programs should promote whole-body physical activity, feature conditioning that includes the upper and lower extremities, and include multiple forms of aerobic activities and exercise equipment.

Stationary Exercise Machines and CHD Exercise Training

Stationary exercise machines (e.g., treadmills, cycles, and elliptical machines) all provide a reasonable cardiorespiratory endurance stimulus for the CHD client. Remember that in the case of motor-driven treadmills, the "motivation" to keep up with high workloads is provided by the self-selected speed and grade, which can be an incentive for some clients and problematic in others who attempt to keep pace with workloads beyond their work capacity. Three considerations should be kept in mind for advising stationary equipment exercise:

- Use a fan for cross-ventilation and to help with evaporative heat loss, which can help reduce cardiac work (i.e., the double-product).
- Some stationary cycles generate a significantly higher blood pressure response than others, particularly in deconditioned adults (Kim, Chun, & Kim, 2013).
- Always warm up and cool down with lower speeds and resistances, which is particularly important in those who have active CHD.

The different types of exercise equipment may include the following:

- Arm ergometer
- Combination of upper and lower (dual action) extremity cycle ergometer
- Upright and recumbent cycle ergometer
- Recumbent stepper
- Rower
- Elliptical
- Stair climber
- Treadmill for walking

Interval/Intermittent Aerobic Exercise Training

Early stages of CHD client training should appropriately use intermittent bouts of aerobic training and progressively elongate the time so that the client can eventually maintain 30 or more minutes of continuous exercise without symptoms. In recent years, there have been programs that have judiciously and cautiously incorporated higher-intensity aerobic training, including high-intensity interval training (HIIT) in stable CHD patients with reasonable improvements in aerobic power without an increase in cardiovascular complications (Rognmo et al., 2012; Guiraud et al., 2011). HIIT, for example, involves alternating three-to four-minute periods of exercise at high intensity (80 to 90% HRR) with exercise at a lower intensity (60 to 70% HRR). Such training for approximately 40 minutes, three times per week has been shown to increase $\dot{V}O_2$ peak in patients with HF and produce greater long-term improvements in $\dot{V}O_2$ peak in patients after CABG compared to standard continuous moderate-intensity exercise (Guiraud et al., 2011). The CMES should be well acquainted with the relationships among $\dot{V}O_2$ max, $\dot{V}O_2$ peak, $\dot{V}O_2$ R, and HRR, particularly when advising and monitoring exercise intensities.

Understanding VO2max, VO2peak, VO2R, and HRR Relationships

 $\dot{V}O_2$ max = Highest personal oxygen uptake capacity possible $\dot{V}O_2$ peak = Highest value of oxygen uptake attained (measured or estimated) in an exercise test $\dot{V}O_2$ reserve ($\dot{V}O_2R$) = The difference between $\dot{V}O_2$ max and $\dot{V}O_2$ (% $\dot{V}O_2R \approx$ % Heart-rate reserve) Heart-rate reserve = The difference between maximal and resting heart rate

HIIT should not be used as an initial training stimulus, particularly in the first eight to 10 weeks of CHD exercise training. The CMES is strongly advised to ensure that the client has a preprogram negative exercise ECG test with physician clearance and consider the aforementioned recommendations on CHD exercise training, particularly the information discussed in Tables 7-7, 7-9 and 7-11.

EXERCISE GUIDELINES FOR STABLE ANGINA

The CMES should ensure that any client with exercise-induced angina or angina equivalent (e.g., reproducible shortness of breath or extreme fatigue) are medically cleared and are stable. Such clients would not be specifically targeted by the CMES but may occasionally be referred by a physician or a discharge referral after completing formalized cardiac rehabilitation. Angina is a primary manifestation of **myocardial ischemia** (i.e., insufficient blood supply to the heart muscle) and is relatively high risk within the category of all CHD patients. Any individual who experiences angina with physical workloads \leq 3 METs (i.e., low physical exertion levels such as walking at 2 mph) should not be trained by the CMES.

