Part Two

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Coronary Artery Disease

Ralph La Forge

Adults with stable and well-managed cardiovascular disease (CVD) represent a key opportunity for competent and experienced personal trainers or ACE-certified Advanced Health & Fitness Specialists (ACE-AHFS). According to the American Heart Association (AHA), an estimated 80.7 million American adults have one or more types of cardiovascular disease. Cardiovascular disease includes the following disease states: hypertension (73 million cases in the United States); coronary artery disease (CAD), also known as coronary heart disease (CHD) (16 million); heart failure (5.3 million); and stroke (5.8 million) (AHA, 2007).

The purpose of this chapter is to briefly acquaint the ACE-AHFS with the CAD component of CVD, describe its process, and provide appropriate training recommendations. Hypertension, diabetes, obesity, and the metabolic syndrome, all of which impact the CAD process, are discussed elsewhere in this text. The ACE-AHFS is strongly encouraged to partner the information in this chapter with ACSM’s Guidelines on Exercise Testing and Prescription (8th edition) [American College of Sports Medicine (ACSM), 2010] and the ACE Clinical Exercise Specialist Manual (ACE, 2007), both of which are good resources for the ACE-AHFS who wishes to work with individuals with stable CAD. There is remarkable agreement between these and other guidelines in terms of the recommended quantity and quality of exercise for those with CAD, although there are some minor differences [American Association of Cardiovascular and Pulmonary Rehabilitation (AACVPR), 2003; ACSM, 2010; AHA/American College of Cardiology (ACC), 2006; ACE, 2007]. The ACE-AHFS should see this chapter as somewhat of a synthesis and update of these guidelines.

Fundamentally, the role of the ACE-AHFS who wishes to work with individuals with CAD is to work only with those clients who have stable CAD and are at low risk for exercise-related cardiovascular complications. Furthermore, the principal role of the ACE-AHFS in this context is to design and allocate appropriate and safe levels of physical activity to improve the client’s functionality, favorably modify CAD risk factors, and further improve the function of the heart. It is strongly recommended that the clients with whom the ACE-AHFS intends to work have successfully completed phase I and II cardiac rehabilitation by formalized outpatient cardiac rehabilitation programs when these programs are locally available. Phase I cardiac rehabilitation is the in-patient phase that includes teaching and low-level hospital-based ambulatory exercise, while Phase II is the early hospital discharge phase of rehabilitation and includes medically monitored exercise and more concentrated risk-factor education. Fewer than 30% of eligible patients receive phase II care, which would drastically narrow the population of individuals who could work with an ACE-AHFS. Therefore, the ACE-AHFS should target those individuals who have not gone through phase II rehabilitation, as well as those at risk for CAD.
Coronary Artery Disease

Coronary artery disease, also known as ischemic heart disease and atherosclerotic heart disease, 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). CAD results from the development of atherosclerosis in the coronary arteries. Atherosclerosis is a disease affecting 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 (Figure 6-1). 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 low-density lipoprotein (LDL) cholesterol in the arterial intima, 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 (inner-most 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, for example a myocardial infarction, is more often caused by rupture of a complicated and vulnerable atherosclerotic plaque than by a gradual closure of the coronary blood vessel.

Figure 6-1
The atherosclerotic process: Response to injury

Source: www.en.wikipedia.org/wiki/Atherosclerosis
The vulnerable plaque is essentially characterized by plaques 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 result in a clot or coronary thrombosis. The formation, progression, and rupture of the vulnerable plaque are viewed as a process related directly to inflammation.

**Principal Diagnostic Tests for CAD**

There are a number of reasons for a physician to order a battery of diagnostic tests to confirm or rule out cardiovascular disease. In many cases, those with mild to moderate CAD 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 his or her diagnostics. In some cases, the patient does not have any complaints signifying CAD, but due to his or her multiple risk factors, warrants a closer look (see Table 1-2, page 13). In addition to standard blood work assessing blood lipids, glucose, and inflammatory markers, diagnostic tests will be ordered to discover the likelihood of any clinically significant blockage.

**Electrocardiogram**

An electrocardiogram (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. Key waveforms of the ECG used for diagnostic purposes include the P wave, P-R interval, QRS complex, S-T segment, and T wave.

**ECG Exercise Testing**

Essentially, the exercise ECG test is a graded (gradual increase in speed and grade) exercise treadmill 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. 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 response, symptoms (angina, shortness of breath or dyspnea, or dizziness), and the exercise level achieved (e.g., metabolic equivalent (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 as a diagnostic tool as a nuclear stress test.

**Radionuclide Stress Test**

Radionuclide stress testing involves injecting a radioactive isotope (typically thallium or cardioylt) into the person’s vein, after which an image of the heart becomes visible with a special camera. The radioactive isotopes are absorbed by the normal 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 CAD.

**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 CAD, 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 CAD in patients suspected of having a “false-positive” stress ECG.

**Coronary Angiography (Cardiac Catheterization)**

Coronary angiography involves inserting a catheter into 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). This technique 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 intima-media thickness (CIMT), and cardiovascular magnetic resonance imaging (CMRI), has enabled the more practical non-invasive evaluation of atherosclerosis at a preclinical stage (see page 133).

### Appropriate Program Candidates and Stable CAD

The individual in need of a supervised cardiac prevention program may not have diagnosed CAD, but instead merely be interested in preventing heart disease (primary prevention). Candidates for this program may also include those with multiple risk factors for CAD, but may never have had a cardiac event (secondary prevention). Those with documented CAD or unstable CAD are most appropriate for formalized and supervised cardiac rehabilitation. Ideally, the CAD client would have completed phase I and II cardiac rehabilitation programs prior to working with the ACE-AHFS. An experienced ACE-AHFS who wishes to work with CAD clients should work only with those who are under a physician’s care and who have stable coronary disease. For purposes of the ACE-AHFS scope of practice, stable CAD means that the individual’s disease process is well managed (i.e., he or she does not have irregular, unpredictable symptoms, unstable angina, heart failure, or malignant ventricular arrhythmias). Stable also means that the individual is currently under the care of a physician and on appropriate medical therapy for his or her level of CAD. The ACE-AHFS is not expected to discriminate between stable and unstable CAD, 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 CAD is periodic exercise electrocardiographic stress testing (exercise ECG) by a physician.
Noninvasive Vascular Imaging Techniques

**Coronary CT Angiography**

In CT angiography, computed tomography using a contrast material produces detailed pictures. CT imaging uses special x-ray equipment to produce multiple images and a computer to join them together in cross-sectional views. This new test is available to diagnose CAD. 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 a little medication to slow the heart rate to less than 64, CT images of the coronary arteries are now possible.

**High-resolution Magnetic Resonance Imaging**

High-resolution cardiovascular magnetic resonance imaging (CMR) 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. 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.

**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 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). Currently, the primary methods for CAC measurement are electron-beam computed tomography (EBCT) and multi-detector computed tomography (MDCT).

**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 coronary artery disease. 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, 2007).
Fitness Assessments

Initial Assessment by the ACE-AHFS

Once the ACE-AHFS has received an appropriate referral for training a CAD client, the first step is to review the individual’s health history and lifestyle information. Both ACE and ACSM provide comprehensive advice regarding the infrastructure for developing necessary components and formats for this initial assessment (ACE, 2007; ACSM, 2010). The essential components of the pre-training assessment are as follows:

- Medical history
- Current medications
- Existing CAD risk factors (see Table 1-2, page 13)
- Symptoms
- Physical-activity history
- Dietary history

A Physical Activity Readiness Questionnaire (PAR-Q) evaluation is also recommended as part of the initial ACE-AHFS evaluation (see Figure 1-3, page 14).

Fitness Testing

The ACE-AHFS can administer most fitness tests to clients with stable CAD, provided that the client meets the overall considerations in Table 6-1 and has no contraindications to resistance exercise (see Table 6-5, page 140). 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 heart rate, or $\dot{V}O_2_{\max}$. Submaximum 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, dysrhythmias). The ACE-AHFS should adhere to the general procedures for submaximal testing of cardiorespiratory fitness as described in the 2010 ACSM guidelines (Table 6-2).

Table 6-1
Overall Recommendations for Training Stable CAD Clients

- Unless otherwise indicated, follow general guidelines for individuals with CAD as put forth by ACSM (2010).
- Ensure that the individual is under appropriate medical care before recommending and/or supervising an exercise program.
- If available, have the individual’s physician give a results summary of his or her most recent exercise ECG test (MET level achieved, exercise heart rate, and any relevant exercise-related symptoms).
- Be knowledgeable of the client’s current medications and which ones may alter exercise heart rate (ACSM, 2010) (see Appendix C).
- Avoid having the individual exert to muscular contraction “failure.”
- Always precede workouts with low-level activity, preferably low-level aerobic activity commensurate with the intensity of the primary conditioning activity.
- Use caution with high- or vigorous-intensity physical activity.
- Avoid sudden strenuous efforts with inadequate warm-up or prior lower-level activity.
- Avoid having the individual perform vigorous exercise when suffering from viral infections, colds, flu, etc.
- Understand and monitor signs and symptoms of cardiac decompensation (excessive shortness of breath, unusual or sudden-onset fatigue, palpitations, lightheadedness or dizziness, chest discomfort) for an extended period of time after cessation of the exercise session.

Essential Exercise Recommendations and Manifestations of CAD

The major benefits of exercise training include the following:

- Improved $\dot{V}O_2_{\max}$ (aerobic capacity)
- Lessening of angina symptoms/raising of the ischemic threshold
- Modest decreases in body fat, blood pressure, total and LDL cholesterol, non–high-density lipoprotein (HDL) cholesterol, and triglycerides
- Increased HDL cholesterol
- Reduction in stress
- Control of diabetes mellitus
- Improved well-being and self-efficacy

Angina Pectoris

Angina pectoris is the discomfort 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 narrowings of the coronary arteries. Angina can be easily graded by a simple angina scale (Table 6-3). The angina pain scale is a useful tool for documenting client pain complaints in SOAP notes. 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 or sublingually, or orally as needed. Unstable angina is angina that changes in intensity, character, or frequency. Unstable angina may precede myocardial infarction and requires urgent medical attention. Individuals who have unstable angina are not appropriate clients for the ACE-AHFS and should always be referred back to their physicians.

### Exercise Guidelines for Stable Angina

The ACE-AHFS should ensure that these individuals are medically cleared and are stable. These clients have myocardial ischemia (insufficient blood supply to the heart muscle) and are relatively high risk within the category of all CAD patients. Any individual who experiences angina with physical workloads ≤3 METs (i.e., low

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**Table 6-2**

**General Procedures for Submaximal Testing of Cardiorespiratory Fitness**

- Obtain resting heart rate and blood pressure immediately prior to exercise in the exercise posture.
- The client should be familiarized with the ergometer. If using a cycle ergometer, properly position the client on the ergometer (i.e., upright posture, 5-degree bend in the knee at maximal leg extension, hands in proper position on the handlebars).
- The exercise test should begin with a two- to three-minute warm-up to acquaint the client with the cycle ergometer 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/minute, steady-state heart rate (i.e., two heart rates within 5 beats/minute) 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.
- Perceived exertion (using either the 6–20 or 0–10 scale) and additional rating scales should be monitored near the end of the last minute of each stage.
- The test should be terminated when the subject reaches 70% heart-rate reserve (85% of age-predicted 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
  - A passive cool-down if the subject experiences signs of discomfort or an emergency situation occurs
- All physiologic observations (e.g., heart rate, blood pressures, signs and 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.

physical exertion levels) should not be trained by the ACE-AHFS.

• 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 VO$_2$max.

• 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, 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.

• 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 angina-resolving medications (e.g., nitroglycerin). Instances when the client uses angina-resolving medications should be documented for the client’s physician.

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 (i.e., under the tongue) 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. He or she will then wait five more minutes and then repeat one more time before calling 911. Nitroglycerine is a vasodilator and will dilate vessels, allowing for a greater oxygen delivery to the heart. Side effects of nitroglycerin administration include severe headache and a drop in blood pressure.

**Cardiac Dysrhythmias**

Cardiac dysrhythmias are cardiac rhythm disturbances that can be of atrial, anterioventricular node (AV node), or ventricular origin. Many patients with CAD 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., radio ablation or implantable defibrillators) that can reduce many types of cardiac dysrhythmias.

Cardiac dysrhythmias, especially ventricular arrhythmias, can be heart-rate and physical-effort related, and thus can be elicited by physical exercise. For this reason, the ACE-AHFS should be particularly conscious of symptoms that are induced by exercise-generated ventricular dysrhythmias. Such rhythm disturbances can come on during or after exercise. 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 two main cautions if such clients are referred to an ACE-AHFS are as follows:

• Always graduate the workload slowly, with no sudden cessation of moderate or vigorous exercise. Always graduate cool-down work.

• Avoid heavy resistance exercise or any exercise in which the client is exerting against

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**Table 6-3**  
**Angina Scale**

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 +</td>
<td>Light, barely noticeable</td>
</tr>
<tr>
<td>2 ++</td>
<td>Moderate, bothersome</td>
</tr>
<tr>
<td>3 +++</td>
<td>Severe, very uncomfortable</td>
</tr>
<tr>
<td>4 ++++</td>
<td>Most severe pain ever experienced</td>
</tr>
</tbody>
</table>

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either an isometric load or very high resistance, particularly if the client also holds his or her 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 myocardial infarction (MI), also known as a heart attack, 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, nausea, or syncope. ECG and cardiac biomarkers (e.g., cardiac troponin taken via blood test at the hospital) also help diagnose an MI. Patients frequently feel suddenly ill. Women often experience different symptoms from men. The most common symptoms of MI in women include shortness of breath, weakness, and fatigue. Approximately one-third of all myocardial infarctions 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 CAD. In approximately half of all MIs, this is the individual’s first indication of CAD. It is very important that the ACE-AHFS be capable of recognizing the signs and symptoms of MI.

Depending on the location of the obstruction in the coronary arteries, different zones of the heart can become injured. Using standard anatomical terms of MI location, one can describe anterior, inferior, lateral, apical, and septal infarctions (and combinations, such as anteroinferior, anterolateral, and so on). For example, an occlusion of the left anterior descending coronary artery will result in an anterior wall MI.

Training the post-MI client requires adherence to precautions very similar to those followed with the angina client. The ACE-AHFS 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 at least phase I and II cardiac rehabilitation or equivalent 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 ACE-AHFS 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 ACE-AHFS. All post-MI clients, but especially those with an MI within the preceding three or four months, should begin any and all exercise sessions with very low workloads (e.g., 2 to 3 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.

**Congestive Heart Failure**

One complication of CAD, particularly MI, is congestive heart failure (CHF). An MI may compromise the function of the heart as a pump for the circulatory system, a state called heart failure. There are different types of heart failure. Left- or right-sided heart failure may occur depending on the affected part of the heart, and it is a low-output type of failure. 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 heart failure 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. The ACE-AHFS should not train CHF clients unless otherwise
appropriately and specifically authorized by physician referral. There are exceptions among those with New York Heart Association Class I or II CHF (mild CHF with no or slight physical limitations), where low-level progressive exercise is appropriate and helpful. Low-level aerobic activity (2 to 5 METs) and many restorative yoga poses are beneficial when appropriately taught by experienced and qualified yoga teachers.

**Post–coronary Artery Bypass Grafting and Percutaneous Transluminal Coronary Angioplasty Intervention**

Two interventions most often employed in CAD patients are coronary artery bypass grafting (CABG) and percutaneous transluminal coronary angioplasty (PTCA). PTCA will usually include intracoronary stenting (using a wire mesh device to open the artery). The ACE-AHFS can work with all of these clients, as long as they have no complications and are stable from symptom, ventricular function, and ECG perspectives. Most often, these individuals require aggressive risk-factor control, especially blood lipid and blood pressure management. The ACE-AHFS should follow the exercise guidelines and precautions in this chapter for all CAD clients. The most important thing 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 enable 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 ACE-AHFS 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. The ACE-AHFS working with clients who are post-PTCA or post-CABG should take notice of any of the following signs and symptoms of restenosis:

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

Since CAD is common among the American population, an astute ACE-AHFS may pick up on these subtle complaints in any of the populations he or she serves.

**Essential Recommendations for Exercise Training of the Stable CAD Client**

**Overall Exercise Energy Expenditure Goals**

To optimize the potential for improvement of CAD risk factors and stabilize the disease process, clients should prioritize achieving a total volume of physical activity of 1000 kcal or more per week (ACSM, 2010). This volume includes systematic workouts, recreational activities, and activities of daily living. The ACE-AHFS must understand how to estimate the energy cost of various physical activities in terms of kcal per session, per day, and per week (Howley, 2006; Ainsworth, Haskell, & Leon, 1993). The ACE-AHFS should adhere to the overall recommendations for exercise in clients with stable CAD as presented in Table 6-1.

**Exercise Intensity**

Exercise intensity or exercise workload is perhaps the most important, and pliable, 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 heart rate and blood pressure. The two most practical intensity-monitoring strategies for the ACE-AHFS are the client’s volitional response to the exercise workload [e.g., ratings of perceived exertion (RPE)] and exercise heart rate. In some cases, HR response may be blunted due to medications (e.g., beta blockers).
ACSM (2010) recommends that the initial stages of aerobic-conditioning programs for low-risk and stable CAD clients have an exercise intensity of 30 to 55% of heart-rate reserve (i.e., approximately 30 to 55% of VO\textsubscript{2max}) or 2 to 4 METs. However, it should be assumed that in most cases the ACE-AHFS will be working with clients who are not in the initial stages of training, but who are in the improvement or maintenance stage of conditioning, in which case an intensity range of 55 to 85% of heart-rate reserve (55 to 85% of VO\textsubscript{2max}) is more appropriate. The majority of CAD clients will do well with 30 to 60 minutes of exercise. For durations of 45 minutes or longer, exercise intensity should be in the moderate range [i.e., 40 to 60% of heart-rate reserve (40 to 60% of VO\textsubscript{2max})]. It is important to understand that some CAD clients fail to achieve predicted maximal heart rates in the absence of medications that lower heart rate (e.g., beta-blocking agents). This phenomenon is known as chronotropic incompetence. These individuals are at higher risk for CVD complications and probably are not within the ACE-AHFS scope of practice.

A good but simple estimate of cardiac workload intensity can be determined using the product of exercise heart rate and systolic blood pressure (SBP) divided by 100:

### 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 corresponds to the anginal threshold (i.e., the point at which angina symptoms occur). Intensive aerobic activities significantly increase heart rate but moderately increase systolic blood pressure, whereas intensive resistance workloads (e.g., resistance training) moderately increase heart rate but cause a more significant rise in systolic blood pressure. Both forms of exercise can dramatically raise the “double product” and therefore raise cardiac workload. Cardiac symptoms and heart-muscle dysfunction are directly related to exertional heart rate and blood pressure. It is not practical for the ACE-AHFS to calculate the double product for each exercise session, but he or she should thoroughly understand the consequences of various aerobic, resistance, and even mindful exercise modalities (e.g., hatha yoga styles, Pilates) and how they influence cardiac work.

It is important for the ACE-AHFS to understand which physical activities and exercises can rapidly increase systolic blood pressure. For example, during heavy resistance exercise where an individual is exerting at ≥80% of maximum voluntary contraction levels, SBP increases quickly along with the diastolic blood pressure (DBP). Additionally, when a person exerts to muscular failure during resistance exercise, the ACE-AHFS can assume a peak or near-peak blood pressure response. Even for individuals with relatively stable CAD, this level of arterial pressure (also called “afterload”), which the heart has to pump against, can be dramatic and deserves serious caution.

### Exercise Time

Exercise duration for most CAD 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. CAD, diabetes, and metabolic syndrome clients should have exercise programming dosed by daily or weekly energy expenditure rather than separately quantifying only frequency, intensity, and time/duration. The total energy expenditure of the exercise sessions is perhaps the single most important program feature associated with risk-factor reduction.

In most cases, the CAD client will require an activity program that is at least 1000 kcal/week gross energy expenditure. In the case of a CAD client with the metabolic syndrome, atherogenic dyslipidemia, and obesity, the physical-activity program’s energy expenditure most often should be ≥2000 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 ACE-AHFS and the client to work together to design a creative and productive activity program. Once again, to constructively do this, the ACE-AHFS will need a good working knowledge of the energy costs of a broad range of physical activities (Ainsworth, Haskell, & Leon, 1993; ACSM, 2006). Refer to Table 7-7, page 159 for a sample program.

**Progression to Independent Exercise**

For those clients who have been in formal cardiac rehabilitation programs and progress to independent exercise programs in the community, the guidelines in Table 6-4 are appropriate. Note that these guidelines are mostly dependent on recent exercise ECG testing information.