- Progressive aerobic endurance exercise is recommended, as long as it is within the individual's exercise tolerance as indicated by the most recent exercise ECG, or is just below the **anginal threshold** or physician-recommended percent of VO₂max.
- Intermittent, shorter-duration exercise on a more frequent basis [e.g., three to five sets of five- to 10-minutes of low- to moderate-intensity aerobic exercise bouts (e.g., cycling or treadmill walking)] may be most appropriate in the initial stages of training. Upper-extremity aerobic training (e.g., rowing or arm cranking) may initially exacerbate angina because of a higher HR and/or SBP.
- Avoid breath holding, isometric exercises, or activities where the individual physically exerts to muscular contraction failure (i.e., very high-resistance exercise).
- Keep close observation of anginal symptoms and ensure that the individual understands when to take anginaresolving medications (e.g., nitroglycerin, such as nitrostat). Instances when the client uses angina-resolving medications should be documented for the client's physician.

Nitroglycerin Administration

Nitroglycerin should be administered by the individual exactly as directed by their physician. Nitroglycerin is available as two types of products that are used for different reasons. The extended-release capsules are used every day on a specific schedule to prevent angina attacks. The oral spray and sublingual tablets work quickly to stop an angina attack that has already started or they can be used to prevent angina if the person plans to exercise or expects a stressful event.

For clients experiencing angina pectoris and who have been prescribed nitroglycerin PRN (as needed), the typical protocol is as follows:

- Discontinue activity and incorporate rest to see if chest discomfort/pain resolves on its own.
- If there is no relief, the client will self-administer one dose of nitroglycerin, either in tablet or spray form. A tablet will be placed sublingually or between the cheek and gums. If a spray is used, it would be delivered in the same locations.
- The client will then wait five minutes to see if the chest pain is resolved. If not, a second dose will be
 administered. They will then wait five more minutes and then repeat one more time before calling 9-1-1.
 Nitroglycerin is a vasodilator and dilates coronary arteries by relaxing vascular smooth muscle, allowing for a greater

oxygen delivery to the heart. Side effects of nitroglycerin administration include severe headache and a drop in BP.

Do you have a plan in the event of an acute onset of angina while training a client who has been diagnosed with stable angina? Visualize the steps you would take during a training session if a client expresses the onset of angina pain, including what you would say to the individual and how you would encourage them to take nitroglycerin medication as prescribed by their physician. As the occurrence of anginal episodes that require the administration of medication should be documented and reported to the client's physician, think about how you will do this (e.g., create a specialized form and phone the physician's office for further direction).

Resistance Training

Resistance-training modalities can clearly improve the client's muscular fitness and functionality. Because there are so many types of resistance training and associated protocols, certain precautions are important to note. The primary consideration for the CMES in this context is the amount and rate of force delivered to the client's muscles relative to their capacity and cardiac ventricular function. Table 7-12 denotes criteria for the resistance training of CHD clients. In this



EXPAND YOUR KNOWLEDGE

instance, resistance training applies to the use of free weights, machines, or other resistive devices that deliver a resistive force \geq 40% of the client's MVC. As long as the client is not straining to concentrically or eccentrically contract a muscle group or performing breath-holding, resistance training can be very beneficial. The contraindications for resistance training, as well as more intense forms of hatha yoga (e.g., Bikram or "hot" yoga and Ashtanga or "power" yoga), are essentially the same (see Table 7-9). There are no contraindications to Pilates exercises, provided that the same guidelines are adhered to as previously stated. One caution, however, would be to avoid resistance-training regimens in clients who have maximal exercise capacities of <6 METs. The CMES should not place themself in the position to recognize these clinical contraindications, but should ensure physician clearance for these contraindications.

TABLE 7-12

CRITERIA FOR RESISTANCE-TRAINING PROGRAMS*

- Low- to moderate-risk clients and possibly higher-risk clients with supervision
- Those who require strength for work or recreational activities, particularly in their upper extremities
- Initiate a minimum of 6 to 10 weeks after date of myocardial infarction or cardiac surgery, including 4 weeks of consistent participation in a supervised cardiac rehabilitation endurance training program
- Initiate a minimum of 3 weeks following transcatheter procedure (i.e., PCI or other), including 2 weeks of consistent participation in a supervised cardiac rehabilitation endurance training program
- No evidence of acute congestive heart failure, uncontrolled dysrhythmias, severe valvular disease, uncontrolled hypertension, and unstable symptoms

*In this table, a resistance-training program is defined as one in which clients lift weight >50% of one-repetition maximum (1-RM). The use of elastic bands, 1- to 3-lb (0.45–1.36 kg) hand weights, and light free weights may be initiated in a progressive fashion at outpatient program entry provided no other contraindications exist.

Note: PCI = Percutaneous coronary intervention

Source: American Association of Cardiovascular and Pulmonary Rehabilitation (2021). *Guidelines for Cardiac Rehabilitation Programs* (6th ed.). Champaign, III.: Human Kinetics..

Recommendations for the Initial Resistance-training Program

While BP certainly may rise excessively during resistance training, the actual rise depends on a variety of controllable factors, including magnitude of the isometric component, the load intensity, the amount of muscle mass involved, and the number of repetitions and/or the load duration. Intraarterial BP measurements in cardiac patients have demonstrated that during low-intensity resistance training (40 to 60% MVC) with 15 to 20 repetitions, only modest elevations in BP are revealed, similar to those seen during moderate-intensity endurance training (Bjarnason-Wehrens et al., 2004). For CHD patients, the use of elastic bands and/or small weights is very suitable. More precise training with less risk of overloading can be achieved through the use of training machines, whereby the load dose can be individually adjusted and the execution of the movement is predetermined.

Mindful Exercise and the CHD Client

Select forms and styles of mindful exercise modalities are most often appropriate or can be easily adapted for clients with CHD. This is particularly true for yogic-based breathwork therapy (Pal et al., 2014). Table 7-13 provides examples of forms of mindful exercise that are appropriate for the majority of individuals with CHD.

Mindful exercise programs can range from those requiring very low energy expenditure and deep relaxation qualities to those that require considerable muscular strength and impose considerable myocardial work. Thus, several considerations are important when choosing

particular mindful exercise modalities. Many styles of hatha yoga, for example, involve acute dynamic changes in body position (i.e., the relationship of the head, chest, and lower limbs to each other). It is therefore important to fundamentally understand the **hemodynamic** and cardiac ventricular responses to such exercise and how these may alter cardiac function in individuals with CHD, including clients with hypertension, metabolic syndrome, or diabetes.

Inverted poses where the head is below the heart (e.g., downward facing dog or headstands), or situations in which such a position is alternated with a "head-up" pose, should be avoided. In most cases, those who are initially deconditioned and/or have CHD should minimize acute changes in body position that require the head to be below the level of the heart in early stages of hatha yoga training and use slower transitions from one yoga pose to the next. Because Ashtanga and traditional lyengar yoga poses and sequences generally require considerable strength, flexibility, and mental concentration, they should be reserved for higher-functioning individuals (i.e., clients with a >10 MET exercise capacity). Some yoga poses significantly increase BP and may also be inappropriate for older adults with stage II or higher hypertension (i.e., BP ≥160/100 mmHg). One study on intermediate and advanced yoga practitioners showed that some lyengar poses can rapidly and significantly increase mean and peak systolic BP particularly with back arch poses (Blank, 2006). Systolic, mean, and diastolic BP can increase significantly during yoga practice. At least one report has indicated that the magnitude of these increases in BP was greatest with standing postures (Miles et al., 2013). This should not be a deterrent to stable CHD clients safely practicing various hatha yoga asanas. The CMES should be aware, however, that this level of BP can impose significant double-product stress on the heart of some CHD clients, particularly if the stress is a sudden increase in SBP rather than the gradual workload increase seen with graduated aerobic exercise work levels. It is strongly recommended that the CMES start the client with restorative yoga poses prior to engaging in a full complement of lyengar or equivalent yoga poses.

TABLE 7-13

EXAMPLES OF APPROPRIATE FORMS OF MINDFUL EXERCISE FOR CLIENTS WITH CHD	
 Restorative yoga Kripalu yoga Viniyoga Integral yoga Select lyengar and yoga poses Tai chi chuan (moderate pace) 	 Tai chi chih Meditation walking Chi walking Yogic breathwork Pilates mat and reformer work NIA at low-to-moderate level

Note: NIA = Neuromuscular integrative action

YOGIC BREATHING

Perhaps most useful in those with any level of CHD is yogic breathing. Although there are many styles of yogic breathing, the breath is generally drawn through the nose during both inhalation and exhalation. Each breath is intentionally slow and deep with an even distribution, or smoothness, of effort. Lengthening exhalations by using the abdominal muscles to expire more air while breathing through the nose will cause a relaxation response. In addition to reduced stress and mental tension, cardiovascular benefits result from yogic breathing. One of the mechanisms responsible for the mental quiescence experienced with yogic breathing is its stimulation of the **parasympathetic nervous system**. When fully stimulated by adequate yogic inspiration and expiration, mechanical receptors in pulmonary tissue (e.g., alveoli) activate parasympathetic nerves, which transiently reduces mental tension and elicits a relaxation response (Pal, Velkumary, & Madanmohan, 2004). A suitable inhalation/exhalation ratio is to inhale for a 2-count, exhale for a 4-count, and then work up to inhaling for an 8-count and exhaling for a 16-count. To test this relaxation response, the client can feel their pulse during this breathing exercise. They may notice a reduced pulse rate with prolonged exhalation. This slight slowing of HR includes a reciprocal slight increase in HR variability—a process called respiratory sinus arrhythmia. Acute reductions in BP also have resulted from yogic breathing training (Pal et al., 2014; Adhana et al., 2013).