**Resistance Training**

Resistance-training modalities can clearly improve the client’s muscular fitness and functionality. Because there are so many permutations of resistance delivery devices and protocols, certain precautions are important to note. The primary consideration for the ACE-AHFS in this context is the amount and rate of force delivered to the client’s muscles relative to his or her capacity and cardiac ventricular function. Table 6-5 denotes criteria for the resistance training of CAD clients. In this instance, resistance training applies to the use of free weights, machines, or other resistive devices that deliver a resistive force ≥40% of the client’s maximum voluntary contraction capacity. Table 6-6 describes absolute and relative contraindications to resistance training for individuals.

**Table 6-5**

**Client Criteria for a Resistance-training Program**

- 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 5 weeks after date of myocardial infarction or cardiac surgery, including 4 weeks of consistent participation in a supervised cardiac rehabilitation endurance training program† [range of motion (ROM) and very light resistance exercise of 1–3 lb (0.45–1.36 kg) may be started earlier if tolerated]
- Initiate a minimum of 2 to 3 weeks following transcatheter procedure (i.e., PTCA or other), including 2 weeks of consistent participation in a supervised cardiac rehabilitation endurance training program† [ROM and very light resistance exercise of 1–3 lb (0.45–1.36 kg) may be started earlier if tolerated]
- No evidence of 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.

†Entry should be a rehabilitation staff decision with approval of the medical director and surgeon as appropriate

**Table 6-4**

**Guidelines for Progression to Independent Exercise with Minimal or No Supervision**

- Estimated functional capacity of ≥7 METs (or measured ≥5 METs) or twice the level of occupational demand
- Appropriate hemodynamic response to exercise (increase in SBP with increasing workload) and recovery
- Appropriate ECG response at peak exercise with normal or unchanged conduction, stable or benign arrhythmias and non-diagnostic ischemic response (i.e., <1 mm ST-segment depression)
- Cardiac symptoms stable or absent
- Stable and/or controlled baseline HR and BP
- Adequate management of risk-factor inter-vention strategy and safe exercise participation such that the client demonstrates independent and effective management of risk factors with favorable changes in those risk factors
- Demonstrated knowledge of the disease process, abnormal signs and symptoms, medication use and side effects
- Adequate management of risk-factor inter-vention strategy and safe exercise participation such that the client demonstrates independent and effective management of risk factors with favorable changes in those risk factors

Note: MET = Metabolic equivalent; SBP = Systolic blood pressure; ECG = Electrocardiogram; HR = Heart rate; BP = Blood pressure


with CAD. These also apply to higher-intensity yoga programs (e.g., Bikram or “hot” yoga and Ashtanga or “power” yoga). The ACE-AHFS should not place him- or herself in the position to recognize these clinical contraindications, but should ensure physician clearance for these contraindications. Table 6-7 lists recommendations for appropriate resistance-training programming and progression. There are no contraindications to Pilates exercises, provided that the same guidelines are adhered to as stated in Tables 6-5, 6-6, and 6-7. As long as the client is not straining to concentrically or eccentrically contract a muscle group or performing breath-holding, Pilates mat or reformer work can be very beneficial to his or her core strength.

Table 6-6
AHA 2007 Contraindications to Resistance Training

<table>
<thead>
<tr>
<th>Absolute Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Unstable CAD</td>
</tr>
<tr>
<td>• Decompensated heart failure</td>
</tr>
<tr>
<td>• Uncontrolled symptoms</td>
</tr>
<tr>
<td>• Severe pulmonary hypertension</td>
</tr>
<tr>
<td>• Severe and symptomatic aortic stenosis</td>
</tr>
<tr>
<td>• Acute myocarditis, endocarditis, or pericarditis</td>
</tr>
<tr>
<td>• Uncontrolled hypertension (&gt;180/110 mmHg)</td>
</tr>
<tr>
<td>• Aortic dissection</td>
</tr>
<tr>
<td>• Marfan’s syndrome</td>
</tr>
<tr>
<td>• High-intensity resistance training (80–100% of 1 RM) in clients with active proliferative retinopathy or moderate or worse nonproliferative diabetic retinopathy</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Relative Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Major risk factors for CHD</td>
</tr>
<tr>
<td>• Diabetes at any age</td>
</tr>
<tr>
<td>• Uncontrolled hypertension (&gt;160/100 mmHg)</td>
</tr>
<tr>
<td>• Low functional capacity (&lt;4 METs)</td>
</tr>
<tr>
<td>• Musculoskeletal limitations</td>
</tr>
<tr>
<td>• Individuals who have implanted pacemakers or defibrillators</td>
</tr>
</tbody>
</table>

Note: 1 RM = One-repetition maximum


Mindful Exercise and the CAD Client

Select forms and styles of mindful exercise modalities are most often appropriate or can be easily adapted for clients with CAD (see Appendix D in the ACE Personal Trainer Manual). Table 6-8 lists more suitable forms of mindful exercise that are appropriate for the majority of individuals with CAD.

Mindful exercise programs can range from those requiring very low energy expenditure and deep relaxation qualities to levels that require

Table 6-8
Some Appropriate Forms of Mindful Exercise for Clients With CAD

| • Restorative yoga                                          |
| • Kripalu yoga                                              |
| • Viniyoga                                                  |
| • Integral yoga                                             |
| • Select iyengar and yoga poses                             |
| • Tai chi chuan (moderate pace)                             |
| • Tai chi chih                                              |
| • Yogic breathwork                                          |
| • Pilates mat and reformer work                             |
| • NIA at low-to-moderate level                              |

Note: CAD = Coronary artery disease; NIA = Neuromuscular integrative action

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 CAD, 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 CAD should minimize acute changes in body position that require the head to be positioned 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 Iyengar yoga poses and sequences generally require considerable strength, flexibility, and mental concentration, they should be reserved for higher functioning individuals (i.e., clients with >12 MET exercise capacity). Some yoga poses significantly increase blood pressure and may also be inappropriate for older adults with stage II or higher hypertension (i.e., blood pressures ≥160/105 mmHg). One study on intermediate and advanced yoga practitioners showed that some Iyengar poses can rapidly and significantly increase mean and peak systolic blood pressure, particularly with back arch poses (Blank, 2006). This level of blood pressure could impose significant double-product stress on the heart of some CAD clients, particularly if the stress is a sudden increase in systolic blood pressure rather than the gradual workload increase seen with graduated aerobic exercise work levels. It is strongly recommended that the ACE-AHFS start the client with restorative yoga poses prior to engaging in a full complement of Iyengar or equivalent yoga poses.

Perhaps most useful in those with any level of CAD 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 as relaxation response (Pal & Velkumary, 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 his or her pulse during this breathing exercise. He or she may notice a reduced pulse rate with prolonged exhalation. This slight slowing of heart rate includes a reciprocal slight increase in heart rate variability—a process that is also called respiratory sinus arrhythmia. Acute reductions in blood pressure also have resulted from yogic breathing training (Murrgesan, Govindarajulu, & Bera, 2000).

Case Study

A 44-year-old mother of three with a history of stage I hypertension, angina, and documented CAD is referred to the ACE-AHFS by an internist. She has a family history of CAD and her father had a myocardial infarction. She has a positive treadmill ECG with mild angina (1 on 4-point scale), achieved a heart rate of 154 bpm, and reached 11 METs. She has been participating in a low-level walking program (five days/week; two-mile walk at 2 to 3 mph).

- Weight: 165 lb (75 kg)
- Height: 5’6” (1.7 m)
- BMI: 36
This client’s goals are as follows:

- Weight: <145 lb (66 kg)
- BMI: <30
- Waist circumference: < 30 inches (76 cm)
- LDL cholesterol: <70 mg/dL
- HDL cholesterol: >50 mg/dL
- Triglycerides: <150 mg/dL
- MET capacity: >13 METs

In the absence of significant further reduction in LDL cholesterol, her physician may choose to increase the simvastatin dose to 40 mg. Note that with documented CAD, her ideal therapeutic option LDL cholesterol goal is 70 mg/dL.

**Summary**

Exercise therapy for the prevention and treatment of CAD 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 blood pressure and body fat stores. Ideally, the CAD client would have completed phase I and II cardiac rehabilitation program prior to working with the ACE-AHFS. An experienced ACE-AHFS who wishes to work with CAD 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 CAD risk factors and stabilize the disease process, clients should achieve a total volume of physical activity of 1500 to 2000 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


Suggested Reading


In This Chapter
Risk Factors for Coronary Artery Disease
Blood Lipid Disorders (Dyslipidemia)
Lipids, Lipoproteins, and Atherosclerosis
  VLDL Cholesterol
  LDL Cholesterol
  HDL Cholesterol
  Non-HDL Cholesterol
  Other Lipoproteins and Associated Biomarkers of CVD Risk
  Supportive Clinical Trials
National Cholesterol Education Program (NCEP) Guidelines
  High and Very High Risk
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Overview of Cholesterol and Exercise
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Prospective Exercise Training Lipid and Lipoprotein Responses
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Exercise and Postprandial Lipemia
Exercise Volume Programming for Overall Lipid Management
  Pedometer Stepcounts, Energy Expenditure, and Lipid Dyslipidemia
Resistance Training and Lipid Disorders
Essential Exercise-programming Steps for Individuals With Dyslipidemia
  Step 1: Evaluate Health and Lifestyle History
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  Step 5: Determine the Exercise Plan From Prior Health History, Level of Fitness, and Current Lipid Profile
  Step 6: Keep Track of the Client’s Lipid-lowering Drugs and Other Medications, if Applicable
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  Step 9: Maintain a Working Knowledge of Other Evidence-based Non-pharmacologic Interventions That Can Help Manage Lipid Disorders
  Step 10: Partner With Healthcare Professionals
The Accreditation Council for Clinical Lipidology
Case Study
Summary

About The Author
Ralph La Forge, M.S., is a physiologist and Accreditation Council on Clinical Lipidology–certified clinical lipid specialist and is the managing director of the Cholesterol Disorder Physician Education Program at Duke University Medical Center, Endocrine Division in Durham, North Carolina. Formerly, he was 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 heart-disease-prevention programs. La Forge has published more than 300 professional and consumer publications on exercise science and preventive endocrinology/cardiology.
Risk Factors for Coronary Artery Disease

It is imperative that the ACE-certified Advanced Health & Fitness Specialist (ACE-AHFS) understand the principal coronary artery disease (CAD) risk factors, as most of these are favorably altered by physical activity. The principal risk factors for CAD include elevated low-density lipoprotein (LDL) cholesterol, low high-density lipoprotein (HDL) cholesterol, high blood pressure, smoking, and diabetes. Of course, obesity, age, gender, family history, and stress all play a role, but the central focus of both lifestyle and pharmacologic therapy is on the principal risk factors. Physical activity, weight loss, smoking cessation, dietary behavior changes, and reduction in stress play key behavioral roles in the modification of these principal atherosclerotic risk factors. Other serum and biomarkers of atherosclerotic risk have also been identified, including the following: elevated triglycerides (TG), LDL cholesterol particle number, and apolipoprotein B levels; low apolipoprotein A-1 levels; elevated lipoprotein(a); and high C-reactive protein, coronary calcium score, and lipoprotein-associated phospholipase A2. This chapter addresses the importance of blood lipid disorders (dyslipidemia) in the prevention and management of CAD. With the exception of smoking, the other principal risk factors are adequately discussed in this text.

Blood Lipid Disorders (Dyslipidemia)

In terms of reducing the risk of CAD progression or a recurrent heart attack, the ACE-AHFS should prioritize a systematic approach to physical activity for the CAD client, while also helping him or her achieve optimal blood lipid control—ideally through therapeutic lifestyle changes. The ACE-AHFS should also understand the role of pharmacotherapy.

A large body of data has established that serum cholesterol and associated lipoproteins are crucial risk factors for atherosclerosis. Blood lipid disorders (also called dyslipidemia or dyslipoproteinemia) represent an important modifiable risk factor for the development and progression of coronary heart disease (CHD). The 2007 Heart and Stroke Statistical Update, published by the American Heart Association (AHA, 2007), reports that 35.6% of Americans were told that they have high cholesterol levels, with the highest percentage in West Virginia at 39.9%. The ACE-AHFS can play a pivotal role in curbing this growing incidence and work directly with physicians in the management of lipid disorders.

Lipids, Lipoproteins, and Atherosclerosis

Cholesterol is a fatty substance that travels in the blood in distinct particles that contain both lipids and proteins. These particles are called lipoproteins. The cholesterol level in the blood is determined partly by genetics.
and partly by lifestyle factors such as diet, body fat, exercise, and even psychological stress. There are four major classes of lipoproteins found in the blood of a fasting individual:

- Very-low-density lipoprotein (VLDL)
- Low-density lipoprotein (LDL)
- High-density lipoprotein (HDL)
- Non-HDL

**VLDL Cholesterol**

VLDL is a major carrier of triglycerides in the plasma. Triglyceride is a major form of fat. A triglyceride consists of three molecules of fatty acid combined with a molecule of glycerol. Synthesized in the liver, triglyceride-rich VLDL carries endogenously (produced by the body) synthesized triglycerides and cholesterol to their sites of utilization. VLDL also contains 10 to 15% of the body’s total serum cholesterol. Increased concentrations of VLDL are associated with a number of lipoprotein disorders, such as familial hypertriglyceridemia, obesity, diabetes, and nephrotic syndrome.

Triglycerides do not accumulate in the vessel wall. Their atherogenicity is based on their association with other substances in the blood. For example, elevated triglycerides tend to be associated with low HDL cholesterol and elevated LDL-particle concentration, especially in individuals with the metabolic syndrome. So the risk of having elevated triglycerides is based primarily on its association with other lipoproteins. A very high triglyceride level (>500 mg/dL) is associated with other clinical problems such as fatty liver and pancreatitis.

**LDL Cholesterol**

LDL cholesterol is the major carrier of cholesterol in the circulation. It contains 60 to 70% of the body’s total serum cholesterol and is directly correlated with the risk for coronary heart disease. LDL cholesterol and LDL particles play a pivotal role in atherogenesis, the early stages of atherosclerosis. This role is illustrated in Figures 7-1 and 7-2, which describe LDL cholesterol infiltration into the arterial endothelium and fatty streak and plaque formation. Although all blood lipids play a role in the development of atherosclerosis, epidemiologic studies suggest that LDL cholesterol is the most significant blood lipid. Without a threshold level of LDL cholesterol and LDL particles, atherosclerosis is rare, despite the presence of other risk factors. LDL cholesterol also is the primary focus of most blood-lipid-lowering therapies. Plasma LDL concentrations are regulated by specialized LDL receptors on the arterial endothelium. When there is a defect in the gene for the synthesis of the LDL receptor, plasma LDL concentrations increase, as seen in individuals with familial hypercholesterolemia. Table 7-1 depicts various LDL-cholesterol-lowering strategies and their respective efficacy.

**HDL Cholesterol**

HDL cholesterol is formed in the intestine and liver. HDL normally contains 20 to 30% of the body’s total cholesterol, and HDL levels are inversely correlated with coronary heart disease risk. HDL plays an important role in reverse cholesterol transport (i.e., removal of cholesterol from cells and transporting it back to the liver). By removing excess cholesterol from the circulation, HDL may provide a protective mechanism against the development of atherosclerosis. Research has shown that each mg/dL increase in plasma HDL cholesterol concentration is associated with approximately 3% reduction in CAD risk (Gordon, Probstfield, & Garrison, 1989).

**Non-HDL Cholesterol**

Non-HDL cholesterol is a very important lipid measure that is strongly associated with the
Figure 7-1
Impact of LDL cholesterol infiltration on coronary artery inflammation


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Figure 7-2
Fatty streak formation in atherosclerosis


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ever-increasing prevalence of obesity, metabolic syndrome, and type 2 diabetes, and thus should be of special therapeutic interest to fitness professionals, particularly the ACE-AHFS and lifestyle coaches. Non-HDL should be specifically used as a measure of risk, particularly in those individuals who have fasting triglycerides above 200 mg/dL (e.g., those who have type 2 diabetes, the metabolic syndrome, or who are obese).

Non-HDL cholesterol is calculated as follows:

\[
\text{Non-HDL cholesterol} = \text{Total cholesterol} - \text{HDL cholesterol}
\]

The goal for non-HDL cholesterol is the same number as for LDL-cholesterol for that particular individual, plus 30 mg/dL. For example, someone with diabetes or cardiovascular disease might have an LDL-cholesterol goal of <100 mg/dL. Therefore his or her non-HDL goal would be <130 mg/dL. LDL-cholesterol is still the principal target of therapy for most patients with, or at high risk for, cardiovascular disease (CVD) and for all patients with type 2 diabetes, but especially those with both CVD and diabetes. In contrast to LDL cholesterol, non-HDL cholesterol values can be calculated in non-fasting patients without measuring triglycerides.

Why is non-HDL so important? Non-HDL cholesterol level is a more comprehensive measure of atherogenic particles than LDL cholesterol level. Available data suggest that non-HDL cholesterol level is as good as, or better than, LDL cholesterol in predicting cardiovascular risk, especially in individuals who have elevated triglycerides (e.g., those with the metabolic syndrome and/or diabetes) (Cui et al., 2001; Lu et al., 2003; Jiang, 2004; Bittner, 2004).

Non-HDL cholesterol is also a good marker for apoprotein B (also designated as apoB), which is even more atherogenic than the calculated non-HDL. ApoB is a measure of total atherogenic particle load and includes a measure of both triglyceride-rich lipoproteins and LDL cholesterol (i.e., there is one apoB particle attached on the surface of each of these atherogenic lipoproteins). The laboratory assay for apoB has been standardized and is available in most clinical laboratories. Exercise training studies in healthy men and women have demonstrated significant reductions in apo B in comparison to LDL cholesterol (Holme, Hostmark, & Anderssen 2007; Ring-Dimitriou et al., 2007).

In both sexes, non-HDL cholesterol levels correlate closely with obesity and especially visceral obesity (Denke, Sempos, & Grundy, 1994). In contrast to total cholesterol and LDL cholesterol, non-HDL cholesterol responds very well to dietary and physical-activity changes, especially in children as demonstrated in the Bogalusa Heart Study (Srinivasan, Myers, & Berenson, 2002). As noted previously, non-HDL cholesterol is a good marker for apoprotein B, which is even more atherogenic than the calculated non-HDL, though apoprotein B is more difficult to measure. An optimal goal for apoprotein B levels would be <80 mg/dL, which would define low-risk.

**Other Lipoproteins and Associated Biomarkers of CVD Risk**

Besides the major lipoproteins and lipids described earlier, there are other lipoproteins, apoproteins, and lipoprotein fractions such as lipoprotein (a) [also designated as Lp(a)]; apoproteins A, B, C, D, and E; and various subfractions of VLDL, LDL, and HDL [e.g., large- and small-dense LDL cholesterol (also designated as LDL phenotype A and B, respectively)]. These lipoproteins are largely used to more definitively diagnose specific lipid disorders and to provide additional CVD risk prediction. For example, Lp(a) is structurally similar to LDL both in protein and lipid composition and is related to thrombotic (blood coagulation) risk. When significantly elevated (i.e., >30 mg/dL), Lp(a) can also help define coronary heart disease risk, but screening for Lp(a) in the general population is not suggested at this time. Lipoprotein subfractions (e.g., the smaller LDL-particle size) can also help further predict CVD risk, but their measurement requires more advanced laboratory technology [e.g., nuclear magnetic resonance (NMR) instrumentation]. The apolipoproteins lie on
the surface of the larger lipoproteins and act as ligands (points that attach to various receptors) for target receptors and regulate lipoprotein metabolism. Their measurement can help determine the cause, many times genetic, of a particular lipid disorder. For example, in the case of some forms of hypertriglyceridemia, apo CII and apo CIII expression can be the culprit.

**Supportive Clinical Trials**

Landmark primary and secondary prevention trials involving aggressive cholesterol lowering, especially of LDL cholesterol, have demonstrated significant clinical benefits, including atherosclerotic plaque regression, diminished atherosclerosis progression, increased arterial function, decreased coronary events (e.g., heart attacks), decreased stroke incidence, and decreased cardiovascular disease mortality (Sacks et al., 1996; Pederson et al., 1994; Shepard et al., 1995; Gotto, 1997; Downs et al., 1998; Rubins et al., 1999; Collins et al., 2003; Colhoun et al., 2004; Cannon et al., 2004; Petersen et al., 2005; LaRosa et al., 2005). Likewise, trials have shown CAD regression with intensive cholesterol-reduction therapy (Nissen et al., 2006; Ballantyne, 2008). In an older but well-controlled trial, Pitt, Mancini, and Ellis (1995) demonstrated that early aggressive lipid management significantly reduces clinical events such as heart attack, the need for coronary artery bypass surgery, and angioplasty after fewer than six months of therapy. Even in those who were clinically free of coronary heart disease (and had average total cholesterol and LDL), systematic reduction of lipids and lipoproteins has been shown to significantly reduce the incidence of first heart attacks and revascularization procedures (Downs et al., 1998). Evidence also indicates that women benefit from lipid lowering in a way that is similar to men (Pederson et al., 1994; Sacks et al., 1996; Colhoun et al., 2004). Furthermore, adverse changes in plasma lipids and lipoproteins that occur with age in sedentary women are not observed in women who regularly exercise (Stevenson et al., 1995 & 1997; Owens et al., 1992).