APPLY WHAT YOU KNOW

THE 2012 ACCF PHYSICAL-ACTIVITY RECOMMENDATIONS FOR CHD PATIENTS EXPAND YOUR In 2012, the American College of Cardiology Foundation, in collaboration with six other health care KNOWLEDGE organizations (ACCF/AHA/ACP/AATS/PCNA/SCAI/STS, 2012), advanced their recommendations for physical activity in patients with CHD. Although these are quite generalized, they are in synergy with ACSM and AACVPR's CHD guidelines. The ACCF CHD physical-activity guidelines are summarized as follows: • For all CHD patients, the CMES should encourage 30 to 60 minutes of moderate-intensity aerobic activity, such as brisk walking, at least five days and preferably seven days per week, supplemented by an increase in daily lifestyle activities (e.g., walking breaks at work, gardening, and household work) to improve cardiorespiratory fitness and move clients out of the least-fit, least-active, high-risk cohort (bottom 20%). For all clients, risk assessment with a physical-activity history and/or an exercise test is recommended to guide exercise programming. • Medically supervised programs (cardiac rehabilitation) and physician-directed, home-based programs are recommended for at-risk patients at first diagnosis. **EXERCISE** Given the aforementioned synergy of physical-activity guidelines for individuals with CHD, the following summarizes the recommendations made in this chapter: **GUIDELINES** CARDIORESPIRATORY TRAINING **SUMMARY FOR** • At least 3, but preferably up to 5 days of the week Frequency **CLIENTS WITH** Current guidelines suggest that any amount of exercise is better than none. Moderate to vigorous intensity* Intensity CARDIOVASCULAR • Intensity may be determined through the following methods: DISEASE ✓ With an exercise test, use 40–80% HRR or VO, R or VO, peak \checkmark Without an exercise test, use RPE of 12–16 (6–20 scale) or add 20–30 bpm to RHR ✓ HR should remain at least 10 bpm below the HR associated with the ischemic threshold (if exercise ischemic threshold has been determined) High-intensity interval training may be a safe and effective method for enhancing cardiorespiratory fitness for individuals with stable disease and a base level of conditioning. Time Eventual goal of 20–60 minutes for cardiorespiratory training • Warm-up and cool-down activities lasting 5–10 minutes should be included in each exercise session. Rhythmic, large muscle group exercise that emphasizes increased caloric expenditure Type and utilizes multiple activities and pieces of equipment, such as: ✓ Arm ergometer ✓ Upright and recumbent cycle ergometer ✓ Recumbent stepper ✓ Rower ✓ Elliptical ✓ Treadmill for walking • Progress following the ACE Integrated Fitness Training[®] Model based on client goals and Progression availability. Sessions may include continuous or intermittent exercise.

MUSCULAR TRAINING	
Frequency	• 2–3 days per week with a minimum of 48 hours separating exercise for the same muscle group
Intensity	 40–60% 1-RM, or a load that can be lifted 10–15 repetitions without straining RPE of 11–13 (6–20 scale)
Time	• 1–3 sets of 8–10 different exercises focused on major muscle groups
Туре	 Select equipment that is safe and comfortable, including: ✓ Elastic resistance ✓ Free weights ✓ Pulleys ✓ Selectorized machines Each major muscle group should be trained initially with one set Multiple-set routines may be introduced later, as tolerated
Progression	 Progress following the ACE Integrated Fitness Training Model based on client goals and availability. Progression can be introduced through increases in resistance, number of repetitions or sets, or decreasing rest periods between sets. Progression should be slow and dependent on tolerance. ✓ Volume can be increased 2–10% once clients comfortably complete 1–2 repetitions beyond the target range on two consecutive training sessions.

*Moderate intensity = Heart rates <VT1 where speech remains comfortable and is not affected by breathing; Vigorous intensity = Heart rates from \geq VT1 to <VT2 where clients feel unsure if speech is comfortable.

Note: HRR = Heart-rate reserve; $\dot{V}0_2R$ = Oxygen uptake reserve; RPE = Rating of perceived exertion; bpm = Beats per minute; HR = Heart rate; RHR = Resting heart rate; 1-RM = One-repetition maximum; VT1 = First ventilatory threshold; VT2 = Second ventilatory threshold

CMES FOCUS

Aerobic exercise and resistance training are safe and effective for most clients with CHD. However, all clients with CHD should be clinically evaluated and classified according to future risk for the occurrence of cardiac-related events during exercise. Exercise programs will be highly individualized and dependent on the client's unique needs and exercise tolerance.

Source: American College of Sports Medicine (2022). ACSM's Guidelines for Exercise Testing and Prescription (11th ed.). Philadelphia: Wolters Kluwer.

CASE STUDY

Client Information

Jerome is a 59-year-old engineer who is sedentary and has a history of CHD, dyslipidemia (LDL cholesterol = 182 mg/dL, HDL cholesterol = 39 mg/dL, triglycerides = 175 mg/dL), and stage I hypertension (162/100 mmHg). He is referred to the CMES by an internist. His CHD was documented after a positive treadmill ECG and an angiogram confirming two-vessel coronary disease. Jerome has not had an MI or significant history of angina and had no reportable angina-like symptoms on his treadmill ECG. His treadmill ECG was positive

beginning at a HR of 155 to 160 bpm and he was capable of achieving an 11 MET exercise capacity. After prescribing a beta blocker, a statin, and an ACE inhibitor for his risk factors, Jerome's doctor decided to refer him to a CMES rather than a formalized cardiac rehabilitation program. Up to this point, the patient was minimally physically active, walking the equivalent of 1 mile a day. Upon referral to the CMES, his baseline numbers are as follows (on his new medications):

- Weight: 190 lb (86 kg)
- Height: 5'9" (1.75 m)
- Body mass index (BMI): 29 kg/m²
- Waist circumference: 39 inches (99 cm)
- LDL cholesterol: 98 mg/dL
- HDL cholesterol: 44 mg/dL
- Triglycerides: 129 mg/dL
- BP: 134/84 mmHg
- Exercise capacity: 11 METs

Medications: Carvedilol (6.25 mg), rosuvastatin (20 mg), Lisinopril (10 mg), and sublingual nitroglycerin if needed

CMES Approach

Exercise plan: The initial exercise plan would build on the client's current activity of walking 1 mile per day, progressing his home walking program to 2 miles per day performed five or more days per week. In addition, the CMES adds the following: 1,000 kcal/week of a combination of three sessions/week of elliptical trainer exercise (starting with two 10-minute sessions at low-to-moderate level stride rate and progressing to one continuous 30-minute session over a six-week period, keeping exercise HR between 130 and 145 bpm) to expend 1,000 kcal/week for the three sessions combined. An effort is made to ensure that he is remaining compliant with his medications and the Healthy Mediterranean-Style Dietary Pattern. Total weekly physical activity energy-expenditure goal is 1,900 to 2,000 kcal.

Follow-up: At three months, he has lost 7 pounds (3 kg) and his follow-up labs are as follows:

- Weight: 183 lb (83 kg)
- BMI: 27.5 kg/m²
- Waist circumference: 38 inches (96 cm)
- LDL cholesterol: 92 mg/dL
- HDL cholesterol: 51 mg/dL
- Triglycerides: 118 mg/dL
- BP: 130/84 mmHg
- Exercise capacity: 13.5 METs

One 60-minute variable-terrain walk per week was added, along with two circuit resistance-training sessions/week (one or two sets of 10 exercises at 15 RM) focused on proper movement patterns and exercises using body weight and light resistance to build muscular endurance. Total weekly energy expenditure, including existing walking/elliptical program, is now 2,500 to 2,700 kcal.

This client's six-month goals are as follows:

- Weight: <165 lb (74 kg)
- BMI: <26 kg/m²
- Waist circumference: <35 inches (88 cm)
- LDL cholesterol: <70 mg/dL

ACE IFT[®] MODEL AT A GLANCE

Cardiorespiratory Training

This client's initial exercise program is focused on consistently performing moderate-intensity cardiorespiratory exercise in the Base Training phase.

ACE IFT[®] MODEL AT A GLANCE

Cardiorespiratory Training

At three months, the client has progressed to the Fitness Training phase.