The use of a NMR imaging spectroscopy assay to assess lipoproteins has demonstrated that measuring the number of LDL cholesterol particles (i.e., LDL particle concentration or particle number) has shown much promise. There is evidence that the LDL particles themselves, not just LDL cholesterol, are the atherogenic culprits in CAD. Cromwell and others have convincingly shown that in a number of patient populations, but especially in those with the metabolic syndrome, LDL particle number is a better predictor of CVD incidence than LDL cholesterol (Cromwell & Otvos, 2007; Cromwell et al., 2007; Otvos et al., 2006). LDL particle number has a relationship with LDL cholesterol (e.g., 100 mg/dL of LDL cholesterol is equal to ~1100 nm/L of LDL particles).

**National Cholesterol Education Program (NCEP) Guidelines**

In 2002, the Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) was published (abbreviated as ATP III) (NCEP, 2002). It is the most definitive set of guidelines for diagnosing and managing dyslipidemia. The understanding and application of these guidelines is essential for an ACE-AHFS who wishes to work with dyslipidemic individuals.

Two important features in this report are the addition of the metabolic syndrome and multiple risk factors, and the Framingham CHD risk scoring tool (see Appendix E), including several modifications to the lipid and lipoprotein classification. Clinical trial findings have shown that LDL cholesterol target goals are impacted by the presence of multiple CHD risk factors (e.g., smoking and high blood pressure). The other major addition to ATP III is the addition of the diagnosis of the metabolic syndrome. Table 7-2 presents the current classifications of LDL cholesterol, total cholesterol, and HDL cholesterol, while Table 7-3 presents the classifications for triglycerides.

In 2004, NCEP ATP III was updated (Grundy et al., 2004) and further clarified that more recent research trials have clearly demonstrated that LDL target goals are best determined by global coronary heart disease risk (Table 7-4). This update necessitates the use of the Framingham risk scoring table to classify low, intermediate, high, and very
high CHD risk (see Appendix E). These tables estimate the percent probability of a major coronary event (death or a heart attack) over the next 10 years. The following sections present these risk classifications and their relationships with LDL cholesterol goals.

### Table 7-2
**ATP III Classification of LDL, Total Cholesterol, and HDL Cholesterol (mg/dL)**

<table>
<thead>
<tr>
<th>LDL Cholesterol</th>
<th>Total Cholesterol</th>
<th>HDL Cholesterol</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;100</td>
<td>&lt;200</td>
<td>&lt;40</td>
</tr>
<tr>
<td>100–129</td>
<td>200–239</td>
<td>40–60</td>
</tr>
<tr>
<td>130–159</td>
<td>&gt;240</td>
<td>&gt;60</td>
</tr>
<tr>
<td>160–189</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥190</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**LDL Cholesterol**
- <100: Optimal
- 100–129: Near optimal/above optimal
- 130–159: Borderline high
- 160–189: High
- ≥190: Very high

**Total Cholesterol**
- <200: Desirable
- 200–239: Borderline high
- ≥240: High

**HDL Cholesterol**
- <40: Low
- ≥60: High

**Note:** LDL = Low-density lipoprotein; HDL = High-density lipoprotein

### Table 7-3
**Triglycerides (mg/dL)**

<table>
<thead>
<tr>
<th>Triglycerides (mg/dL)</th>
<th>Normal</th>
<th>Borderline-high</th>
<th>High</th>
<th>Very high</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;150</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>150–199</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>200–499</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥500</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**High and Very High Risk**

For high-risk individuals, the overall goal remains an LDL level of less than 100 mg/dL. But for people at very high risk, a group that is considered a “subset” of the high-risk category, the update offers a new therapeutic option of reducing LDL to under 70 mg/dL. For very-high-risk people whose LDL levels are already below 100 mg/dL, there is also an option to use drug therapy to reach the <70 mg/dL goal. Note that the very-high-risk category is reserved only for individuals with established cardiovascular disease plus other risk factors.

The NCEP defines high-risk individuals as those who have coronary heart disease or disease of the blood vessels to the brain or extremities, diabetes, or two or more risk factors (e.g., smoking, hypertension) that give them a greater than 20% chance of having a heart attack within 10 years.

Very-high-risk individuals are those who have cardiovascular disease together with either multiple risk factors (especially diabetes), or severe and poorly controlled risk factors (e.g., continued smoking), or the metabolic syndrome (a constellation of risk factors associated with obesity, including high triglycerides and low HDL). Patients hospitalized for acute coronary syndromes such as heart attack are also at very high risk.

### Table 7-4
**ATP III LDL Cholesterol Goals and Cutpoints for TLC and Drug Therapy in Different Risk Categories and Proposed Modifications Based on Clinical Trial Evidence**

<table>
<thead>
<tr>
<th>Risk Category</th>
<th>LDL Goal</th>
<th>Initiate TLC</th>
<th>Consider Drug Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>High risk: CHD or CHD risk equivalents (10-year risk &gt;20%)</td>
<td>&lt;100 mg/dL (optional goal: &lt;70 mg/dL)</td>
<td>≥100 mg/dL</td>
<td>≥100 mg/dL (100–129 mg/dL; consider drug options)</td>
</tr>
<tr>
<td>Moderately high risk: 2+ risk factors (10-year risk: 10 to 20%)</td>
<td>&lt;130 mg/dL (optional goal: &lt;100 mg/dL)</td>
<td>≥130 mg/dL</td>
<td>≥130 mg/dL (100–129 mg/dL; consider drug options)</td>
</tr>
<tr>
<td>Moderate risk: 2+ risk factors (10-year risk: &lt;10%)</td>
<td>&lt;130 mg/dL</td>
<td>≥130 mg/dL</td>
<td>≥160 mg/dL</td>
</tr>
<tr>
<td>Low risk: 0–1 risk factor</td>
<td>&lt;160 mg/dL</td>
<td>≥160 mg/dL</td>
<td>≥190 mg/dL (160–189 mg/dL; consider drug options)</td>
</tr>
</tbody>
</table>

**Note:** LDL = Low-density lipoprotein; TLC = Therapeutic lifestyle changes; CHD = Coronary heart disease
Moderate and Moderately High Risk

For moderately high-risk individuals, the goal remains an LDL <130 mg/dL, but the update provides a therapeutic option to set a lower LDL goal of <100 mg/dL and to use drug therapy at LDL levels of 100 to 129 mg/dL to reach this lower goal. Moderately high-risk individuals are those who have multiple (two or more) risk factors for coronary heart disease together with a 10 to 20% risk of heart attack within 10 years. Moderate-risk individuals also have multiple risk factors, but a less than 10% risk of heart attack within 10 years.

Low Risk

Low-risk individuals have fewer than two risk factors and an LDL-cholesterol goal of <160 mg/dL (see Table 7-4).

The Metabolic Syndrome

The metabolic syndrome, also termed the insulin resistance syndrome—a cluster of factors associated with increased risk for CHD and diabetes—is becoming increasingly common, largely as a result of the increase in the prevalence of obesity (Grundy et al., 2004; Grundy, 2006). The core metabolic risk factors are elevated blood triglycerides, low HDL cholesterol, elevated blood pressure, elevated plasma glucose, a prothrombotic state, and a pro-inflammatory state. The root causes of this syndrome are overweight/obesity, physical inactivity, and genetic factors. On the basis of data collected in the 1990s from the National Health and Examination Survey III data, an estimated 47 million U.S. residents have the metabolic syndrome (more likely >50 million today, considering the population growth since the 1990s). The main purpose of identifying individuals with the metabolic syndrome is not to predict CHD or CAD, but to get healthcare providers to pay more attention to the medical aspects of obesity and its complications and to prioritize lifestyle therapy, especially physical activity and healthier dietary behavior.

The term cardiometabolic risk has been used to describe a broadened view of the metabolic syndrome. Cardiometabolic risk is essentially defined by a merger of the traditional Framingham CHD risk factors (e.g., smoking, cholesterol, diabetes) and the metabolic syndrome risk factors. Cardiometabolic risk goes beyond the metabolic syndrome. It encompasses a cluster of modifiable risk factors and markers that identify individuals at increased risk of cardiovascular disease (myocardial infarction, stroke, peripheral arterial disease) and type 2 diabetes. That said, one important utility of the metabolic syndrome itself is that it is a better predictor of persons at high risk for type 2 diabetes than traditional Framingham CHD risk factors (Stern, Williams, & Haffner, 2002). Prospective population studies have shown that the metabolic syndrome confers a twofold relative risk for atherosclerotic cardiovascular events (e.g., heart attack) and a fivefold increase in risk compared with people who do not have the syndrome (Grundy et al., 2005).

Although it is generally agreed that first-line clinical intervention for the metabolic syndrome is lifestyle change, this is insufficient to normalize the risk factors in many patients, and so residual risk could be high enough to justify drug therapy. Elevated triglycerides, low HDL cholesterol, elevated plasma glucose, and excess abdominal fat are among the core metabolic risk factors. A more thorough description and management strategy for the metabolic syndrome is discussed in Chapter 11.

Medications for Dyslipidemia

As indicated in the most recent NCEP guidelines, drug therapy should be considered only after patients have received at least six months of nonpharmacologic therapy, specifically intensive dietary and exercise therapy. Exceptions may include those with overt coronary heart disease, including post-myocardial infarction patients who have lipid disorders. It is important for the ACE-AHFS to recognize these drugs and understand their effects on blood lipids and lipoproteins. The drug classes and drugs most commonly prescribed for patients with lipid disorders include the following.

Bile acid sequestrants (cholestyramine, colestipol, colesevelam): These agents bind bile acids in the small intestine and cause decreased bile acid absorption, in turn lowering total and LDL cholesterol. The agents are available as dry powders in bulk or individual packets, and are usually consumed...
within one hour of eating and/or are taken with the evening meal. The bile acid sequestrants are quite effective for patients younger than age 55 who have LDL cholesterol between 160 and 220 mg/dL. Side effects are primarily gastrointestinal, with constipation being the most common.

Nicotinic acid (niacin, niaspan, niacor): Niacin is a water-soluble B vitamin that is very effective in lowering LDL cholesterol and triglycerides, but is especially utilized to increase HDL cholesterol when used in relatively high doses (>1,500 mg/day). Nicotinic acid is relatively inexpensive, making it an attractive choice as either single therapy or in combination with other drugs, such as statins. Its chief side effects include significant cutaneous flushing and vasodilatation. Niacin must be used with caution in patients with liver disease, diabetes, or gout and those at risk for peptic ulcer disease. It is important to note that many over-the-counter (OTC) forms of “niacin” consist of inositol hexanicotinate (often termed “no-flush niacin”) and are relatively void of sufficient free nicotinic acid, and therefore do very little for LDL, triglycerides, or HDL.

HMG-CoA reductase inhibitors (also known as statins, which include lovastatin, pravastatin, simvastatin, fluvastatin, atorvastatin, rosuvastatin, and pitavastatin in Japan): These drugs effectively lower LDL cholesterol by interfering with cholesterol synthesis. They are competitive inhibitors of HMG-CoA reductase, the enzyme responsible for the rate-limiting step in the cholesterol biosynthetic pathway. They also block the formation of mevalonic acid and decrease intracellular cholesterol synthesis. These drugs are the most effective and expensive class of medications available for lowering LDL cholesterol. Several of the statins have substantial clinical trial support for reducing cardiovascular morbidity and mortality, as well as clinical events and the need for cardiovascular intervention procedures.

Combination nicotinic acid and statin drugs: Advicor® and Simcor® are combination drugs that combine in one pill various combinations of niaspan and lovastatin (Advicor) and niaspan and simvastatin (Simcor).

Fibrates (gemfibrozil, fenofibrate): Gemfibrozil and fenofibrate primarily lower triglycerides and, to a lesser extent, increase HDL cholesterol by reducing VLDL (triglyceride) synthesis and increase VLDL clearance by increasing lipoprotein lipase activity.

Cholesterol transport inhibitors (ezetimibe): These drugs can lower LDL cholesterol by 15 to 20% and are frequently added to statin therapy to further boost LDL-lowering efficacy. Vytorin® (simvastatin + ezetimibe in several formulations) is a potent drug designed to lower LDL cholesterol by 40 to 65%.

Omega-3 fatty acids (select OTC brands or Lovaza®): Marine omega-3 fatty acid therapy (fish oil capsules) is prescribed for individuals with high or very high triglycerides. The prescription may be for 1 to 4 grams per day of omega-3 fatty acids in the presence of high or very high triglycerides (200 to 500 mg/dL or higher). It is important to understand that only at the higher intakes of omega-3 fatty acids (≥2 grams/day) are there significant reductions in triglycerides, whereas the lower dosages have other benefits not directly related to triglyceride lowering.

Other agents (non-FDA-approved): A variety of other nutritive and herbal OTC agents are available to consumers. Many of these agents (e.g., “natural cholesterol-lowering products”) will be accompanied by ad campaigns claiming the power to lower blood cholesterol. For example, several red yeast rice–based supplements claim on their package inserts that they can lower LDL cholesterol by 13 to 16%. The same holds true for policosanol nutrient products (a sugar-case extract). While not particularly harmful, these products have claims of 15 to 25% reductions in LDL cholesterol. Guggulipid and green tea extract product campaigns also promote lipid lowering and may have some promise in select individuals, but lack controlled clinical trials substantiating their benefit. Consideration must also be given to the potential adverse interaction that these supplements may have when combined with prescription medications. Until better-controlled research is conducted and published, these supplements should not be considered primary modes of therapy in lipid management, and any client considering these supplements should first consult a licensed practitioner.
Overview of Cholesterol and Exercise

Exercise is not generally considered primary therapy for lipid disorders, especially in the current era of lipid-modifying drug therapy. This is unfortunate, because exercise of appropriate quality and quantity can clearly reduce cardiometabolic risk through nonlipid mechanisms, but it can also induce significant favorable changes in the lipid and lipoprotein profile, in part as a result of reductions in adiposity. One meta-analysis of 13 studies representing 613 subjects reported that only triglycerides were significantly altered as a result of exercise training (Kelley, Kelley, & Tran, 2005). However, one must apply caution when attempting to generalize large review studies. Depending on the type of blood lipid disorder and baseline lipids, exercise training of sufficient volume can quite favorably alter blood lipids. Dietary reduction of fat, especially saturated and trans fat; exercise; and weight loss are still the cornerstones of therapy for individuals who have elevated blood lipids, despite the overwhelming number of lipid-lowering drug trials and LDL-cholesterol-lowering drug promotional campaigns extolling the benefits of statin therapy. With the exception of those who have existing CAD and diabetes, the essential first steps of therapy should be diet and exercise. This recommendation is clearly emphasized in the NCEP ATP III guidelines.

One of the hallmark findings of lipid-lowering drug therapy is improved arterial endothelial function, primarily through enhanced nitric oxide formation and function (nitric oxide is the most potent endogenous arterial vasodilator). Improved endothelial function is thought by many to be one of the primary mechanisms responsible for reduced CVD morbidity and mortality (Bugiardini et al., 2004; Gotto, 1997). Research also has demonstrated similar improvements in endothelial function with sufficient exercise training (Hambrecht et al., 2000; Clarkson et al., 1999; Utriainen et al., 1996).

The Lipid/Lipoprotein Response to Exercise Training

Exercise training of sufficient volume (i.e., kcal energy expenditure per week) generally increases HDL cholesterol and lowers total cholesterol, LDL cholesterol, and triglycerides via a number of mechanisms, including reduced body-fat stores, decreased hepatic lipase activity, and increased lipoprotein lipase activity. Most studies demonstrating exercise-improved lipid profiles have involved subjects with relatively normal blood lipids. There are very few randomized controlled studies appraising the lipid response to exercise training in people with lipid disorders. It is very possible that individuals with lipid disorders may respond differently to a given dose of exercise, depending on the type of dyslipidemia (e.g., those with lipoprotein lipase deficiencies or specific apolipoprotein E genotypes). For most individuals, there appears to be a minimum weekly physical-activity volume required for significant changes in blood lipids. For example, Church and colleagues (2007) reported no significant change in body weight, lipids, or lipoproteins after six months of 400, 800, or 1200 kcal/week exercise training in 464 postmenopausal women. Higher exercise energy expenditure thresholds (e.g., >2200 kcal/week) are likely required for individuals with elevated total and LDL cholesterol (Crouse et al., 1997; Kraus et al., 2002; ACSM, 2010).

As a general rule, fat weight reduction is required for the most favorable blood lipid response in those who have elevated total and LDL cholesterol. This volume of exercise (150 minutes or more per week, optimally 200 to 300 minutes per week, or ≥2000 kcal per week) is similar to that recommended for obesity (ACSM, 2010). If exercise is of sufficient volume, exercise intensity is not of primary importance in improving the overall blood lipid profile, although most research supports a minimum intensity of at least 40% of peak work capacity (Durstine & Moore, 1997; ACSM, 2010).

Table 7-5 lists factors that play a role in determining serum lipids and the exercise/blood-lipid response. This large number of factors is the reason...
the exercise/blood-lipid response is complex and very individualized.

Decreased body fat tends to correlate reasonably well with reduced LDL cholesterol and increased HDL cholesterol, which is why it is important to periodically evaluate body-fat composition in dyslipidemic clients. Most exercise trials support between 700 and nearly 2000 kcal of exercise per week to significantly alter HDL cholesterol and, to a lesser extent, LDL cholesterol (Williams, 1998; Kokkinos et al., 1995; King et al., 1995; Kraus et al., 2002; Shadid et al., 2006).

### Prospective Exercise Training Lipid and Lipoprotein Responses

#### Triglycerides

Compared to other lipids, such as LDL cholesterol, elevated blood triglycerides (TG) are generally more responsive to exercise training. Triglyceride mobilization and utilization appears to be in direct proportion to exercise energy expenditure. Blood triglycerides frequently decrease with exercise training, depending on baseline values and volume of exercise. Unlike total and LDL cholesterol, triglycerides generally decrease immediately after a session of high-volume endurance exercise (e.g., greater than 45 to 50 minutes of sustained effort), and remain lower for up to 48 hours after the session. Trejo-Gutierrez and Fletcher (2007) reported a mean exercise-induced reduction in triglycerides of 24% (range of 4% to 37%).

The exercise program should follow the optimal mode, intensity, duration, and frequency for fat-weight reduction (e.g., 40 to 70% of aerobic capacity for 40 to 60+ minutes, four to six days per week). Four days a week of endurance exercise (e.g., four miles/day of jogging or 400+ kcal of energy expenditure) has been shown in a number of research trials to significantly reduce TG, especially in individuals with elevated baseline triglycerides (Durstine & Haskell, 1994). Overall, a threshold of fat-weight loss may be required for sustained TG reduction. Additionally, a reduction in TG is generally associated with an increase in HDL cholesterol, especially in hypertriglyceridemic subjects.

Subjects who have familial hypertriglyceridemia (e.g., lipoprotein lipase or apo C-II deficiencies) will have fasting triglycerides well above 500 mg/dL and often greater than 1000 mg/dL. While there is very little published research on this population’s response to exercise, it is important to note that a relatively high volume of exercise in addition to drug therapy will be the most appropriate combination therapy.

#### LDL Cholesterol and Total Cholesterol

Most studies evaluating the total cholesterol and/or LDL cholesterol response to exercise training have found zero to only moderate decreases in these lipids and lipoproteins. Many studies used inadequate exercise volumes and/or energy expenditure or failed to control for confounding variables such as training-induced changes in plasma volume, dietary habits, or seasonal variation in cholesterol and lipoproteins (Durstine & Haskell, 1994; Durstine et al., 2002).