Muscular Training

He is also performing a circuit resistance-training workout that is focused primarily on Movement Training.

- HDL cholesterol: >50 mg/dL
- Triglycerides: <150 mg/dL
- Exercise capacity: >15 METs

In the absence of significant further reduction in LDL cholesterol, his physician may choose to increase the rosuvastatin dose to 40 mg. Note that with documented CHD, his ideal therapeutic LDL cholesterol goal is 70 mg/dL. Intensive statin therapy (i.e., high-dose rosuvastatin or atorvastatin) (ACC/AHA, 2013; International Atherosclerosis Society, 2017) may also need to be implemented.

SUMMARY

Exercise therapy for the prevention and treatment of CHD works well beyond its moderate lipid-lowering effects by improving functional capacity, antioxidant defenses, arterial endothelial function, insulin sensitization, glucose transport, fibrinolytic capacity, and psychological well-being, and by reducing BP and body-fat stores. Ideally, the CHD client would have completed early phase cardiac rehabilitation (i.e., phase I and II cardiac rehabilitation) prior to working with the CMES. An experienced CMES who wishes to work with CHD clients should work only with those who are under a physician's care and who have stable coronary disease. To optimize the potential for improvement of CHD risk factors and stabilize the disease process, clients should achieve a total volume of moderate physical activity of 1,500 to 2,000 kcal or more per week. This volume includes systematic workouts (including aerobic, resistance, and select mindful exercise training), recreational activities, and activities of daily living.

REFERENCES

Academy of Nutrition and Dietetics Evidence Analysis Library 1 (2013a). *Disorders of Lipid Metabolism Evidence Analysis Project*. Retrieved November 10, 2013. <u>http://andevidencelibrary.com/topic.cfm?cat=4527</u>

Academy of Nutrition and Dietetics Evidence Analysis Library 2 (2013b). *Heart Failure Evidence-based Nutrition Practice Guidelines*. Retrieved November 10, 2013. <u>http://andevidencelibrary.com/topic.cfm?cat=4527</u>

ACCF/AHA/ACP/AATS/PCNA/SCAI/STS (2012). Guideline for the diagnosis and management of patients with stable ischemic heart disease: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, and the American College of Physicians, American Association for Thoracic Surgery, Preventive Cardiovascular Nurses Association, Society for Stephan D. Fihn, MD, MPH, Chair. *Circulation*, 126, e354–e471.

Adhana, R. et al. (2013). The influence of the 2:1 yogic breathing technique on essential hypertension. *Indian Journal of Physiology and Pharmacology*, 57, 1, 38–44.

Ainsworth, B.E. et al. (2011). (2011), Compendium of physical activities: A second update of codes and MET values. *Medicine & Science in Sports & Exercise*, 43, 8, 1575–1581.

American Association of Cardiovascular and Pulmonary Rehabilitation (2021). *Guidelines for Cardiac Rehabilitation Programs* (6th ed.). Champaign, III.: Human Kinetics.

American College of Cardiology/American Heart Association (2013). Guidelines on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*, published online November 12, 2013.

American College of Sports Medicine (2022). ACSM's Guidelines for Exercise Testing and Prescription (11th ed.). Philadelphia: Wolters Kluwer.

American Council on Exercise (2020). *The Exercise Professional's Guide to Personal Training.* San Diego, Calif.: American Council on Exercise.

American Heart Association (2014). AHA statistical update: Heart disease and stroke statistics—2014 update: A report from the American Heart Association. *Circulation*, published online December 18, 2013. <u>http://circ.ahajournals.org/content/early/2013/12/18/01.cir.0000441139.02102.80.citation</u>

Balady, G.J. et al. (2007). 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*, 115, 2675–2682.

Bjarnason-Wehrens, B. et al. (2004). Recommendations for resistance exercise in cardiac rehabilitation: Recommendations of the German Federation for Cardiovascular Prevention and Rehabilitation. *European Journal of Cardiovascular Prevention and Rehabilitation*, 11, 4, 352–361.

Blank, S. (2006). Physiological responses to lyengar yoga poses performed by trained practitioners. *Journal of Exercise Physiology*, 9, 7–23.

Boden, W.E. et al. (2007). Optimal medical therapy with or without PCI for stable coronary disease. *New England Journal of Medicine*, 12, 356, 1503–1516.

Centers for Disease Control and Prevention (2013). Surveillance for certain health behaviors among states and selected local areas—United States, 2010. *Morbidity and Mortality Weekly Report: Surveillance Summaries*, 62(ss01), 1–247.

Cook, N.R. et al. (2007). A randomized factorial trial of vitamins C and E and beta carotene in the secondary prevention of cardiovascular events in women: Results from the Women's Antioxidant Cardiovascular Study. *Archives of Internal Medicine*, 167, 15, 1610–1618.

Dalleck, L.C. & Kravitz, L. (2006). Relationship between percent heart rate reserve and percent VO₂ reserve during elliptical crosstrainer exercise. *Journal of Sports Science and Medicine*, 25, 662–671.

de Lorgeril, M. et al. (1999). Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: Final report of the Lyon Diet Heart Study. *Circulation*, 99, 779–785.

Donnelly, J.E. et al. (2009). Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Medicine & Science in Sports & Exercise*, 41, 2, 459–471.

Eckel, R.H. et al. (2013). 2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Journal of the American College of Cardiology*, 63, 25. DOI: 10.1016/j.jacc.2013.11.003

Gibbons, R. et al. (2002). ACC/AHA 2002 guideline update for exercise testing: Summary article—A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*, 106, 1883–1892.

Gidding, S. et al. (2009). Implementing American Heart Association Pediatric and Adult Nutrition Guidelines: A Scientific Statement from the American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity and Metabolism, Council on Cardiovascular Disease in the Young, Council on Arteriosclerosis, Thrombosis and Vascular Biology, Council on Cardiovascular Nursing, Council on Epidemiology and Prevention, and Council for High Blood Pressure Research. *Circulation*, 119, 1161–1175.

Goldman, L. et al. (1981). Comparative reproducibility and validity of systems for assessing cardiovascular functional class: Advantages of a new specific activity scale. *Circulation*, 64, 1227–1234.

Greenland, P. et al. (2004). Coronary artery calcium score combined with Framingham score for risk prediction in asymptomatic individuals. *Journal of the American Medical Association*, 291, 210–215.

Guiraud, T. et al. (2011). Acute responses to high-intensity intermittent exercise in CHD patients. *Medicine & Science in Sports & Exercise*, 43, 2, 211–217.

Harris, W.S. (2010). Omega-6 and omega-3 fatty acids: Partners in prevention. *Current Opinions in Clinical Nutrition and Metabolic Care*, 13, 2, 125–129.

Hoevenaar-Blom, M.P. et al. (2012). Mediterranean style diet and 12-year incidence of cardiovascular diseases: The EPIC-NL cohort study. *PloS One*, 7, 9, e45458.

Houle, J. et al. (2013). Daily steps threshold to improve cardiovascular disease risk factors during the year after an acute coronary syndrome. *Journal of Cardiopulmonary Rehabilitation and Prevention*, 33, 6, 406–410.

Howley, E. (2012). Energy costs of physical activity. In: Howley, E. & Franks, B.D. (Eds.) *Health and Fitness Instructor's Manual* (6th ed.). Champaign, III.: Human Kinetics.

Institute of Medicine, Food and Nutrition Board (2005). *Dietary Reference Intakes: Energy, Carbohydrates, Fiber, Fat, Fatty Acids, Cholesterol, Protein and Amino Acids.* Washington, D.C.: National Academies Press.

International Atherosclerosis Society (2017). An International Atherosclerosis Society Position Paper: Global Recommendations for the Management of Dyslipidemia—Full Report. www.lipidjournal.com/article/ S1933-2874(13)00354-1/fulltext Ismail, H. et al. (2013). Clinical outcomes and cardiovascular responses to different exercise training intensities in patients with heart failure: A systematic review and meta-analysis. *Journal of the American College of Cardiology*, 1, 6, 514–522.

Kenney, W.L., Wilmore, J.H., & Costill, D.L. (2012). *Physiology of Sport and Exercise* (5th ed.). Champaign, III.: Human Kinetics.

Kim, Y.J., Chun, H., & Kim, C.H. (2013). Exaggerated response of systolic blood pressure to cycle ergometer. *Annals of Rehabilitation Medicine*, 37, 3, 364–372.

LaForge, R. (2013). *Clinical Pedometry: A Brief Overview and Instructions for Health Care Providers*. Jacksonville, Fla.: National Lipid Association.

Lavie, C.J. et al. (2009). Exercise training and cardiac rehabilitation in primary and secondary prevention of coronary heart disease. *Mayo Clinic Proceedings*, *84*, 4, 373–383.