The total and LDL cholesterol response to exercise training is quite variable, but tends to positively correlate with associated fat weight loss. LDL cholesterol reduction appears to be slightly more responsive than total cholesterol to endurance exercise training. When LDL cholesterol

---

**Table 7-5**

Primary Factors Influencing Blood Lipids and the Exercise Blood Lipid Response

- Frequency, duration, and intensity of exercise (as these increase, total caloric expenditure increases)
- Type of lipid disorder (e.g., variety of genetic hyperlipidemias)
- Total exercise energy expenditure
- Length of training period (e.g., one month, six months, 18 months)
- Coexisting body-fat loss
- Corresponding and compensatory dietary changes
- Concomitant alcohol intake
- Baseline lipid values
- Plasma volume changes
- Gender and menopausal status
- Genetic factors, (e.g., apolipoprotein E isoforms)
- Biologic variation (seasonal and diurnal changes)
concentrations are lower (e.g., <130 mg/dL), the reduction has been inversely related to the distance run each week (Wood et al., 1983). Most studies have provided mixed findings for plasma cholesterol concentrations for male runners, female runners, cross-country skiers, and other endurance-trained athletes compared with inactive controls (Superko, 1991; Durstine & Haskell, 1994). More recent results from endurance-training studies have been more encouraging. For example, Halverstadt and colleagues (2007) reported a significant reduction in total cholesterol, LDL cholesterol, and LDL particle concentration in 100 sedentary, healthy 50- to 75-year-olds after 24 weeks of endurance training. Altena and colleagues (2006) compared the effects of four weeks of continuous versus intermittent aerobic exercise on lipoproteins and lipoprotein subfractions and likewise demonstrated significant reductions in total cholesterol, LDL cholesterol, and the total cholesterol:HDL cholesterol ratio. Inactivity also can induce increases in blood lipids. Slentz and colleagues (2007) have shown that 15 days of inactivity can increase LDL cholesterol and LDL particle number and that a modest amount of exercise (approximately 2 miles of walking per week at 40 to 55% of $\text{VO}_2\text{max}$) can prevent this increase.

Exercise training that results in weight loss and plasma volume expansion is more likely to result in lower LDL cholesterol and total cholesterol. Most research indicates minimal thresholds of a 1000 kcal of exercise per week (e.g., 12 or more miles of walking per week). Ideally, 2000 kcal or more per week for four to six months is required for significant reductions in LDL cholesterol (ACSM, 2010).

**HDL Cholesterol**

Overall, there is a modest increase in HDL cholesterol in response to exercise training. When diet is held constant, Leon and Sanchez (2001) reported an average increase in HDL of 4.38% (range −5.8% to +25%) in response to exercise training. There have been mixed findings among studies investigating the relationship between exercise intensity and increases in HDL, with some studies reporting the necessity for more vigorous exercise intensities. Inactive subjects may not increase HDL through energy expenditure as easily as physically active subjects (Durstine & Moore, 1997). Individuals with various genetic forms of very-low-HDL cholesterol (hypoalphalipoproteinemia, HDL levels <30 mg/dL) in general will respond minimally to even high levels and volumes of exercise. There are several forms of familial hypoalphalipoproteinemia (genetically related very-low-HDL cholesterol) and the prevalence is approximately one in 50 adults.

Baseline fasting triglycerides also may contribute to the HDL-raising effects of exercise training. Couillard and others (2005) studied 200 men enrolled in the HERITAGE Family Study and demonstrated that regular endurance exercise training may be particularly helpful in men with low HDL cholesterol, elevated triglycerides, and abdominal obesity. Other research suggests that HDL cholesterol may transiently increase after a single bout of endurance exercise in men (Pronk, 1993; Visich et al., 1996). Park and Ransone (2003) observed a 6% increase in 24-hour HDL cholesterol (HDL2 and HDL3 subspecies) after 350 kcal of treadmill exercise at lactate threshold in 18 college-age men. There were no significant changes in HDL cholesterol when these subjects ran at 70% of lactate threshold. Triglycerides also may respond favorably to a single bout of exercise commensurate with an increase in HDL cholesterol. Such acute changes may be realized eight to 12 hours after 300 to 500 kcal of exercise. Sustained increases in HDL cholesterol and HDL-2 appear to require a relatively high exercise-volume threshold. This threshold ranges from running seven miles per week, or approximately 700 kcal (Kokkinos et al., 1995), to running 15 miles per week, or approximately 1500 kcal (Williams, 1996). Hartung (1995) reported that for each six miles run per week, HDL was approximately 3 mg/dL higher in both men and women. In general, for inactive individuals, ≥1000 kcal per week of exercise above their weekly physical activity baseline may be necessary for significant increases in HDL cholesterol.

There is evidence that HDL cholesterol may be more responsive to a higher daily frequency of exercise (e.g., three 15-minute sessions vs. one 40-minute session). Baseline HDL cholesterol
and genetic factors have a significant impact on the capacity to increase HDL via exercise. Research also indicates that exercise-induced HDL cholesterol increases can be independent of exercise intensity, especially in men and women older than 45 to 50 years of age (King et al., 1995; Crouse et al., 1997). Slentz and colleagues (2007) have shown that higher versus moderate volumes of exercise may in some cases be required for significant HDL cholesterol increases. Studies also demonstrate that older adults may take longer to increase HDL cholesterol with exercise training, perhaps as long as two years (King et al., 1995).

Exercise and Postprandial Lipemia

Postprandial lipemia is essentially the blood fat, particularly triglyceride, response to a meal. Depending on how much fat or sugar is consumed in a meal, a person with normal fasting triglycerides will increase his triglycerides by 120 to 300+ mg/dL for two to six hours after the meal. Those with visceral obesity, the metabolic syndrome, or type 2 diabetes can have much larger increases in post-meal triglycerides. The problem of prolonged elevated postprandial triglyceride states is that for the amount of time triglycerides are elevated much above 250 to 300 mg/dL there is diminished arterial function, lower HDL cholesterol, and exposure of the arterial wall to atherogenic lipoprotein particles (e.g., VLDL remnants). Over the past 10 years, there has been abundant research supporting the finding that sufficient exercise timed anywhere from one to 12 hours before a fat-rich meal will reduce postprandial lipemia by 25 to 40% (Zhang et al., 1998; Petitt & Cureton, 2003; Malkova & Gill, 2006). This observation was also found in men with baseline hypertriglyceridemia with a 30 to 39% reduction in postprandial triglycerides with moderate and vigorous exercise, respectively (Zhang, 2006). This somewhat blunted triglyceride response to high-fat or high-glycemic meals is one of the benefits of a daily aerobic exercise program.

Exercise Volume Programming for Overall Lipid Management

Although there will be significant individual variation, it appears that to improve overall lipoprotein status (LDL, HDL, non-HDL, and triglycerides), an exercise volume of at least 1500+ kcal per week (e.g., running 15 miles or walking 20+ miles per week) may be the minimum necessary based on available research. Optimally, 2000 kcal/week or more is recommended. The ACSM guidelines (2010), based on numerous clinical trials, state that the exercise volume to reduce dyslipidemia should be consistent with the same guidelines for long-term weight control (Table 7-6). Table 7-7 depicts sample exercise protocols with approximate weekly energy expenditures ranging from 600 to 2000+ kcal per week.

Pedometer Stepcounts, Energy Expenditure, and Lipid Dyslipidemia

Systematic use of pedometers (pedometry) or walking stepcounts has been employed as an acceptable estimate of walking energy expenditure. For blood lipid changes or weight loss, the minimal weekly physical-activity goals should be at least 1500 kcal more than entry level physical activity (entry level being evaluated on the first visit). This weekly energy expenditure would be equivalent to about 30,000 or more walking stepcounts beyond the client’s weekly baseline stepcount. The ACE-AHFS is encouraged to recommend reliable well-engineered pedometers to estimate walking energy expenditure. Perhaps most applicable would be the use of pedometer models that have a step filter, which is incorporated software that minimizes the recording of meaningless spontaneous movements. Optimal goals are ≥2000 kcal/wk, 200+ minutes per week, and/or ≥70,000 total steps per week.

As a general rule, about 2000 steps (+ or – approximately 150 steps) with a step-only pedometer is equivalent to approximately one mile, which is approximately 100 kcal of gross energy expenditure for individuals who are
Blood Lipid Disorders

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Harris, 1997). It is likely, therefore, that the blood lipid response to strength training is related to total net energy expenditure of the session (kcal per workout), as is the case with endurance exercise. One example of a relatively high-energy-expenditure resistance-training session is low-resistance, high-repetition circuit weight training performed for extended periods and approaching 300 kcal or more per session.

Essential Exercise-programming Steps for Individuals With Dyslipidemia

Step 1: Evaluate Health and Lifestyle History

- Relevant comorbidities
- Blood lipid history and current blood lipid profile
- Exercise treadmill test history if at high CVD risk
- Medications, especially lipid-altering medications
- Diet and exercise history

Within 10 to 20 pounds of their ideal body weight. Of course, for those individuals who have a significantly higher body mass index (BMI) (e.g., >35), the gross caloric cost per mile is greater (about 120 to 150 kcal per mile) and in proportion to their body weight.

Resistance Training and Lipid Disorders

As with hypertension management, resistance training is not recommended as the primary form of exercise therapy for individuals with blood lipid disorders. Resistance training certainly may be recommended as a component of a complete exercise program. Research has shown that the blood lipid response to strength training is negligible, with some studies reporting slight-to-moderate reductions in total and LDL cholesterol and others reporting no change (Tucker, Martin, & Harris, 1997; Boyden et al., 1993; Kokkinos et al., 1988). In a study of 8499 men, only those investing more than four hours a week to resistance training maintained this benefit (Tucker, Martin, & Harris, 1997). It is likely, therefore, that the blood lipid response to strength training is related to total net energy expenditure of the session (kcal per workout), as is the case with endurance exercise. One example of a relatively high-energy-expenditure resistance-training session is low-resistance, high-repetition circuit weight training performed for extended periods and approaching 300 kcal or more per session.

Table 7-6
Dyslipidemia Physical-activity Guidelines

Based on known therapeutic effects of habitual physical activity, ACSM makes the following recommendations regarding exercise programs for persons with dyslipidemia:

- Primary activity: Aerobic exercise using large muscle groups; resistance training should be incorporated
- Intensity: 40–75% \( \text{VO}_2 \text{R} \) or HRR
- Frequency: 5 or more days per week
- Duration: 30–60 minutes

Note: This exercise program is consistent with recommendations for long-term weight control: 200–300 minutes/week of moderate physical activity or >2000 kcal/week; \( \text{VO}_2 \text{R} = \text{VO}_2 \text{res} \); HRR = Heart-rate reserve


Table 7-7
Sample Graduated Weekly Exercise Energy Expenditures [assumes 160–180 pound (72–81 kg) body weight] (values are expressed in estimated gross energy expenditure costs)

Protocol A (600–800 kcal/wk)
Monday, Wednesday, Friday: Walk 2 miles/day* = 600 kcal
Sunday: 20 minutes of low-level stationary cycling = 100 kcal

Protocol B (1000–1200 kcal/wk)
Monday, Wednesday, Friday: Walk 2 miles/day* = 600 kcal
Tuesday: Walk 3 miles* = 300 kcal
Sunday: Nine holes of golf or 30 minutes of singles tennis = 300 kcal

Protocol C (1500–1800 kcal/wk)
Monday, Wednesday, Friday: Walk 3 miles/day* = 900 kcal
Tuesday, Thursday: 30 min of cycling (50–60% \( \text{VO}_2 \text{max} \)) = 300 kcal
Sunday: 60 min of singles tennis plus 2-mile walk* = 500 kcal

Protocol D (2000+ kcal/wk)
5 days per week, average 300 kcal workout (e.g., 30- to 45-minute aerobic session) = 1500 kcal
1 day per week, perform a long slow-distance workout (e.g., 2-hour moderate- to fast-pace variable-terrain walk) = 600+ kcal

* Walking at moderate pace (2.5–4 mph)
The following evaluations may be appropriate prior to beginning an exercise program for lipid management:
- Baseline lipid/lipoprotein profile
- Exercise ECG
- Physician evaluation
- Clinical dietary assessment and diet prescription
- Evaluation for dyslipidemia medications

Step 3: Perform Anthropometric Measures

Many lipid disorders are sensitive to changes in body-fat stores. For this reason, it is essential that an ACE-AHFS initially and serially assess valid measures of body fat. Body weight, abdominal girth (measured at the level just above the iliac crest, a measure of central visceral fat stores), and/or upper-body skinfolds (e.g., triceps or subscapular) as assessed by Lange or equivalent skinfold calipers should be recorded during the client’s initial visit and at four- to six-week intervals throughout the course of exercise training. It is important to focus on the change in waist circumference, BMI, and anthropometric measures rather than their relationship to normative body-fat data. Total cholesterol, LDL cholesterol, non-HDL cholesterol, and triglycerides usually decrease with a diminution in body fat, especially abdominal or visceral fat reduction. HDL cholesterol may or may not directly correlate with fat-weight changes.

Step 4: Set a Realistic Target Lipid Goal for Exercise Therapy

It is important to emphasize that exercise-lipid responses vary among people and that the volume of exercise required for significant changes in blood lipids is generally at a higher weekly energy expenditure threshold than that for reducing blood pressure or improving psychological well-being. For this reason, it may take more time to realize the clinical benefits. The ACE-AHFS must be conservative with short-term lipid-reduction goals, especially with total and LDL cholesterol reductions. A 5 to 15% LDL cholesterol reduction or a 10 to 20% reduction in non-HDL...
cholesterol is generally a realistic goal for the first
12 to 16 weeks of exercise training, assuming suffi-
cient weekly exercise energy expenditure. Because
non-HDL cholesterol includes triglyceride-rich lipoproteins, it may be more responsive and a
better target of exercise therapy than LDL choles-
terol, depending on baseline LDL cholesterol and triglycerides. Many clients may take six months or
longer to show significant decreases in total and
LDL cholesterol. This is not unusual, as there are
a considerable variety of lipid disorders and blood
lipid phenotypes. When possible, the ACE-AHFS
should incorporate other co-variants of lipid
reduction, such as valid anthropometric measures
of obesity and abdominal-visceral fat, to demon-
strate progress toward predetermined goals.

Laboratory lipid-assessment values character-
istically vary by 8 to 12%, based on biological
variation, lab bias, and analytic factors. This
is important to consider when interpreting
serial blood lipid values. Examples of variation
in LDL and HDL cholesterol concentration
include hospitalization, estrogen replacement
therapy, pregnancy, type 2 diabetes, smoking,
acute infection, posture, venous occlusion, and
seasonal and circadian biological variation. The
National Heart, Lung, and Blood Institute
(NHLBI) published a comprehensive set of rec-
ommendations for laboratories and healthcare
providers on ensuring valid lipoprotein mea-
surement and interpretation (NHLBI, 1995).

Step 5: Determine the Exercise Plan
From Prior Health History, Level of
Fitness, and Current Lipid Profile
The exercise plan should be written clearly and
concisely and include exercise mode, frequency,
duration, intensity, progression plan, and safety
precautions. The client’s health history and initial
fitness level are integral to formulating the weekly
volume. For example, for clients with stable CAD
who have had a recent exercise ECG, it will be
important to review exercise electrocardiographic
and hemodynamic data to appropriately set the
exercise intensity and duration range. The indi-
vidual’s exercise capacity in metabolic equivalents
(METs) or measured VO₂ will also be helpful in
determining initial exercise work levels.

Overall, it may be most straightforward to
program exercise by energy expenditure or total
weekly caloric expenditure with pedometry
(i.e., weekly stepcounts), or simply by duration
and intensity. Initial weekly exercise volumes
should be set realistically according to the cli-
ent’s initial level of fitness, body composition,
and existing comorbidities. See Table 7-7 for a
sample set of exercise energy-expenditure pro-
tocols. Since many clients may be significantly
deconditioned and overweight, it may be most
appropriate to start on a progressive walking
program. In this case, walking distance, speed,
and the difficulty of the terrain should be grad-
ually increased over the course of the program
to generate higher energy expenditures. Since
overall blood lipid improvement is responsive
to weekly exercise volumes (total physical activ-
ity energy expenditures) and exercise-generated
fat-weight loss, it is imperative that the ACE-
AHFS knows how to reliably estimate session,
daily, and weekly exercise energy expenditures
in kcal. The following are examples of gross
energy-expenditure target goals by lipid and
lipoprotein:

- Elevated LDL and/or total cholesterol: ≥2000 kcal per week
- Low HDL: ≥1000 kcal per week
- Elevated triglycerides: ≥1000 kcal per week
- Combined dyslipidemia (elevated LDL and triglycerides with low HDL): ≥2000 kcal per week

Step 6: Keep Track of the Client’s
Lipid-lowering Drugs and Other
Medications, if Applicable
If a client is on lipid-lowering drugs, it is wise
to know which drug or combination of drugs
he or she is taking and any associated dosage
changes. The combined use of exercise and
lipid-lowering drug therapy can significantly
reduce the time needed to achieve the lipid
goal. As a group, lipid-lowering drugs have
little, if any, effect on exercise hemodynamics.
Beta-blocking medications, with the exception
of the few that have intrinsic sympathomi-
metic activity (e.g., acebutolol and pindolol),
will have a tendency to increase triglycerides
and decrease HDL cholesterol. Individuals on significant dosages of niacin therapy (e.g., >1500 mg/day) may have a greater tendency to experience a drop in blood pressures after exercise in warm weather. Niacin can also cause flushing and headaches in early stages of this form of pharmacotherapy. As a final note, many lipid-lowering drugs (e.g., statins, fibrates, and niacin) require periodic liver-function tests to assess the possibility of liver toxicity.

**Step 8: Follow Up**

Encourage follow-up blood lipid profile laboratory evaluations in accordance with the referring physician or the lipid clinic’s follow-up protocol. Exercise counseling follow-up would ideally be executed in conjunction with the routine lipid clinic follow-up visit, or at six- or eight-week intervals. The exercise plan should be revised as needed, with documentation of weekly energy expenditures and exercise mode(s). Anthropometric measures should be assessed at every clinic evaluation or at the follow-up visit. Dietary and medication compliance should also be routinely assessed at each session.

**Step 7: Track and Document the Client’s Musculoskeletal Symptom Status During the Exercise Program**

Clients on statins who are exercising at relatively high intensities or volumes are somewhat more susceptible to exercise-associated muscle aches (myalgia). One report suggests statin exacerbated exercise-induced skeletal muscle injury, as measured by elevated creatine kinase (CK) levels (an index of skeletal muscle injury), in a group of 59 men who took 40 mg of lovastatin per day while embarking on a five-week vigorous endurance-exercise program (Thompson et al., 1997). Although statins are usually well-tolerated, they have occasionally been associated with myopathy (myalgia and muscle weakness, with CK elevations) and there is some chance that this situation could be exacerbated with exercise. Myopathy is rapidly reversible if diagnosed early by a physician and treated with discontinuance of drug and hydration. Although the rare occurrence of statin-induced myopathy should not alarm the ACE-AHFS, it does reinforce the need for the ACE-AHFS to keep reasonably close track of any acute and/or recovery musculoskeletal symptoms through at least the early stages of the exercise program and after statin dose changes. The co-administration of approximately 100 mg a day of the antioxidant coenzyme Q10 can possibly help reduce statin-related myalgia (Caso et al., 2007) but not all coenzyme Q-statin studies show this benefit, even at higher doses (Young, 2007). The client should discuss adding coenzyme Q10 to his or her supplement regimen with a physician.

**Step 9: Maintain a Working Knowledge of Other Evidence-based Non-pharmacologic Interventions That Can Help Manage Lipid Disorders**

For optimal results, it will often be important to use exercise as a complement to other nonpharmacologic (and pharmacologic) measures. For example, exercise stands the best chance of helping a client reach his or her NCEP lipid goal if it is combined with dietary therapy (NCEP, 2002). Supplemental antioxidant vitamin intake (e.g., vitamin E) may be of value in reducing LDL cholesterol oxidation, although these supplements do not directly affect blood lipid levels (Rimm & Stampfer, 1997). Folic acid, vitamin B6, and vitamin B12 supplementation may also be of help in reducing serum homocysteine levels. Homocysteine is an amino acid that contributes to the build-up of lipids in arteries and increases blood clotting tendency (Wald et al., 1998). The client should discuss adding any of the aforementioned supplements with his or her physician.

Stress- and anger-management interventions, when applicable, should also be included in a comprehensive lipid-management plan. The rationale for such behavioral programs stems from stress-related catecholamine production and its putative relationship with LDL oxidation, LDL receptor regulation, and macrophage activation, all of which are integral in the development of atherosclerosis (Williams et al., 1991; Muldoon et al., 1999).
Step 10: Partner With Healthcare Professionals

In some cases, the ACE-AHFS will be collaborating with a physician-directed lipid clinic team in providing therapy. In this sense, the ACE-AHFS is a member of a medical team and may be required to provide progress reports to the lipid clinic’s patient record. A helpful description of lipid clinic operations and referral affiliations is available through the National Lipid Association (La Forge, 2006). In other instances, the ACE-AHFS may act independently through self-referral. In this case, it will be necessary to communicate exercise progress to the client’s physician and to discuss the relevance of additional tests. Becoming a member of the National Lipid Association (www.lipid.org) is an essential first step.