Libby, P. (2012). Inflammation in atherosclerosis. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 32, 2045–2051.

Lorenz, M.W. et al. (2007). Prediction of clinical cardiovascular events with carotid intima-media thickness: A systematic review and meta-analysis. *Circulation*, 115, 459–467.

Marshall, S.J. et al. (2009). Translating physical activity recommendations into a pedometer-based step goal: 3000 steps in 30 minutes. *American Journal of Preventive Medicine*, 36, 410.

Merchant, R.M. et al. (2020). 2020 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*, 142, 16, S337–S357.

Miles, S.C. et al. (2013). Arterial blood pressure and cardiovascular responses to yoga practice. *Alternative Therapies in Health and Medicine*, 19, 1, 38–45.

National Cholesterol Education Program (2002). *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. National Heart, Lung, and Blood Institute National Institutes of Health NIH Publication No. 02-5215.

Nordmann, A.J. et al. (2011). Meta-analysis comparing Mediterranean to low-fat diets for modification of cardiovascular risk factors. *American Journal of Medicine*, 124, 841–851, e842.

Ornish, D. et al. (1998). Intensive lifestyle changes for reversal of coronary heart disease. *Journal of the American Medical Association*, 280, 2001–2007.

Ornish, D. et al. (1990). Can lifestyle changes reverse coronary heart disease? Lancet, 336, 129-133.

Pal, G.K., Velkumary, S., & Madanmohan (2004). Effect of short-term practice of breathing exercises on autonomic functions in normal human volunteers. *Indian Journal of Medical Research*, 120, 115–121.

Pal, G.K. et al. (2014). Slow yogic breathing through right and left nostril influences sympathovagal balance, heart rate variability, and cardiovascular risks in young adults. *North American Journal of Medical Sciences*, 6, 3, 145–151.

Rognmo, Ø. et al. (2012). Cardiovascular risk of high- versus moderate-intensity aerobic exercise in coronary heart disease patients. *Circulation*, 126, 12, 1436–1440.

Sesso, H.D. et al. (2008). Vitamins E and C in the prevention of cardiovascular disease in men: The Physicians' Health Study II randomized controlled trial. *Journal of the American Medical Association*, 300, 18, 2123–2133.

Sofi, F. et al. (2010). Review accruing evidence on benefits of adherence to the Mediterranean diet on health: An updated systemic review and meta-analysis. *American Journal of Clinical Nutrition*, 92, 5, 1189–1196.

Trichopoulou, A. et al. (2003). Adherence to a Mediterranean diet and survival in a Greek population. *New England Journal of Medicine*, 348, 2599–2608.

Tudor-Locke, C. et al. (2011). Accelerometer steps/day translation of moderate-to-vigorous activity. *Preventive Medicine*, 53, 1–2, 31–33.

U.S. Department of Agriculture (2020). 2020-2025 Dietary Guidelines for Americans (9th ed.) www. dietaryguidelines.gov

U.S. Department of Health & Human Services (2018). *Physical Activity Guidelines for Americans* (2nd ed.). <u>www.</u> <u>health.gov/paguidelines/</u>

Williams, M.A. (2001). Exercise testing in cardiac rehabilitation: Exercise prescription and beyond. *Cardiology Clinics*, 19, 3, 415–431.

SUGGESTED READING

ACCF/AHA/ACP/AATS/PCNA/SCAI/STS (2012). Guideline for the diagnosis and management of patients with stable ischemic heart disease: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, and the American College of Physicians, American Association for Thoracic Surgery, Preventive Cardiovascular Nurses Association, Society for Stephan D. Fihn, MD, MPH, Chair. *Circulation*, 126, e354–e471.

American College of Cardiology/American Heart Association (2013). Guidelines on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*, published online November 12, 2013.

American Association of Cardiovascular and Pulmonary Rehabilitation (2021). *Guidelines for Cardiac Rehabilitation Programs* (6th ed.). Champaign, III.: Human Kinetics.

American College of Sports Medicine (2022). ACSM's Guidelines for Exercise Testing and Prescription (11th ed.). Philadelphia: Wolters Kluwer.

American Heart Association (2014). AHA statistical update: Heart disease and stroke statistics—2014 update: A report from the American Heart Association. *Circulation*, published online December 18, 2013. <u>http://circ.ahajournals.org/content/early/2013/12/18/01.cir.0000441139.02102.80.citation</u>