The Accreditation Council for Clinical Lipidology

The Accreditation Council for Clinical Lipidology (ACCL) (www.lipidspecialist.org) is an independent certifying organization that has developed standards and an examination in the field of clinical lipidology for the growing number of mid- and advanced-level healthcare practitioners who manage individuals with lipid and other related disorders. This organization was developed in association with the National Lipid Association specifically for non-physician advanced-practice clinical professionals, including the ACE-AHFS. An ACE-AHFS can qualify, prepare for, and sit for the ACCL board exam. This is an academically and clinically robust exam that is offered multiple times per year around the U.S. and is very similar to the clinical lipidology board exam offered to physicians. The board credential is “certified clinical lipid specialist.” Professional knowledge and skill competencies for this credential include the following:

- Lipoprotein metabolism
- Molecular lipidology
- Epidemiology and clinical trials
- Metabolic syndrome
- Nutrition and nonpharmacologic therapy
- Risk assessment and NCEP guidelines
- Pharmacological therapy
- Safety, behavior, and compliance
- Pharmacodynamics
- Anthropometry

To learn more about this accreditation, visit www.lipidspecialist.org.

Case Study

The client is a 58-year-old male computer programmer with a family history of cardiovascular disease (mother had myocardial infarction at age 51), a 12-year history of obesity, elevated LDL cholesterol (ranging from 168 mg/dL to 185 mg/dL), HDL ranging from 42 to 49 mg/dL, and triglycerides ranging from 170 to 184 mg/dL. He is 5’6” (1.7 m) tall, 175 pounds (79 kg), and has a waist circumference of 35 inches (89 cm). The client’s initial level of physical activity was several recreational activities, including participation in a bowling league for two hours a week and a 30- to 40-minute walk twice a week.

Minimal goal: <130 mg/dL LDL cholesterol, <160 non-HDL cholesterol, and <35 inches (89 cm) waist circumference

Optimal lipid goal: <100 mg/dL LDL cholesterol, <130 non-HDL cholesterol

He was put on 10 mg/day of rosuvastatin, but over the last two years failed to consistently take the dose every day. He was also prescribed a NCEP therapeutic lifestyle dietary program (saturated and trans fat reduction). Subsequently, he decreased his LDL cholesterol to 119 mg/dL after complying with 10 mg of rosuvastatin every day, but his body weight remained at 175 pounds (79 kg) and his waist circumference remained at 35 inches (89 cm). Six months ago, the client was encouraged to continue to comply with 10 mg of rosuvastatin once a day, every day, as well as the following exercise program.

Exercise program: 2000+ kcal/week.

Variable-terrain walking four times per week at approximately 60% of peak heart rate as determined by his most recent exercise ECG; the walking duration began at 20 minutes per session and progressed to 70 minutes per session after
12 weeks. He also performed two 50-minute aerobic (stationary bike, treadmill, elliptical trainer) exercise sessions a week at a local fitness center, where his average exercise intensity was 65 to 70% of peak heart rate (50 to 65% of heart-rate reserve).

Follow-up: At six months, his initial follow-up (while on the 10 mg rosuvastatin and 2,000 kcal exercise program) showed that he had LDL cholesterol of 102 mg/dL, triglycerides of 111 mg/dL, and HDL cholesterol of 56 mg/dL. His body weight and waist circumference decreased to 164 pounds (74 kg) and 32 inches (81 cm), respectively.

Summary

On average, exercise by itself will reduce LDL cholesterol by 5 to 15% and increase HDL cholesterol by 5 to 15%, depending on exercise volume and the nature of the lipid disorder. Even with a modest 5 to 10% reduction in total and LDL cholesterol, there is a significant decrease in cardiovascular disease risk. Epidemiologic studies have clearly demonstrated that for every 1% reduction in LDL, there is a 2 to 3% reduction in the incidence of CAD (NCEP, 2002).

Lipid management represents a worthwhile opportunity for the ACE-AHFS, given the growth of supportive clinical trials justifying aggressive lipid therapy in dyslipidemic patients and the burgeoning growth of physician-directed lipid and cardiometabolic risk-reduction clinics. Currently, the majority of these programs do not adequately address exercise in any systematic manner. Any ACE-AHFS who is interested in lipid disorders should find this a welcome challenge and seek to forge strong alliances with these outpatient healthcare provider teams.
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Church, T.S. et al. (2007). Effects of different doses of physical activity on sedentary or obese post menopausal women with high blood pressure. Journal of the American Medical Association, 297, 2081–2091.


Cui, Y. et al. (2001). Non-high-density lipoprotein cholesterol level as a predictor of cardiovascular disease mortality. Archives of Internal Medicine, 161, 1413–1419.


Grundy, S.M. et al. (2005). Diagnosis and management of the metabolic syndrome: An


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**Suggested Reading**


In This Chapter

Epidemiology
Overview of Hypertension (Mechanisms)
  Control of Blood Pressure
  Physiology of Hypertension
Diagnostic Criteria
NHLBI Guidelines
Treatment of Hypertension
  Non-pharmacological Treatment
  Pharmacological Treatment
  Surgical Treatment
  Exercise Treatment
Hypertensive Medications and Cardiovascular Response
  Diuretics
  Beta Blockers
  Angiotensin Converting Enzyme (ACE) Inhibitors
  Angiotensin-receptor Blockers (ARBs)
  Aldosterone-receptor Antagonists
  Alpha Blockers
  Calcium Channel Blockers
  Central Acting Alpha 2 Blockers
  Peripheral Vasodilators
The Role of Exercise in Hypertension Prevention
  Exercise-related Treatment and Management of Hypertension
Cardiovascular Responses to Exercise Training
  Changes to the Sympathetic Nervous System
  Changes to the Renin-Angiotensin-Aldosterone System
  Changes to the Peripheral Blood Vessels
Thermoregulation
Training Guidelines
  Programming and Progression Guidelines and Considerations
  Cardiovascular Training
  Resistance Training
  Mind-body Exercise
Case Studies
  Case Study 1
  Case Study 2
Summary

About The Authors

W. Larry Kenney, Ph.D., is a professor of kinesiology and physiology at Pennsylvania State University, and former president of the American College of Sports Medicine. His research focuses on thermal physiology and the biophysics of heat transfer, including the effect of aging and hypertension on human skin blood flow. Dr. Kenney has published more than 150 papers and has been continually funded by that National Institutes of Health since 1985.

Lacy A. Holowatz, Ph.D., is a research associate in kinesiology at Pennsylvania State University. Dr. Holowatz has received awards from the American College of Sports Medicine and the American Physiological Society for her work on impairments in the mechanisms that increase skin blood flow during heat stress in aged and hypertensive populations.
Hypertension

W. Larry Kenney & Lacy A. Holowatz

An independent predictor of mortality, hypertension affects more than 76 million adults in the United States [American Heart Association (AHA), 2011]. Exercise is a cornerstone therapy in the prevention and treatment of this disease. It is important for an ACE-certified Advanced Health & Fitness Specialist (ACE-AHFS) to know the role of regular exercise therapy in the management of hypertension and how physical activity produces a decrease in blood pressure (BP). Because most hypertensive individuals will take two or more antihypertensive medications to control their BP, it is also important to know the potential effects and interactions of antihypertensive medications on the cardiovascular system during exercise.

Epidemiology

Hypertension is an independent risk factor for coronary artery disease, stroke, and renal failure. The relationship between BP and adverse cardiovascular events is direct—the higher the BP, the greater the chance of heart attack, heart failure, stroke, and kidney disease (Chobanian et al., 2003). Hypertension is defined as having a systolic blood pressure (SBP) ≥140 mmHg, a diastolic blood pressure (DBP) ≥90 mmHg, and/or being on antihypertensive medication. According to these criteria, approximately 76 million individuals in the United States and 1 billion individuals worldwide have hypertension (AHA, 2011; Burt et al., 1995; Hajjar & Kotchen, 2003; Rosendorff et al., 2007).

The incidence of hypertension increases with advancing age, with over half of Americans over the age of 65 having some form of hypertension (Rosendorff et al., 2007). The relationship between BP and age is complex. After age 50, SBP steadily increases, whereas DBP plateaus around the sixth decade of life and decreases thereafter. Accordingly, the incidence of isolated systolic hypertension (SBP ≥140 mmHg) or combined systolic-diastolic hypertension (SBP ≥140 mmHg and DBP ≥90 mmHg) increases with age, while the incidence of isolated diastolic hypertension (DBP ≥90 mmHg) decreases with age. The longitudinal Framingham Heart Study has estimated that the 20-year projected risk for developing hypertension is >90% for men and women who are not yet hypertensive by middle age (Vasan et al., 2002).

With the increased mortality risk associated with hypertension, combined with the age-related increase in BP, the early identification and treatment of individuals who will likely become hypertensive has become increasingly important. These individuals are categorized as having “prehypertension”—a SBP of 120 to 139 mmHg or a DBP of 80 to 89 mmHg. For each 20 mmHg rise in SBP or 10 mmHg rise in DBP, the risk of cardiovascular disease doubles (Chobanian et al., 2003). Thus, effective diet, exercise, lifestyle modifications, and pharmacological (drug) therapy to lower BP in prehypertensive and hypertensive individuals is vital to decreasing their total cardiovascular risk. Randomized clinical trials have shown that lowering BP decreases an individual’s cardiovascular risk by as much as 50% (Chobanian et al., 2003).
Even though the benefit of reducing and controlling BP in prehypertensive and hypertensive individuals is clear, the control rates for hypertension are undesirable. Approximately 30% of adults in the United States are unaware of their high BP. Of those diagnosed with hypertension, two-thirds are not achieving appropriate BP control (BP <140/90 mmHg) (Chobanian et al., 2003).

### Overview of Hypertension (Mechanisms)

Cardiac output is the product of heart rate and stroke volume, the volume of blood the heart pumps in one beat. BP is the product of cardiac output, the amount of blood the heart pumps out in one minute, and total peripheral resistance (TPR), the resistance to blood flow that the blood vessels provide.

\[
\text{Cardiac output} = \text{Heart rate} \times \text{Stroke volume}
\]
\[
\text{Blood pressure} = \text{Cardiac output} \times \text{Total peripheral resistance}
\]

Stroke volume is altered by the amount of blood filling the heart (preload), the pressure the heart must pump against (afterload), and the force of cardiac contractility. Therefore, alterations in these variables resulting in an increase in either cardiac output or total peripheral resistance can cause hypertension.

### Control of Blood Pressure

**Short-term Reflex Control of Blood Pressure**

Maintaining an adequate BP to ensure sufficient blood flow to the brain is one of the main priorities of the cardiovascular system. Blood pressure is integratively controlled by cardiovascular, neural, renal, and hormonal networks. Like many other controlled variables in physiology, BP is controlled via negative feedback with sensors, a defended set point, and effector responses. The sensors that regulate BP regulation are baroreceptors (pressure receptors) and they are located in the aortic arch and the carotid artery walls. Baroreceptors send neural signals to the cardiovascular control centers in the brain regarding the current BP. The cardiovascular control centers in the brain set a predetermined set point and integrate the incoming signals from the baroreceptors regarding the current BP. Depending on what the current BP is and how it compares to this predetermined set point, signals are sent out to the heart, blood vessels, and kidneys via the autonomic nervous system to cause a change in BP to bring it closer to the set point. When blood pressure is low or below the set point, there is little stretch on the baroreceptors, resulting in a reduction in the afferent nervous system signal going to the brain. The cardiovascular control centers integrate the signal from the baroreceptors and send out signals to the heart and blood vessels in an attempt to increase BP. These efferent signals are carried through the autonomic nervous system (parasympathetic and sympathetic nervous systems). During a low-BP state, parasympathetic activity to the heart is quickly decreased, which serves to rapidly increase heart rate. In addition, sympathetic nervous system activity increases, which also increases heart rate and cardiac contractility. The actions of the parasympathetic and the sympathetic nervous systems on the heart increase cardiac output. Increased sympathetic activity also causes vasoconstriction in the blood vessels, which increases total peripheral resistance (Folkow, 1982).

When blood pressure is high, the baroreceptors are stretched and the afferent signal to the cardiovascular control centers in the brain is increased. The resulting efferent response is to increase parasympathetic activity to the heart to slow heart rate and to inhibit sympathetic activity to cause a passive vasodilation of the peripheral blood vessels. This decreases BP, bringing it closer to the set point.

### Long-term Neural-hormonal Control of Blood Pressure

In addition to the short-term mechanisms that alter BP, increased sympathetic nervous system activity also has prolonged effects through its action on the kidneys and the release of hormones to increase blood volume. During a drop in BP, when there is decreased stretch on the baroreceptors, the hormone vasopressin (antidiuretic hormone) is released to help increase blood
volume. This hormone causes water to be reabsorbed in the kidneys. In addition to vasopressin release, increased sympathetic nerve activity activates the beta receptors in the kidneys, which results in a decrease in blood flow to the kidneys, causing the release of another hormone called renin. Renin, in turn, activates a cascade of hormonal events to cause further vasoconstriction in the peripheral blood vessels and an increase in salt and water reabsorption in the kidney to enhance blood volume. Figure 8-1 illustrates the renin-angiotensin-aldosterone system (RAAS). Renin is an enzyme released from the kidneys that activates the hormone angiotensinogen by cleaving off part of the protein to form angiotensin I. Angiotensin I is then converted to angiotensin II through another enzyme called angiotensin converting enzyme (ACE). Angiotensin II is a potent vasoconstrictor that binds to receptors on peripheral blood vessels, causing the vascular smooth muscle in the blood vessels to contract. Angiotensin II also causes the mineral corticoid hormone aldosterone to be released from the adrenal cortex. Aldosterone causes sodium and water to be reabsorbed in the kidneys. Overall, the actions of aldosterone serve to increase blood volume. The RAAS system is extremely important for the long-term regulation of BP (Rowell, 1993), and in the pathogenesis of hypertension.

**Physiology of Hypertension**

Any disturbance in the normal negative feedback control of BP between the cardiovascular, neural, renal, or hormonal systems can result in
hypertension. There are several identifiable causes of hypertension that are summarized in Table 8-1. When there is an identifiable cause of hypertension, this is termed secondary hypertension, because it is secondary to a disease state. In these cases, correcting the underlying pathology can sometimes cure the hypertension. However, in greater than 90% of cases of hypertension, there is no single identifiable cause (AHA, 2011). Hypertension without an identifiable cause is clinically termed essential or primary hypertension. While the etiology of essential hypertension is unclear, in general, central cardiovascular control centers and the baroreceptors in patients with hypertension acquire a new elevated set point and become less sensitive, meaning that it takes a larger change in BP for the system to respond and correct that change. As a result of the elevated set point, there is a decrease in parasympathetic nerve activity to the heart and an increase in resting sympathetic nerve activity to the heart, vasculature, and kidneys. This situation causes an increase in heart rate and cardiac contractility via stimulation of the beta receptors in the myocardium, as well as an increase in peripheral vasoconstriction via the action of the sympathetic nervous system neurotransmitters (e.g., norepinephrine) on the blood vessels. Furthermore, in the kidneys, increased sympathetic stimulation causes renin to be released, which activates the RAAS. RAAS activation increases peripheral vasoconstriction through the actions of angiotensin II on the vasculature and increases salt and water reabsorption in the proximal tubules of the kidneys. The resulting increase in total body extracellular volume increases preload on the heart, which serves to further increase cardiac contractility. Thus, feedback regulation in this tightly controlled BP regulatory system is impaired and results in hypertension (Folkow, 1982).

Because the control of BP involves many different organ systems, there can be various mechanisms that ultimately lead to hypertension. There are several common pathophysiological findings associated with high BP. There appears to be a change in the BP set point, an overall increase in sympathetic nervous system activity, and a decrease in the sensitivity of the baroreceptors to a change in BP (Hesse et al., 2007). In addition, there are also alterations in the local control of peripheral blood vessels. To help control blood flow, blood vessels release substances called vasodilators, which cause the blood vessels to open wider, and vasoconstrictors, which cause the blood vessels to narrow. With hypertension, there is a shift from locally released vasodilators to vasoconstrictors. This change is associated with an increase in free radical (oxidant) stress in the blood vessels. This pro-vasoconstrictor state, along with the elevated pressure on the blood vessels themselves, causes remodeling of the blood vessel walls. This remodeling makes the blood vessel walls hypertrophy and become stiff.

Long-term elevations in BP can also induce changes in the heart itself. Just like in the blood vessels, the heart muscle begins to hypertrophy. This is not an advantageous hypertrophy like

<table>
<thead>
<tr>
<th>Table 8-1</th>
<th>Identifiable Causes of Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Sleep apnea</td>
<td>• Nonadherence</td>
</tr>
<tr>
<td>• Drug-induced or related causes (see below)</td>
<td>• Inadequate doses</td>
</tr>
<tr>
<td>• Chronic kidney disease</td>
<td>• Inappropriate combinations</td>
</tr>
<tr>
<td>• Primary aldosteronism</td>
<td>• Nonsteroidal anti-inflammatory drugs; cyclooxygenase 2 inhibitors</td>
</tr>
<tr>
<td>• Renovascular disease</td>
<td>• Cocaine, amphetamines, other illicit drugs</td>
</tr>
<tr>
<td>• Chronic steroid therapy and Cushing's syndrome</td>
<td>• Sympathomimetics (decongestants, anorectics)</td>
</tr>
<tr>
<td>• Pheochromocytoma</td>
<td>• Oral contraceptives</td>
</tr>
<tr>
<td>• Coarctation of the aorta</td>
<td>• Adrenal steroids</td>
</tr>
<tr>
<td>• Thyroid or parathyroid disease</td>
<td>• Cyclosporine and tacrolimus</td>
</tr>
</tbody>
</table>

Drug-induced or other causes
- Nonadherence
- Inadequate doses
- Inappropriate combinations
- Nonsteroidal anti-inflammatory drugs; cyclooxygenase 2 inhibitors
- Cocaine, amphetamines, other illicit drugs
- Sympathomimetics (decongestants, anorectics)
- Oral contraceptives
- Adrenal steroids
- Cyclosporine and tacrolimus
- Erthythropoietin
- Licorice (including some chewing tobacco)
- Selected over-the-counter dietary supplements and medicines (e.g., ephedra, ma huang, bitter orange)

Associated conditions
- Obesity
- Excess alcohol intake

the changes that are induced in the myocardium during endurance-exercise training. Instead, with hypertension, the heart muscle becomes thicker through concentric hypertrophy and less efficient as a pump. This is referred to as left ventricular hypertrophy and can be detected on an electrocardiogram.

Prolonged uncontrolled hypertension has many deleterious effects on the cardiovascular system, including remodeling of the myocardium and vascular smooth muscle. This type of remodeling can cause target organ damage in the heart, brain, kidneys, and peripheral blood vessels. Table 8-2 lists the major pathologies caused by hypertension-induced target organ damage.

Table 8-2
Pathologies Cause by Hypertension-induced Target Organ Damage

<table>
<thead>
<tr>
<th>Heart</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Left ventricular hypertrophy</td>
</tr>
<tr>
<td>• Angina or prior myocardial infarction</td>
</tr>
<tr>
<td>• Prior coronary revascularization</td>
</tr>
<tr>
<td>• Heart failure</td>
</tr>
<tr>
<td>Brain</td>
</tr>
<tr>
<td>• Stroke or transient ischemic attack</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
</tr>
<tr>
<td>Peripheral arterial disease</td>
</tr>
<tr>
<td>Retinopathy</td>
</tr>
</tbody>
</table>


**Genetics and Hypertension**

The underlying genetic and pathophysiological mechanisms contributing to essential hypertension vary widely and depend on individual characteristics and environmental interactions. Genetic studies in humans to identify the genes that cause essential hypertension are in their infancy. Essential hypertension is genetically encoded on multiple genes and has several phenotypic (outward physical manifestations of disease) and genotypic (genetic code) subtypes. Therefore, each gene that encodes for hypertension only has a small effect on BP (Weder, 2008). However, the genetic predisposition for hypertension is permissive, meaning that environmental influences are necessary for hypertension to ultimately develop.

Considering the genetic and environmental contributing factors in the development of hypertension, it is not surprising that there are many different physiological mechanisms responsible for high BP. The mechanisms that cause hypertension and the individual responses to treatment can differ depending on several variables, including race and age. For example, the incidence of hypertension is greater among African Americans than it is among other ethnic groups. Furthermore, it has also been demonstrated that African Americans respond to certain drug therapies for hypertension better than others (Ferdinand, 2008). Certainly, the data suggest that there are physiological differences in the mechanisms of hypertension depending on genetics, but there are also many environmental and socioeconomic explanations for some of these findings. One additional example to illustrate different causes and physiological mechanisms of hypertension involves individuals who develop high BP at a young age versus those who develop it later in life. In younger individuals, the onset of hypertension is associated with an increase in salt and water retention that results in an increase in cardiac output. In contrast, hypertension in older patients is more commonly associated with an increase in peripheral vascular resistance. These different mechanisms and contributing factors to high BP should be considered, especially when selecting suitable pharmacological and non-pharmacological interventions to treat high BP.

**Diagnostic Criteria**

Table 8-3 provides the classification of BP for adults 18 and older (Chobanian et al., 2003). These classifications are based on the average of two or more properly measured seated BP readings on two or more occasions.

The auscultatory method of BP measurement with a properly calibrated and validated sphygmomanometer (BP cuff) should be used to measure BP. To obtain a proper resting BP measurement, clients should avoid activities that increase their BP, such as exercise, ingestion of caffeine, and
smoking, for at least 30 minutes prior to having their BP measured. The client should be seated quietly in a chair with both feet on the floor for at least five minutes prior to obtaining the measurement. BP should be measured with the arm at heart level. The force of gravity will artifactually influence the BP measurement if the arm is not at heart level. In addition, an appropriately sized BP cuff (cuff bladder encircling at least 80% of the arm) should be used to ensure accuracy of the measurement. Most sphygmomanometers have a line on the inside of the cuff (facing the client) that is useful for ensuring that the appropriate size cuff is used. At least two measurements of BP should be made, with five minutes between measurements. The client should continue resting quietly (no talking) in a seated position during the time between BP measurements. When measuring BP, SBP is the pressure at the point where the first of two or more Korotkoff sounds is heard. This is the maximum pressure generated when the heart is contracting. DBP is the pressure before the disappearance of the Korotkoff sounds when the heart is relaxed. BP readings should be confirmed by reading BP in the contralateral (i.e., non-dominant) arm. BP can vary by as much as 10 mmHg between arms. However, the higher pressure (usually the lower pressure is in the left arm) more accurately reflects intra-arterial pressure (Grim & Grim, 2008).

It is occasionally necessary to monitor ambulatory BP in individuals to record their BP reading over a period of 24 hours. This is especially necessary in cases of “white coat hypertension,” in which BP increases under perceived stressful situations, such as being a patient at a physician’s office. In this situation, there is a learned stress response and BP is acutely elevated in the physician’s office or under other stressful conditions, but is not chronically elevated. White coat hypertension is suspected when BP is acutely elevated, but there is no history of hypertension or evidence of target organ damage. Note: Twenty-four hour ambulatory blood pressure is also more predictive of target organ damage.

### NHLBI Guidelines

In 2003, the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-VII) provided guidelines for hypertension prevention and management (Chobanian et al., 2003). These guidelines are based on a systematic evaluation of studies and are meant to increase awareness, prevention, treatment, and control of hypertension.

One of the key elements of the JNC-VII report was the inclusion of a new prehypertensive category (see Table 8-3). Those individuals who fall within the prehypertensive category have twice

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**Table 8-3**

**Classification of Blood Pressure for Adults Age 18 and Older**

<table>
<thead>
<tr>
<th>Category</th>
<th>Systolic (mmHg)</th>
<th>Diastolic (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal†</td>
<td>&lt;120 and &lt;80</td>
<td></td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120–139 or 80–89</td>
<td></td>
</tr>
<tr>
<td>Hypertension‡</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 1</td>
<td>140–159 or 90–99</td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>≥160 or ≥100</td>
<td></td>
</tr>
</tbody>
</table>

* Not taking antihypertensive drugs and not acutely ill. When systolic and diastolic blood pressures fall into different categories, the higher category should be selected to classify the individual’s blood pressure status. For example, 140/82 mmHg should be classified as stage 1 hypertension, and 154/102 mmHg should be classified as stage 2 hypertension. In addition to classifying stages of hypertension on the basis of average blood pressure levels, clinicians should specify presence or absence of target organ disease and additional risk factors. This specificity is important for risk classification and treatment.

† Normal blood pressure with respect to cardiovascular risk is below 120/80 mmHg. However, unusually low readings should be evaluated for clinical significance.

‡ Based on the average of two or more readings taken at each of two or more visits after an initial screening.

the risk of developing hypertension than those who have a lower BP. The guidelines set forth by the National Heart, Lung, and Blood Institute (NHLBI) emphasize the importance of identification of prehypertensive individuals. Individuals in this category are not candidates for pharmacological treatment of their BP, but instead should adopt lifestyle modifications to control their BP and prevent cardiovascular disease. Importantly, there are a number of causal factors for hypertension, including excess body weight, excess salt intake, inadequate intake of fresh fruits and vegetables, sedentary lifestyles, and excess alcohol consumption. Simply modifying these causal factors can treat and prevent hypertension in these individuals. Thus, exercise professionals play a key educational and motivational role in treating individuals with prehypertension and established hypertension.

Figure 8-2 is the algorithm healthcare providers use for the treatment of hypertension. The primary goal of any treatment (non-pharmacological and pharmacological) is to decrease BP to <140/90 mmHg. However, in patients with diabetes or existing chronic kidney disease, the goal of BP treatment is to attain a BP of <130/80 mmHg, as these populations are at an increased risk of further target organ damage and cardiovascular events. The majority of individuals with hypertension will require two or more

### LIFESTYLE MODIFICATIONS

| Not at goal blood pressure (<140/90 mmHg) | (130/80 mmHg for patients with diabetes or chronic kidney disease) |

#### INITIAL DRUG CHOICES

| Without compelling indications | With compelling indications |

#### Stage 1 hypertension
- (SBP 140–159 or DBP 90–99 mmHg)
- Thiazide-type diuretics for most. May consider ACEI, ARB, BB, CCB, or combination.

#### Stage 2 hypertension
- (SBP ≥160 or DBP ≥100 mmHg)
- Two-drug combination for most (usually thiazide-type diuretic and ACEI, or ARB, or BB, or CCB).

#### Drug(s) for the compelling indications
- Other antihypertensive drugs (diuretics, ACEI, ARB, BB, CCB) as needed.

### NOT AT GOAL BLOOD PRESSURE

- Optimize dosages or add additional drugs until goal blood pressure is achieved.
- Consider consultation with hypertension specialist.

**Note:** DBP = Diastolic blood pressure; SBP = Systolic blood pressure; ACEI = Angiotensin converting enzyme inhibitor; ARB = Angiotensin receptor blocker; BB = Beta blocker; CCB = Calcium channel blocker

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**Figure 8-2**
Algorithm for treatment of hypertension

medications, in addition to lifestyle modifications, to reach their target BP. However, the first line of defense in treating mild hypertension is to adopt a healthy lifestyle that includes regular aerobic exercise. Table 8-4 lists the recommended lifestyle modifications and the resulting average decrease in systolic BP. The most significant decrease in BP with lifestyle modification comes in the form of weight loss. Because exercise is a key component to weight loss and results in a significant decrease in BP, appropriate exercise programs should be incorporated into the treatment plan for clients with hypertension. Lifestyle modifications, including exercise and weight reduction, can prevent a progressive rise in BP and cardiovascular disease, especially in prehypertensive individuals.

If the target BP is not achieved by adopting healthy lifestyle changes, then pharmacotherapy is necessary. Depending on the initial stage of hypertension (see Table 8-3), one to two drugs are initially prescribed to lower BP. For stage I hypertension, the most commonly prescribed drug is a thiazide-type diuretic. This type of drug works on the kidneys to decrease salt and water load, thereby decreasing excess extracellular volume. Alternatively, other BP-lowering drugs may be prescribed based on specific individual variables and the primary mechanisms causing the hypertension. With stage II hypertension, a two-drug combination therapy is used to reach the target BP. The therapy regimen typically includes a thiazide-type diuretic and one other antihypertensive drug. If drug therapy is ineffective at reducing BP to the desired level, modification of dosages and the addition of different classes of antihypertensive drugs may be necessary to achieve the goal BP. After antihypertensive therapy is initiated, BP should be routinely monitored to determine the efficacy of treatment and determine if the goal BP has been reached.

### Table 8-4
**Lifestyle Modifications to Manage Hypertension***†

<table>
<thead>
<tr>
<th>Modification</th>
<th>Recommendation</th>
<th>Approximate SBP Reduction (Range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight reduction</td>
<td>Maintain normal body weight (body mass index 18.5–24.9 kg/m²)</td>
<td>5–20 mmHg/10 kg weight loss</td>
</tr>
<tr>
<td>Adopt DASH eating plan</td>
<td>Consume a diet rich in fruits, vegetables, and low-fat dairy products with a reduced content of saturated and total fat</td>
<td>8–14 mmHg</td>
</tr>
<tr>
<td>Dietary sodium reduction</td>
<td>Reduce dietary sodium intake to no more than 100 mmol per day (2.4 g sodium or 6 g sodium chloride)</td>
<td>2–8 mmHg</td>
</tr>
<tr>
<td>Physical activity</td>
<td>Engage in regular aerobic physical activity such as brisk walking (at least 30 minutes per day, most days of the week)</td>
<td>4–9 mmHg</td>
</tr>
<tr>
<td>Moderation of alcohol consumption</td>
<td>Limit consumption to no more than two drinks (1 oz or 30 mL ethanol; e.g., 24-oz beer, 10-oz wine, or 3-oz 80-proof whiskey) per day in most men and to no more than one drink per day for women and lighter weight persons</td>
<td>2–4 mmHg</td>
</tr>
</tbody>
</table>

*For overall cardiovascular risk reduction, stop smoking.
†The effects of implementing these modifications are dose- and time-dependent, and could be greater for some individuals.

**Note:** SBP = Systolic blood pressure; DASH = Dietary Approaches to Stop Hypertension

Treatment of Hypertension

The goal of hypertension treatment is to decrease BP to <140/90 mmHg in uncomplicated cases and to <130/80 mmHg in individuals with diabetes or kidney disease. The treatment of hypertension involves adopting a healthy lifestyle, including regular aerobic exercise, and when necessary, pharmacological intervention. In most cases of hypertension, two or more medications will be required to achieve the goal BP. It is important for exercise professionals to work in collaboration with clinicians and nutritionists to optimize the BP-lowering potential of lifestyle modifications with pharmacotherapy. This collaborative relationship creates a support network for clients with hypertension, thereby improving adherence to treatment.

Non-pharmacological Treatment

Non-pharmacological treatments for hypertension include significant lifestyle modifications. These lifestyle modifications, along with the corresponding decrease in systolic BP, are detailed in Table 8-4. Weight reduction of as little as 10 pounds (4.5 kg) in overweight individuals significantly reduces and/or prevents a rise in BP (Hypertension Prevention Research Group, 1997). In addition to weight reduction and incorporating regular aerobic physical activity, it is recommended that hypertensive individuals limit dietary sodium intake to less than 2400 mg per day, which is the equivalent of 1 teaspoon. Most Americans consume at least 75% of their sodium from processed foods (AHA, 2011). Therefore, adopting a healthy eating plan, including fresh fruits and vegetables, low-fat dairy products, and reduced saturated and total fat content, is vital to reducing dietary sodium intake from prepared foods. There is an additive effect of each of these lifestyle modifications on BP reduction. For example, significantly reducing dietary sodium intake and adopting the Dietary Approach to Stop Hypertension (DASH) eating plan has a similar effect on BP as a single pharmacotherapy (Chobanian et al., 2003). Furthermore, the addition of lifestyle modifications to pharmacotherapy results in a greater reduction in BP than pharmacotherapy alone.

The DASH eating plan emphasizes fruits, vegetables, low-fat dairy products, whole grains, poultry, fish, and nuts, and contains only a small amount of red meat, sweets, and sugar-containing beverages (Table 8-5). The DASH eating plan contains a decreased total amount of saturated fat and cholesterol and is lower in total fat. In large clinical trials where total caloric intake was held constant (compared to the pre-diet enrollment), one to two months of dietary modifications with the DASH eating plan reduced SBP by 5 mmHg and DBP by 3 mmHg. Furthermore, reducing the sodium content of the diet had an additive effect on the total reduction in blood pressure combined with the DASH eating plan. Trials that have tested the impact of individual nutrients (e.g., fat, fiber, calcium, or magnesium) on BP have not found an effect large enough to account for the overall DASH-diet response. Thus, the reduction in BP with the DASH eating plan cannot be attributed to any single nutrient, but instead to the total composition of the DASH eating plan.

Pharmacological Treatment

There are many pharmacological options available for the treatment of hypertension. Treatment options depend on other diseases and confounding pathologies that are unique to the individual. Considering that there are many potential contributing mechanisms to the development of hypertension, the optimal pharmacotherapy to treat hypertension will differ for each individual. In general, the following drug classes are used to treat hypertension:

- Diuretics
- Beta blockers
- Angiotensin converting enzyme (ACE) inhibitors
- Angiotensin receptor blockers (ARBs)
- Aldosterone-receptor antagonists
- Alpha 1 blockers
- Ca++ channel blockers
- Centrally acting alpha 2 blockers
- Peripheral vasodilators

Figure 8-3 illustrates where each of the antihypertensive drug classes alters the physiological mechanisms that control BP. The
## Table 8-5
The DASH Eating Plan

<table>
<thead>
<tr>
<th>Food Group</th>
<th>Daily Servings (except as noted)</th>
<th>Serving Sizes</th>
<th>Examples and Notes</th>
<th>Significance of Each Food Group to the DASH Eating Plan</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grains and grain products</td>
<td>7–8</td>
<td>1 slice bread&lt;br&gt;1 oz dry cereal*&lt;br&gt;½ cup cooked rice, pasta, or cereal</td>
<td>Whole-wheat bread, English muffin, pita bread, bagel, cereals, grits, oatmeal, crackers, unsalted pretzels, popcorn</td>
<td>Major sources of energy and fiber</td>
</tr>
<tr>
<td>Vegetables</td>
<td>4–5</td>
<td>1 cup raw leafy vegetable&lt;br&gt;½ cup cooked vegetable&lt;br&gt;6 oz vegetable juice</td>
<td>Tomatoes, potatoes, carrots, green peas, squash, broccoli, turnip greens, collards, kale, spinach, artichokes, green beans, lima beans, sweet potatoes</td>
<td>Rich sources of potassium, magnesium, and fiber</td>
</tr>
<tr>
<td>Fruits</td>
<td>4–5</td>
<td>6 oz fruit juice&lt;br&gt;1 medium fruit&lt;br&gt;½ cup dried fruit&lt;br&gt;½ cup fresh, frozen, or canned fruit</td>
<td>Apricots, bananas, dates, grapes, orange juice, grapefruit, grapefruit juice, mangos, melons, peaches, pineapples, prunes, raisins, strawberries, tangerines</td>
<td>Important sources of potassium, magnesium, and fiber</td>
</tr>
<tr>
<td>Low-fat or fat-free dairy foods</td>
<td>2–3</td>
<td>8 oz milk&lt;br&gt;1 cup yogurt&lt;br&gt;1½ oz cheese</td>
<td>Fat-free (skim) or low-fat (1%) milk, fat-free or low-fat buttermilk, fat-free or low-fat regular or frozen yogurt, low-fat and fat-free cheese</td>
<td>Major sources of calcium and protein</td>
</tr>
<tr>
<td>Meats, poultry, and fish</td>
<td>2 or less</td>
<td>3 oz cooked meats, poultry, or fish</td>
<td>Select only lean; trim away visible fats; broil, roast, or boil, instead of frying; remove skin from poultry</td>
<td>Rich sources of protein and magnesium</td>
</tr>
<tr>
<td>Nuts, seeds, and dry beans</td>
<td>4–5 per week</td>
<td>½ cup or 1½ oz nuts&lt;br&gt;2 Tbsp or ½ oz seeds&lt;br&gt;½ cup cooked dry beans</td>
<td>Almonds, filberts, mixed nuts, peanuts, walnuts, sunflower seeds, kidney beans, lentils, peas</td>
<td>Rich sources of energy, magnesium, potassium, protein, and fiber</td>
</tr>
<tr>
<td>Fats and oils¹</td>
<td>2–3</td>
<td>1 tsp soft margarine&lt;br&gt;1 Tbsp low-fat mayonnaise&lt;br&gt;2 Tbsp light salad dressing&lt;br&gt;1 tsp vegetable oil</td>
<td>Soft margarine, low-fat mayonnaise, light salad dressing, vegetable oil (such as olive, corn, canola, or safflower)</td>
<td>DASH has 27% of calories as fat, including fat in or added to foods</td>
</tr>
<tr>
<td>Sweets</td>
<td>5 per week</td>
<td>1 Tbsp sugar&lt;br&gt;1 Tbsp jelly or jam&lt;br&gt;½ oz jelly beans&lt;br&gt;8 oz lemonade</td>
<td>Maple syrup, sugar, jelly, jam, fruit-flavored gelatin, jelly beans, hard candy, fruit punch, sorbet, ices</td>
<td>Sweets should be low in fat</td>
</tr>
</tbody>
</table>

* Equals ½–1½ cups, depending on cereal type. Check the product’s Nutrition Facts label.

¹ Fat content changes serving counts for fats and oils. For example, 1 Tbsp of regular salad dressing equals one serving; 1 Tbsp of a low-fat dressing equals ½ a serving; 1 Tbsp of a fat-free dressing equals 0 servings.

Source: National Heart Lung and Blood Institute of the National Institutes of Health
Another cause of secondary hypertension is an epinephrine-secreting tumor on the adrenal gland called a pheochromocytoma. The excess epinephrine caused by the tumor increases heart rate and contractility to increase cardiac output. Treatment for this condition includes surgical removal of the tumor, which normally corrects the hypertension.

**Exercise Treatment**

For the ACE-AHFS, it is important that clients have appropriate medical clearance from their healthcare providers prior to beginning an exercise program. Incorporation of exercise and a healthy lifestyle is a cornerstone therapy in the treatment of hypertension. However, proper medical clearance is necessary to ensure...
that exercise is appropriate and will not lead to potential cardiovascular events in unstable hypertensive clients. After a diagnosis of hypertension is made, further medical evaluation is necessary to assess lifestyle, identify other cardiovascular risk factors, and reveal potential identifiable causes of hypertension (see Table 8-1). Medical evaluation is also necessary to assess the presence or absence of target organ damage and cardiovascular disease. In addition to a thorough physical examination, the following routine laboratory tests are recommended:

- An electrocardiogram to assess rate, rhythm, and structural changes in the heart
- Fasting blood glucose measurements to examine the presence or absence of diabetes
- Serum potassium, creatinine, or other measure of glomerular filtration rate to assess kidney function
- A lipid profile that includes the breakdown of high- and low-density lipoprotein concentrations

These laboratory tests help to identify additional risk factors for cardiovascular disease and potential contributing factors to hypertension. If warranted, clinicians may want clients to undergo supervised exercise testing to ensure that it is safe for them to engage in a regular exercise program. Furthermore, these tests are helpful for clinicians to prescribe the appropriate individualized pharmacological and non-pharmacological treatments for hypertension. Table 8-6 lists the major cardiovascular risk factors and associated diseases caused by hypertension-induced target organ damage.

Aerobic exercise training is a cornerstone in the prevention and treatment of hypertension. Exercise training induces many physiological adaptations that lower BP both acutely and chronically. In uncomplicated cases of prehypertension or stage I hypertension, aerobic exercise training along with dietary modification may be sufficient to lower BP to reach the target BP without the addition of pharmacotherapy. In more complicated cases of hypertension, the addition of regular aerobic exercise to pharmacotherapy has an additive effect on BP reduction. Exercise training also modifies other cardiovascular risk factors, including blood lipoprotein profile, insulin sensitivity, and body composition, which combine to decrease cardiovascular risk.

### Acute Effects of Exercise

Dynamic exercise induces an acute post-exercise reduction in both SBP and DBP. This response is known as **post-exercise hypotension (PEH)**. The PEH cardiovascular response is characterized by a reduction in peripheral vascular resistance that is not compensated for by an increase in cardiac output, resulting in a decrease in BP (Halliwill, 2001). On average, the magnitude of the effect of PEH on BP in hypertensive individuals is approximately 15 and 4 mmHg on SBP and DBP, respectively, and can persist for up to 22 hours following an exercise bout (Pescatello et al., 2004). The PEH response occurs in normotensive and hypertensive men and women of all ages, although the largest reductions in BP occur in hypertensive individuals. This acute exercise-induced reduction in BP is clinically significant. It is unknown whether there is a dose-response effect of exercise duration and intensity on PEH, with longer duration or higher intensity of exercise resulting in a greater reduction in BP. It is known that PEH occurs with relatively short-duration bouts of

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**Table 8-6**

<table>
<thead>
<tr>
<th>Cardiovascular Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Cigarette smoking</td>
</tr>
<tr>
<td>Obesity (body mass index ≥30 kg/m²)</td>
</tr>
<tr>
<td>Physical inactivity</td>
</tr>
<tr>
<td>Dyslipidemia</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Microalbuminuria or estimated GFR &lt;60 mL/min</td>
</tr>
<tr>
<td>Age (older than 55 for men, 65 for women)</td>
</tr>
<tr>
<td>Family history of premature cardiovascular disease (men under age 55 or women under age 65)</td>
</tr>
</tbody>
</table>

*Note: GFR = Glomerular filtration rate*

Hypertension

Chapter eight

Hypertensive Medications and Cardiovascular Response

There are a number of classes of antihypertensive medications. Although lifestyle modification is a cornerstone therapy for treatment of prehypertension and hypertension, most hypertensive individuals will require two or more medications to reach their target BP. It is important that an ACE-AHFS knows the medications his or her clients are taking, how they act on the cardiovascular system to lower BP, and any effects they have on the cardiovascular responses to exercise. Table 8-7 provides a list of different antihypertensive drugs arranged by their class.

Diuretics

Diuretics are the most commonly prescribed BP-lowering drugs and are typically the first-line antihypertensive drugs prescribed for hypertensive patients. This drug class has been used for many years and is very effective at lowering BP, especially in cases in which the hypertension is caused by excess extracellular fluid volume. Diuretics initially work to decrease BP by stimulating the excretion of sodium in the proximal tubule of the nephron (the functional unit of the kidneys). To maintain osmotic balance, water follows the sodium, resulting in a loss of extracellular fluid volume. Diuretics initially work to decrease BP by stimulating the excretion of sodium in the proximal tubule of the nephron (the functional unit of the kidneys). To maintain osmotic balance, water follows the sodium, resulting in a loss of extracellular fluid volume. It is hypothesized that the excess sodium contributes to the peripheral blood vessels’ rigidity, thus promoting an increase in peripheral vascular resistance. The long-term BP-lowering capabilities of diuretics may be a result of decreasing sodium and indirectly lowering blood vessel rigidity (Kester et al., 2007).

Clients taking diuretics to manage their hypertension should be instructed to pay particular attention to hydration during exercise, especially when exercising in warm environments. Because diuretics decrease plasma volume, hypertensive clients can easily become dehydrated during exercise. Clients should be instructed to drink fluids throughout exercise to replace the fluid that they are losing through sweat. The volume of sweat lost can be
## Table 8-7
**Oral Antihypertensive Drugs**

<table>
<thead>
<tr>
<th>Class</th>
<th>Drug (Trade Name)</th>
<th>Usual Dose Range (mg/day)</th>
<th>Usual Daily Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Thiazide diuretics</strong></td>
<td>Chlorothiazide (Diuril)</td>
<td>125–500</td>
<td>1–2</td>
</tr>
<tr>
<td></td>
<td>Chlorothalidone (generic)</td>
<td>12.5–25</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Hydrochlorothiazide (Microzide, HydroDIURIL†)</td>
<td>12.5–50</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Polythiazide (Renese)</td>
<td>2–4</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Inclapamide (Lozol†)</td>
<td>1.25–2.5</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Metolazone (Mykrox)</td>
<td>0.5–1.0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Metolazone (Zaroxolyn)</td>
<td>2.5–5</td>
<td>1</td>
</tr>
<tr>
<td><strong>Loop diuretics</strong></td>
<td>Bumetanide (Bumex†)</td>
<td>0.5–2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Furosemide (Lasix†)</td>
<td>20–80</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Torsemide (Demadex†)</td>
<td>2.5–10</td>
<td>1</td>
</tr>
<tr>
<td><strong>Potassium-sparing diuretics</strong></td>
<td>Amloride (Midamore†)</td>
<td>5–10</td>
<td>1–2</td>
</tr>
<tr>
<td></td>
<td>Triamterene (Dyrenium)</td>
<td>50–100</td>
<td>1–2</td>
</tr>
<tr>
<td><strong>Aldosterone receptor blockers</strong></td>
<td>Eplerenone (Inspra)</td>
<td>50–100</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Spironolactone (Aldactone†)</td>
<td>25–50</td>
<td>1</td>
</tr>
<tr>
<td><strong>Beta blockers</strong></td>
<td>Atenolol (Tenormin†)</td>
<td>25–100</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Betaxolol (Kerlone†)</td>
<td>5–20</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Bisopropol (Zebeta†)</td>
<td>2.5–10</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Metoprolol (Lopressor†)</td>
<td>50–100</td>
<td>1–2</td>
</tr>
<tr>
<td></td>
<td>Metoprolol extended release (Toprol XL)</td>
<td>50–100</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Nadolol (Corgard†)</td>
<td>40–120</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Propanolol (Inderal†)</td>
<td>40–160</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Propanolol long-acting (Inderal LA†)</td>
<td>60–180</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Timolol (Blocadren†)</td>
<td>20–40</td>
<td>2</td>
</tr>
<tr>
<td><strong>Beta blockers with intrinsic sympathomimetic activity</strong></td>
<td>Acebutolol (Sectral†)</td>
<td>200–800</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Penbutolol (Levatol)</td>
<td>10–40</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Pindolol (generic)</td>
<td>10–40</td>
<td>2</td>
</tr>
<tr>
<td><strong>Combined alpha and beta blockers</strong></td>
<td>Carvedilol (Coreg)</td>
<td>12.5–50</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Labetalol (Normodyne, Trandate†)</td>
<td>200–800</td>
<td>2</td>
</tr>
<tr>
<td><strong>ACE inhibitors</strong></td>
<td>Benazepril (Lotensin†)</td>
<td>10–40</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Captopril (Capoten†)</td>
<td>25–100</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Enalapril (Vasotec†)</td>
<td>5–40</td>
<td>1–2</td>
</tr>
<tr>
<td></td>
<td>Fosinopril (Monopril)</td>
<td>10–40</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Lisinopril (Prinivil, Zestril†)</td>
<td>10–40</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Moexipril (Univasc)</td>
<td>7.5–30</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Perindopril (Aceon)</td>
<td>4–8</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Quinapril (Accupril)</td>
<td>10–80</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Ramipril (Altace)</td>
<td>2.5–20</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Trandolapril (Mavik)</td>
<td>1–4</td>
<td>1</td>
</tr>
<tr>
<td><strong>Angiotensin II antagonists</strong></td>
<td>Candesartan (Atacand)</td>
<td>8–32</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Eprosartan (Teveten)</td>
<td>400–800</td>
<td>1–2</td>
</tr>
<tr>
<td></td>
<td>Irbesartan (Avapro)</td>
<td>150–300</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Losartan (Cozaar)</td>
<td>25–100</td>
<td>1–2</td>
</tr>
<tr>
<td></td>
<td>Olmesartan (Benicar)</td>
<td>20–40</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Telmisartan (Micardis)</td>
<td>20–80</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Valsartan (Diovan)</td>
<td>80–320</td>
<td>1–2</td>
</tr>
</tbody>
</table>
in cardiac output. Blocking the beta receptors therefore decreases heart rate and contractility, collectively causing a decreased cardiac output. Initially, the antihypertensive effect of beta blockers is due to this decrease in cardiac output.

Beta Blockers
There are many different types of beta blockers with different specificities and mechanisms of action. In general, beta blockers primarily work to lower BP by antagonizing the beta receptors in the heart and the kidneys. In the heart, normal stimulation of beta receptors by epinephrine increases heart rate and increases calcium entry into the myocardial cells, thereby increasing contractility. Stimulation of these receptors causes an increase in cardiac output. Blocking the beta receptors therefore decreases heart rate and contractility, collectively causing a decreased cardiac output. Initially, the antihypertensive effect of beta blockers is due to this decrease in cardiac output.

Beta blockers also inhibit renin release as a result of sympathetic nerve stimulation to the kidney. Recall that the release of renin stimulates a cascade of events that cause peripheral vasconstriction through angiotensin II, as well as salt and water retention in the kidneys through the RAAS (see Figure 8-1). Thus, blocking the beta receptors in the kidneys inhibits this cascade of events and results in a passive vasodilation of the peripheral vasculature, thereby lowering peripheral vascular resistance.
Hypertension

Beta blockers blunt the normal elevation in heart rate that is observed during exercise. Therefore, gauging exercise intensity via target heart rate when working with hypertensive clients who are taking beta blockers is not appropriate. Instead, ratings of perceived exertion (RPE) of “somewhat hard,” which equates to a 13 on the Borg scale (6 to 20 scale), should be used to evaluate exercise intensity. This perceived exertion correlates well with exercise intensity in the absence of an appropriate exercise heart-rate response due to the beta-blocker effect.

In addition to the heart-rate response, there are additional precautions that an ACE-AHFS should be aware of with this class of drugs. First, beta blockers can sometimes mask the symptoms of hypoglycemia (low blood sugar). Because exercise also decreases blood sugar, significant hypoglycemia during exercise can sometimes occur in clients taking beta blockers. Second, some clients taking beta blockers may complain of exercise intolerance due to the blunted heart-rate response. If this occurs, it is important that the client sees his or her healthcare provider to adjust the dosage of medication. The client must not abruptly stop taking his or her medication, as doing so can result in rebound hypertension. Lastly, beta blockers can cause fatigue, sedation, depression, and sexual dysfunction. If a client complains of these symptoms, it is important that he or she sees his or her healthcare provider.

Angiotensin Converting Enzyme (ACE) Inhibitors

ACE inhibitors block the conversion of angiotensin I to angiotensin II in the RAAS (see Figure 8-1), which results in an inhibition of peripheral vasoconstriction mediated by angiotensin II in the vasculature. It also results in an inhibition of aldosterone release from the adrenal cortex, thus preventing sodium and water reabsorption in the kidney. Recall that BP is integratively controlled by many physiological systems working in concert. In some cases, when one component of a body system is altered by pharmacology (such as causing a reduction in peripheral vascular resistance), there is a compensatory response by the other systems (increased cardiac output and sympathetic nerve activity) in an attempt to return to a homeostatic balance. However, in the case of hypertension, the desired effect of the drug treatment is to lower BP, avoiding the compensatory changes that may occur in other systems that regulate BP. One of the advantages of ACE inhibitors is that they cause vasodilation of the peripheral vasculature without inducing a compensatory increase in sympathetic nerve activity.

Angiotensin-receptor Blockers (ARBs)

ARBs are similar to ACE inhibitors in the way that they lower BP. However, instead of inhibiting the production of angiotensin II, they block the receptor on which angiotensin II acts. ARBs cause a decrease in peripheral vasoconstriction, resulting in a decrease in peripheral vascular resistance. Furthermore, ARBs also inhibit the release of aldosterone from the adrenal cortex, which ultimately inhibits sodium and water reabsorption in the kidneys.

Aldosterone-receptor Antagonists

Aldosterone-receptor blockers inhibit the effect of aldosterone on the kidney. Thus, sodium and water are not reabsorbed and plasma volume is decreased.

ACE inhibitors, ARBs, and aldosterone-receptor antagonists can all cause hyperkalemia (increase in serum potassium levels). When clients are taking these medications, it is important that the ACE-AHFS monitors them for signs of electrolyte imbalances. Signs and symptoms of hyperkalemia can include a general feeling of fatigue, muscle weakness, nausea, tingling sensations, and, most seriously, slow heart beat and a weak pulse.

Alpha Blockers

This class of drugs works by inhibiting the alpha 1 receptors in the peripheral blood vessels, leading to a reduction in vasoconstriction and reduced peripheral vascular resistance. However, when the alpha 1 receptors are blocked, there is a compensatory increase in heart rate in an attempt to increase BP to achieve homeostatic balance. The baroreceptors send signals to the
cardiovascular control centers that BP is low and a response ensues through the parasympathetic and sympathetic nervous systems to increase heart rate. However, the effect of the increased sympathetic activity on the peripheral vasculature is blocked by the drug. Alpha 1 blockers are no longer routinely used to manage hypertension.

A common side effect of alpha 1 blockers is orthostatic hypotension. If clients are taking this drug class, they should be reminded to change body positions slowly and to not abruptly stop exercising. In addition, clients should be instructed to do an extended cool-down to limit the potential for a rapid decrease in BP with the cessation of exercise.

**Calcium Channel Blockers**

Calcium (Ca++) channel blockers prevent calcium from entering the cardiac and vascular smooth muscle cells. This in turn causes a decrease in cardiac contractility, a decrease in conduction of the electrical signal that controls heart rate, and a decrease in peripheral blood vessel vasoconstriction. The BP-lowering action of Ca++ channel blockers is therefore two-fold, in that they reduce both cardiac output (decreased stroke volume and heart rate) and peripheral vascular resistance (decreased vasoconstriction). However, some types of Ca++ channel blockers have a greater effect on the heart and others have a greater effect on the peripheral blood vessels.

In relation to exercise training, similar to beta blockers these drugs cause heart rate to be slowed (bradycardia) and can cause a substantial drop in BP upon standing. Clients taking these drugs should be advised to use RPE to monitor exercise intensity instead of a target heart rate, prolong the cool-down, and change body positions slowly.

**Central Acting Alpha 2 Blockers**

Central acting alpha 2 blockers work in the BP control centers in the brain to reset and lower the BP set point, which reduces sympathetic activity to the heart and increases parasympathetic activity, resulting in a slowing of the heart rate.

In addition, the reduction in sympathetic activity to the peripheral blood vessels and kidneys decreases vasoconstriction, which decreases renin release and vasoconstriction, resulting in decreased peripheral vascular resistance.

This class of antihypertensive drugs is rarely used in cases of essential hypertension. They have several side effects, including severe sedation. Orthostatic hypotension (low BP upon standing) is also common with central acting alpha 2 blockers, so caution should be used when changing body positions if clients are taking this class of antihypertensive drugs.

**Peripheral Vasodilators**

These drugs cause vasodilation and reduce BP by relaxing the vascular smooth muscle in the peripheral blood vessels. Typically, peripheral vasodilators are only used in combination with other antihypertensive drugs in cases of resistant hypertension or a hypertensive crisis.

**The Role of Exercise in Hypertension Prevention**

Unequivocally, regular physical activity and exercise training are associated with a lower incidence of cardiovascular disease. In broad survey-based epidemiological studies, there is a negative association between exercise training and the development of hypertension. Individuals with the highest levels of physical activity or who participate in vigorous sporting activities show the lowest incidence of hypertension. Furthermore, a higher fitness level is also associated with a lower risk for developing hypertension. People with a low fitness level have a higher relative risk of developing hypertension compared with highly fit people (Blair et al., 1984).

Overall, exercise training and greater baseline fitness levels are associated with a lower incidence of hypertension. While exercise training is pivotal for the prevention and treatment of hypertension, hypertension is a multifaceted pathology and is caused by an interaction of genes and environmental factors. There are certain populations with a strong genetic propensity for hypertension, and
Chapter Eight

Hypertension

Exercise training may delay the onset of hypertension and/or decrease the severity of the disease.

Exercise-related Treatment and Management of Hypertension

Numerous studies have been conducted to examine the effect of regular aerobic exercise on BP at rest, throughout the day (ambulatory), and during exercise. These studies have examined the effect of dynamic moderate-intensity aerobic exercise such as walking, jogging, running, and cycling. These studies show that resting BP is significantly reduced after aerobic training. The magnitude of the reduction in resting BP is greatest in hypertensive individuals, although people with normal BP still show a reduction in their resting BP after aerobic training. Dynamic exercise training also reduces BP throughout the day and during an acute bout of exercise.

Cardiovascular Responses to Exercise Training

There are many cardiovascular adaptations that cause a reduction in BP as a result of exercise training. Many of these adaptations positively affect BP both at rest and during acute bouts of exercise. An examination of the determinants of mean arterial pressure reveal that for BP to be reduced, a decrease in either cardiac output or total peripheral resistance must occur. With exercise training, there are alterations in the determinants of cardiac output, but the primary mechanism for the decrease in BP is through a decrease in peripheral vascular resistance. The BP-lowering effects of exercise training occur as a result of changes to the systems that integratively control BP.

Changes to the Sympathetic Nervous System

One of the changes that occur as a result of exercise training takes place in the sympathetic nervous system. Essential hypertension is associated with an increase in the nerve traffic from the sympathetic nervous system to the heart and the peripheral blood vessels. In general, there is a decrease in sympathetic nerve traffic with exercise training. Some studies have shown that direct measures of sympathetic nerve traffic at rest decrease after exercise training (Ray & Hume, 1998), though not all studies have been able to consistently replicate this finding (Morlin, Wallin, & Eriksson, 1983).

An additional way to indirectly measure globalized systemic changes in sympathetic nerve activity is to measure the concentration of norepinephrine (the sympathetic adrenergic neurotransmitter) in the blood. Recall that the sympathetic nerves release norepinephrine that binds to receptors on blood vessels and causes the peripheral blood vessels to vasoconstrict. Measuring the amount of norepinephrine in the blood is an indirect assessment of sympathetic nerve activity, because the absolute concentration is influenced by how much norepinephrine is released and how well it is cleared. In individuals with hypertension, norepinephrine concentration in the blood has been shown to decrease after exercise training (Meredith, 1990).

Changes to the Renin-Angiotensin-Aldosterone System

The renin-angiotensin-aldosterone system (RAAS) is involved in the long-term regulation of BP. It would be expected that changes to this system would contribute to the reduction in BP observed with exercise training. Decreased renin and angiotensin II are observed in normotensive individuals after exercise training and contribute to the reduction in BP. However, this response is not observed in hypertensive subjects after exercise training (Pescatello et al., 2004).

Changes to the Peripheral Blood Vessels

One prominent change induced by exercise training in hypertensive individuals takes place in the peripheral blood vessels. Exercise training reduces the amount of vasoconstriction that occurs when norepinephrine binds to the receptors on the blood vessels. In addition, there are changes in the locally produced vasoconstrictors and vasodilators in the blood vessels. The two most prominent changes are to a vasoconstrictor...
cardiovascular pathologies like hypertension. Most heat-related injuries and deaths occur in individuals with cardiovascular pathology (McGeehin & Mirabelli, 2001). There are several underlying factors contributing to this increased risk, including impairments in the cardiovascular response to heat stress directly caused by the hypertensive disease as well as the inability for the cardiovascular system to respond appropriately during heat stress caused by the pharmacology used to treat hypertension.

Hypertensive individuals have a reduction in skin blood flow during heat stress, which results in a decreased ability to transfer body heat to the environment (Kenney, 1985). The decrease in heat transfer results in a significant increase in body core temperature. There is also a significant reduction in skin blood flow in hypertensive individuals, which is related to structural alteration in the skin blood vessels and to changes in the locally produced signaling molecules in the skin that allow the skin blood vessels to vasodilate during heat stress (Holowatz & Kenney, 2007).

In hypertensive individuals, the increase in cardiac work during heat stress is especially dangerous in individuals with cardiac or other end organ damage (left ventricular hypertrophy or coronary artery disease).

Exercise training has beneficial thermoregulatory effects in hypertensive individuals. Exercise training sufficient to increase VO2 max strengthens the central cardiovascular system. The amount of cardiac work during exercise at the called endothelin 1 and the vasodilator nitric oxide. After exercise training, endothelin 1 is reduced and nitric oxide is increased (Pescatello et al., 2004). Long-term exercise training can also cause beneficial adaptations of the structure of the blood vessels themselves, increasing their elasticity. Together, reduced responsiveness to norepinephrine, decreased endothelin 1, increased nitric oxide, and structural adaptations in the blood vessels result in decreased vascular resistance and contribute to the reduction in BP.

Finally, just as there is a complex interaction among genetic and environmental factors in the development of hypertension, there is significant variation in the response to exercise training. Certain genetic profiles may respond more favorably to exercise training. It has been suggested that genetic factors can explain some of the reduction in systolic BP after exercise training (Pescatello et al., 2004).

**Thermoregulation**

Individuals with hypertension have a diminished ability to dissipate body heat to the environment during heat stress (Kenney, 1985). Thermal heat stress occurs during exercise, passive exposure to hot and humid environments, or the combination that comes with exercising in the heat. The normal physiological response to rising body core temperature during heat stress includes activation of the sympathetic nervous system, which causes an integrated cardiovascular response in an effort to increase blood flow to the skin for thermoregulation. Increased skin blood flow allows warm blood from the body core to flow through the cooler skin circulation, where heat is lost through convection. In addition, the evaporation of sweat from the skin cools the skin and serves as a major avenue of heat loss. The integrated cardiovascular response to heat stress includes an increase in heart rate and cardiac contractility, which together increase cardiac output and vasoconstriction in the renal and the splanchnic circulations, thereby allowing the redistribution of blood flow to the skin (Rowell, 1974).

Heat stress significantly challenges the cardiovascular system, especially in individuals with cardiovascular pathologies like hypertension. Most heat-related injuries and deaths occur in individuals with cardiovascular pathology (McGeehin & Mirabelli, 2001). There are several underlying factors contributing to this increased risk, including impairments in the cardiovascular response to heat stress directly caused by the hypertensive disease as well as the inability for the cardiovascular system to respond appropriately during heat stress caused by the pharmacology used to treat hypertension.

Hypertensive individuals have a reduction in skin blood flow during heat stress, which results in a decreased ability to transfer body heat to the environment (Kenney, 1985). The decrease in heat transfer results in a significant increase in body core temperature. There is also a significant reduction in skin blood flow in hypertensive individuals, which is related to structural alteration in the skin blood vessels and to changes in the locally produced signaling molecules in the skin that allow the skin blood vessels to vasodilate during heat stress (Holowatz & Kenney, 2007).

In hypertensive individuals, the increase in cardiac work during heat stress is especially dangerous in individuals with cardiac or other end organ damage (left ventricular hypertrophy or coronary artery disease).

In addition to the hypertension-induced physiological changes that occur with heat stress, many of the pharmacological treatments for hypertension also blunt the integrated cardiovascular response to heat stress. Specifically, diuretics decrease plasma volume. Individuals taking diuretics can easily become dehydrated during heat stress, and dehydration alone decreases blood flow to the skin and impairs thermoregulation. Other drugs that adversely affect the cardiovascular response to heat stress include beta blockers, Ca++ channel blockers, and alpha blockers. These drugs impair the central cardiovascular and peripheral blood flow responses that occur during heat stress.

Exercise training has beneficial thermoregulatory effects in hypertensive individuals. Exercise training sufficient to increase VO2 max strengthens the central cardiovascular system. The amount of cardiac work during exercise at the
same absolute workload will be less after exercise training. In addition, peripheral sweating and blood vessel changes allow individuals to start sweating and vasodilating their skin blood vessels at lower body core temperatures. Together, these adaptations confer positive benefits on thermoregulation.

Training Guidelines

Programming and Progression Guidelines and Considerations
Prior to beginning a new exercise program with a hypertensive client, several important safety issues should be addressed. In normal healthy individuals, the initiation of exercise acutely increases SBP. This acute increase in SBP is normal and necessary to increase blood flow to the exercising muscle to deliver oxygen and clear metabolic by-products. However, some hypertensive individuals experience an exaggerated increase in SBP and/or DBP during dynamic exercise, which is predictive of future morbidity from cardiovascular disease. Depending on the initial resting BP and whether the client has target organ damage or additional cardiovascular risk factors, a medically supervised exercise-tolerance test should be conducted. Table 8-8 presents the recommendations for exercise testing prior to engaging in a regular exercise program (Simons-Morton, 2008b). Furthermore, these high-risk clients may benefit from a medically supervised cardiac rehabilitation program. Clients may be unaware that they may qualify for such programs.

Table 8-9 lists the general indications set forth by American College of Sports Medicine (ACSM) for the termination of exercise. An absolute contraindication to exercise is if resting SBP is greater than 200 mmHg or if DBP is greater than 115 mmHg.

Cardiovascular Training
Exercise programming guidelines following the FITT principle (frequency, intensity, time, and type) are suitable for individuals with hypertension. The Centers for Disease Control and Prevention (CDC) and ACSM have endorsed the recommendation that, “Every U.S. adult should accumulate 30 minutes or more of moderate-intensity physical activity on most, preferably all, days of the week” (Pescatello et al., 2004).

The optimal cardiovascular training guidelines necessary to see a reduction in BP are unclear. To a certain extent, the dose of exercise needed to observe a reduction in BP is related to initial fitness level. In sedentary individuals, clinically significant reductions in BP can be observed with relatively modest increases in physical activity. Researchers have found that significant reductions in resting systolic and diastolic BP can occur with as little as 60 minutes of exercise at an intensity

Table 8-8
Recommendations for Exercise Testing Before Exercise Participation

<table>
<thead>
<tr>
<th>Exercise intensity</th>
<th>No TOD or CVD; no risk factors, no symptoms, BP &lt;180/110 mmHg</th>
<th>No symptoms; BP &lt;180/110 mmHg</th>
<th>Symptoms or BP &gt;180/110 mmHg</th>
<th>Known TOD or CVD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate</td>
<td>Not necessary</td>
<td>Not necessary</td>
<td>Recommended</td>
<td>Recommended</td>
</tr>
<tr>
<td>Vigorous</td>
<td>Not necessary</td>
<td>Not necessary</td>
<td>Recommended</td>
<td>Recommended</td>
</tr>
</tbody>
</table>

Notes: TOD and CVD include ischemic heart disease, heart failure, stroke, renal disease, neuropathy, or retinopathy; also includes diabetes as a “CVD equivalent” CVD risk factors include hypertension, smoking, dyslipidemia, age older than 60 years, male gender or postmenopausal woman, family history of cardiovascular disease (in women <65 years or in men <55 years). BP = Blood pressure; TOD = Target organ damage; CVD = Cardiovascular disease

experience the beneficial effects of exercise at an intensity of 40 to 60% of \( \dot{V}O_2 \text{max} \). This exercise intensity corresponds to approximately a 12 to 13 on the Borg RPE scale (6 to 20 scale). Using the RPE scale to gauge exercise intensity is beneficial, especially when clients are taking antihypertensive medications that alter the cardiovascular responses to exercise (e.g., beta blockers).

**Time**

The duration of exercise should be at least 30 minutes to have a beneficial effect on cardiovascular health. This 30-minute duration can be continuous or intermittent, but if intermittent exercise is conducted, the bouts should be at least 10 minutes in length and total 30 to 60 minutes of exercise each day. This can also be expressed in terms of calories burned (700 kcal/week is a good initial goal, progressing to 2000 kcal/week).

**Type**

Aerobic, endurance-type exercise such as walking, jogging, running, swimming, and cycling is recommended. Walking is one of the easiest exercise modalities to start with, especially in previously sedentary clients. Any physical activity that engages the large muscle groups and is rhythmic and aerobic will be beneficial. The type of exercise performed should be individualized so that clients are more likely to adhere to their programs. Thus, the exercises should be relatively simple and offer some variety.

Table 8-10 summarizes the cardiovascular exercise recommendations for hypertensive clients.

**Progression**

The progression of exercise training in hypertensive clients should follow the basic principles of approximately 50% of \( \dot{V}O_2 \text{max} \) per week (Ishikawa-Takata, Ohta, & Tanaka, 2003). There is an even greater reduction when the exercise duration was increased to 90 minutes.

**Frequency**

Studies have consistently shown that training frequencies between three and five days a week will cause a significant reduction in BP. However, increasing the frequency of exercise training to most days of the week may confer additional BP-lowering benefits for hypertensive individuals. These additional benefits are likely due to the acute BP-lowering effects of exercise (i.e., post-exercise hypotension).

**Intensity**

The intensity of aerobic exercise for hypertensive clients should be moderate. Significant reductions in BP are observed with exercise intensities between 40 and 70% of \( \dot{V}O_2 \text{max} \). Exercise intensities higher than 70% of \( \dot{V}O_2 \text{max} \) do not cause greater reductions in BP and may in fact blunt the BP-lowering effect of exercise (Hagberg, Park, & Brown, 2000). Thus, hypertensive clients

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**Table 8-9**

**General Indications for Stopping Exercise**

- Onset of angina or angina-like symptoms
- Drop in SBP of >10 mmHg from baseline despite an increase in workload
- Excessive rise in blood pressure: SBP >250 mmHg or DBP >115 mmHg
- Signs of poor perfusion: lightheadedness, confusion, ataxia, pallor, cyanosis, nausea, or cold and clammy skin
- Failure of heart rate to increase with increased exercise intensity
- Noticeable change in heart rhythm
- Subject requests to stop
- Physical or verbal manifestations of severe fatigue
- Failure of the exercise equipment

**Note:** SPB = Systolic blood pressure; DBP = Diastolic blood pressure


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**Table 8-10**

**Cardiovascular Training Recommendations for Hypertensive Clients**

| Frequency: On most, preferably all, days of the week | Intensity: Moderate (40–60% \( \dot{V}O_2 \text{max} \) or 12–13 RPE) | Time: 30–60 minutes of continuous or accumulated physical activity | Type: Rhythmic aerobic exercise that targets large muscle groups; should be individualized |
of an initial conditioning stage, an improvement stage, and a maintenance stage that is tailored to the client’s initial fitness level and attains the client’s specific fitness goals. In general, hypertensive clients should progress to exercising three to seven days per week, for 30 to 60 minutes, at an intensity of 40 to 60% of \( \text{VO}_2\text{max} \). The initial conditioning stage should include a moderate level of aerobic exercise that causes minimal muscle soreness or discomfort. For a hypertensive client, this may include walking for two 10-minute bouts twice a week (Simons-Morton, 2008b). This stage may last up to four weeks depending on the adaptation of the individual to the training program. The duration of an exercise session during this initial stage may begin at 15 to 20 minutes and progress to 30 minutes three to four days per week. During the improvement stage, the intensity of exercise is progressively increased every two to three weeks until the client is able to exercise at a moderate intensity for 20 to 30 minutes. During the maintenance stage, the exercise frequency, intensity, and time are maintained while program goals are reviewed and new goals are set.

**Resistance Training**

There has been significant controversy surrounding the safety of resistance training for hypertensive clients. During isometric exercise (resistance), both SBP and DBP increase. The magnitude of the increase in BP with resistance exercise is related to the intensity of exercise and the amount of muscle mass involved in the exercise. This increase in BP is potentially dangerous for hypertensive clients because heavy resistance training can cause large increases in both SPB and DBP. In general, moderate resistance training is beneficial and safe for this population, as long as the hypertension is controlled. According to the guidelines from the AHA, resistance training is contraindicated in individuals with unstable angina (chest pain), uncontrolled hypertension (systolic BP \( \geq 160 \text{ mmHg} \) and/or diastolic BP \( \geq 100 \text{ mmHg} \)), uncontrolled cardiac arrhythmias, a recent history of congestive heart failure, significant heart valve disease, or pathological enlargement of the heart (hypertrophic cardiomyopathy) (Braith & Stewart, 2006). While aerobic exercise is the mainstay in the treatment of hypertension, it can be safely supplemented with resistance training.

Resistance training offers several beneficial physiological effects. It increases muscular strength, endurance, and mass. The increase in muscle mass causes an increased basal metabolic rate (BMR). Perhaps one of the most beneficial effects of resistance training on BP results from the positive effects on insulin sensitivity. There is a significant link between decreased insulin sensitivity and hypertension. Because consistent and long-term resistance training increases insulin sensitivity, this may be one of the mechanisms mediating the reduction in BP. Resistance training also attenuates the rate-pressure product (an index of cardiac work) when lifting any given load, which decreases the demand on the heart when performing activities of daily living (ADL) and various work- and recreation-related tasks:

\[
\text{Rate-pressure product} = \text{Heart rate (HR)} \times \text{Systolic blood pressure (SBP)}
\]

In addition, resistance training is beneficial for the prevention and management of other chronic conditions, such as low back pain, osteoporosis, obesity, sarcopenia (the loss of skeletal muscle mass that may accompany aging), diabetes, and the susceptibility to falls. Resistance training of the major muscle groups decreases resting systolic and diastolic BP in hypertensive individuals by approximately 3 mmHg (Braith & Stewart, 2006). In studies examining different types of resistance training (circuit training vs. conventional), no difference in BP reduction was found among modalities. However, because circuit training uses lighter weights with limited rest periods between exercises, thereby introducing an aerobic component, it is the type of resistance training recommended for hypertensive clients.

A preliminary orientation with clients should establish appropriate weight loads, and the ACE-AHFS should instruct the client on proper lifting techniques and correct breathing patterns to avoid straining or performing the
Valsalva maneuver. Straining during resistance training causes significant and dangerous increases in BP. At minimum, one exercise per major muscle group should be performed. For example, a resistance-training program for a hypertensive client might include eight to 12 repetitions of the following exercises performed two to three days per week:

- Chest press
- Seated row
- Shoulder press
- Lower-back extension
- Triceps extension
- Biceps curl
- Abdominal crunch/curl-up
- Quadriceps extension or leg press
- Leg curls (hamstrings)
- Heel raises

Older or frail clients should initially perform 10 to 15 repetitions at a lower relative resistance to prevent injury. The ACE-AHFS can slowly increase the number of sets until clients are performing three sets two to three days per week.

Mind-body Exercise

Mind-body exercises such as yoga and tai chi are increasingly being incorporated into exercise training programs to promote flexibility, strength, and relaxation. There are few randomized controlled studies examining the effects of these alternative exercise programs specifically on high BP. The few studies that have been conducted suggest a beneficial reduction in BP attributable to both physical activity and relaxation (Santaella et al., 2006; La Forge, 2010).

Randomized clinical studies on the effects of hatha yoga on blood pressure demonstrate a large reduction in SBP with regular yoga therapy. These studies showed a 33 mmHg and 26 mmHg reduction in SBP after practicing yoga and biofeedback for six hours a week for 11 weeks (Patel & North, 1975; Murugesan, Govindarajulu, & Bera, 2000). The combined effects on BP of physical activity and relaxation, as practiced through mind-body exercise, appear to be synergistic, meaning that the combined effect is greater than the individual BP-lowering capabilities of either physical activity or relaxation alone (Cohen & Townsend, 2007).

Mind-body exercise practiced regularly (three times per week for 60 minutes) also improves balance and upper- and lower-body muscular strength and endurance (Taylor-Piliae et al., 2006). Further, these mind-body exercises also improve proprioceptive awareness. Several general precautions should be used when recommending mind-body exercise to hypertensive clients. First, many styles of hatha yoga involve isometric muscle contractions combined with dynamic movement of the body into different positions. This work should be done with caution, because isometric muscle contractions cause significant increases in BP. In addition, hypertensive clients taking certain antihypertensive medications may experience dramatic decreases in BP when changing body position. Specifically, diuretics, alpha blockers, calcium channel blockers, and beta blockers can cause orthostatic hypotension. Hypertensive clients should avoid holding strenuous poses, avoid inverted poses (e.g., downward-facing dog and shoulder stands), and be encouraged to transition slowly between poses. Caution should be used when practicing yoga with stage I and II hypertensive clients. Bikram yoga (rapidly paced yoga in very hot environments) should be avoided altogether because of the impaired thermoregulatory mechanisms seen in hypertensive clients.

Case Studies

Case Study 1

Edith is a 45-year-old woman who would like to start an exercise program to lose weight. She is 5’4” tall (1.6 m) and weighs 150 pounds (68 kg). Her physician has also told her that she has high total cholesterol, with a HDL cholesterol of 68 mg/dL and an LDL cholesterol of 140 mg/dL. She has been diagnosed with hypertension in the past, but admits that she gets very nervous when she visits her doctor’s office. She is currently not involved in a regular exercise program but is motivated to start an exercise program to...
lose weight. Edith’s diet is high in total calories, saturated fat, cholesterol, and sodium and low in fruits, vegetables, and fiber. After being seated in a quiet consulting room with both feet on the floor for five minutes, her resting BP is 138/86 mmHg. After an additional five minutes of sitting quietly, her BP drops to 130/82 mmHg.

What is Edith’s body mass index? 68 kg + (1.6 m)² = 26.6

According to the JNC VII guidelines, is Edith hypertensive? If so what stage of hypertension does she have? Edith has been diagnosed with hypertension in the past. However, her current BP measurements indicate that she is prehypertensive, as her readings fall within the 120/80 to 139/89 mmHg range.

What types of exercise should Edith incorporate into her new training program? How should an ACE-AHFS progress her training program? Edith should begin with an aerobic endurance-training program following the basic principles of an initial conditioning stage, an improvement stage, and a maintenance stage. In general, she should progress to exercising three to seven days per week, for 30 to 60 minutes, at an intensity of 40 to 70% of \( \text{VO}_{2\text{max}} \). The initial conditioning stage should include a moderate level of aerobic exercise that causes minimal muscle soreness or discomfort. This stage may last up to four weeks, depending on her adaptation to the training program. The duration of an exercise session during this initial stage may begin at 15 to 20 minutes and progress to 30 minutes three to four days per week. During the improvement stage, the intensity of exercise is progressively increased every two to three weeks until Edith is able to exercise at a moderate-to-vigorous intensity for 20 to 30 minutes. During the maintenance stage, the exercise frequency, intensity, and time are maintained while program goals are reviewed and new goals are set.

In general, moderate resistance training should be safe for Edith, as long as her hypertension is controlled. While aerobic exercise is the mainstay in the treatment of hypertension, it can be safely supplemented with resistance training. Because circuit training uses lighter weights with limited rest periods between exercises, thereby introducing an aerobic component, it is a good choice for Edith. However, she should be instructed on proper lifting techniques and correct breathing patterns to avoid straining or performing the Valsalva maneuver. Additionally, mind-body exercises such as yoga and tai chi may be incorporated into Edith’s exercise training program to promote flexibility, strength, and relaxation.

What other lifestyle modifications should Edith incorporate into her life? Edith can make the following lifestyle modifications (in addition to her exercise program) to help control her BP:

- Weight reduction of at least 10 pounds (4.5 kg)
- Limiting dietary sodium intake to less than 2400 mg per day
- Adopting a healthy eating plan that includes fresh fruits and vegetables, low-fat dairy products, and reduced saturated and total fat content (DASH eating plan)
- Avoiding or limiting alcohol consumption to no more than one drink per day

Case Study 2

Harry is a 60-year-old man who takes a diuretic and a beta blocker to control his blood pressure. His resting blood pressure with his medication is not very well controlled and today his blood pressure reading is 144/88 mmHg. Harry is relatively healthy, with the exception of his blood pressure, but he does have a strong family history of heart disease (his father died at age 44 from a heart attack). Harry has recently retired from his job and has found that his level of regular physical activity has decreased substantially. Because Harry had a physically demanding job, he has never engaged in a regular exercise program. Harry’s age, high blood pressure, family history of heart disease, and current sedentary lifestyle give him four cardiovascular risk factors.

Is Harry’s blood pressure adequately controlled with his medication? No, Harry’s BP appears unstable.

Should Harry have a supervised exercise test prior to engaging in a new exercise program? Since Harry’s BP is less than 180/110 mmHg and he has four cardiovascular disease risk factors, it is not necessary that he have a clinically supervised
exercise test prior to participating in a new low-to-moderate intensity (40 to 60% of \( \dot{V}O_2 \text{max} \)) exercise program. It may, however, be beneficial to conduct an exercise test if he plans to engage in vigorous-intensity exercise (>60% \( \dot{V}O_2 \text{max} \)).

**What exercise program should an ACE-AHFS recommend to Harry?** In general, an exercise program for Harry should follow the FITT principle and take place on most, preferably all, days of the week, at a moderate intensity (40 to 60% \( \dot{V}O_2 \text{max} \)), and be primarily endurance-type activity. Low-resistance, high-repetition resistance training and mind/body activity such as yoga or Pilates can supplement Harry’s regular endurance training. The ACE-AHFS should be prepared to assess Harry’s BP before, during, and after exercise or as recommended by his physician.

**Summary**

Exercise is a cornerstone therapy for the treatment and prevention of hypertension. Exercise therapy combined with dietary modification can prevent the development of hypertension in prehypertensive clients, and can have an additive effect with antihypertensive drugs in reducing blood pressure. Exercise therapy for all hypertensive clients should follow the FITT principle and take place on most, preferably all, days of the week, at a moderate intensity (40 to 60% \( \dot{V}O_2 \text{max} \)), and be primarily endurance-type activity. Low-resistance, high-repetition resistance training and mind/body exercise activities can also supplement regular endurance exercise in hypertensive clients.
References


Hypertension Prevention Research Group (1997). Effects of weight loss and sodium reduction intervention on blood pressure and hypertension incidence in overweight people with high-normal blood pressure: The Trials of Hypertension Prevention, phase II. Archives of Internal Medicine, 157, 657–667.


Suggested Reading


