



Exercise Standards for Testing and Training: A Statement for Healthcare Professionals From the American Heart Association Gerald F. Fletcher, Gary J. Balady, Ezra A. Amsterdam, Bernard Chaitman, Robert Eckel, Jerome Fleg, Victor F. Froelicher, Arthur S. Leon, Ileana L. Piña, Roxanne Rodney, Denise A. Simons-Morton, Mark A. Williams and Terry Bazzarre Circulation 2001;104;1694-1740 DOI: 10.1161/hc3901.095960 Circulation is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 72514 Copyright © 2001 American Heart Association. All rights reserved. Print ISSN: 0009-7322. Online ISSN: 1524-4539

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Exercise Standards for Testing and Training A Statement for Healthcare Professionals From the American Heart Association

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The purpose of this report is to provide revised standards and guidelines for the exercise testing and training of individuals who are free from clinical manifestations of cardiovascular disease and those with known cardiovascular disease. These guidelines are intended for physicians, nurses, exercise physiologists, specialists, technologists, and other healthcare professionals involved in exercise testing and training of these populations. This report is in accord with the "Statement on Exercise" published by the American Heart Association (AHA).¹

These guidelines are a revision of the 1995 standards of the AHA that addressed the issues of exercise testing and training.² An update of background, scientific rationale, and selected references is provided, and current issues of practical importance in the clinical use of these standards are considered. These guidelines are in accord with the American College of Cardiology (ACC)/AHA Guidelines for Exercise Testing.³

Exercise Testing

The Cardiovascular Response to Exercise

Exercise, a common physiological stress, can elicit cardiovascular abnormalities that are not present at rest, and it can be used to determine the adequacy of cardiac function. Because exercise is only one of many stresses to which humans can be exposed, it is more appropriate to call an exercise test exactly that and not a "stress test." This is particularly relevant considering the increased use of nonexercise stress tests.

Types of Exercise

Three types of muscular contraction or exercise can be applied as a stress to the cardiovascular system: isometric (static), isotonic (dynamic or locomotory), and resistance (a combination of isometric and isotonic).^{4,5} Isotonic exercise, which is defined as a muscular contraction resulting in movement, primarily provides a volume load to the left ventricle, and the response is proportional to the size of the working muscle mass and the intensity of exercise. Isometric exercise is defined as a muscular contraction without movement (eg, handgrip) and imposes greater pressure than volume load on the left ventricle in relation to the body's ability to supply oxygen. Cardiac output is not increased as much as in isotonic exercise because increased resistance in active muscle groups limits blood flow. Resistance exercise combines both isometric and isotonic exercise (such as free weight lifting).

Exercise Physiology

In the early phases of exercise in the upright position, cardiac output is increased by an augmentation in stroke volume mediated through the use of the Frank-Starling mechanism and heart rate; the increase in cardiac output in the latter phases of exercise is primarily due to an increase in heart rate. At fixed submaximal workloads below ventilatory threshold in healthy persons, steady-state conditions are usually reached within minutes after the onset of exercise; after this occurs, heart rate, cardiac output, blood pressure, and pulmonary ventilation are maintained at reasonably constant levels. During strenuous exertion, sympathetic discharge is maximal and parasympathetic stimulation is withdrawn, resulting in vasoconstriction in most circulatory body systems, except for that in exercising muscle and in the cerebral and coronary circulations. As exercise progresses, skeletal muscle blood flow is increased, oxygen extraction increases as much as 3-fold, total calculated peripheral resistance decreases, and

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systolic blood pressure, mean arterial pressure, and pulse pressure usually increase. Diastolic blood pressure may remain unchanged or decrease to a minimal degree. The pulmonary vascular bed can accommodate as much as a 6-fold increase in cardiac output without a significant increase in pulmonary artery pressure. In normal subjects, this is not a limiting determinant of peak exercise capacity. Cardiac output can increase as much as 4- to 6-fold above basal levels during strenuous exertion in the upright position, depending on genetic endowment and level of training. In the postexercise phase, hemodynamics return to baseline within minutes of termination. Vagal reactivation is an important cardiac deceleration mechanism after exercise; it is accelerated in well-trained athletes but may be blunted in deconditioned and/or "medically ill" patients.

Maximum Oxygen Uptake

Oxygen uptake quickly increases when dynamic exercise is begun or increased. During staged exercise testing, oxygen uptake usually remains relatively stable (steady state) after the second minute of each intensity of exercise below the ventilatory threshold. Maximal oxygen consumption ($\dot{V}O_{2 max}$) is the greatest amount of oxygen a person can take in from inspired air while performing dynamic exercise involving a large part of total muscle mass.⁶ It is considered the best measure of cardiovascular fitness and exercise capacity. $\dot{V}O_{2 max}$ represents the amount of oxygen transported and used in cellular metabolism. It is convenient to express oxygen uptake in multiples of sitting/resting requirements. One metabolic equivalent (MET) is a unit of sitting/resting oxygen uptake (\approx 3.5 mL of O₂ per kilogram of body weight per minute $[mL \cdot kg^{-1} \cdot min^{-1}]$). $\dot{V}O_{2 max}$ is influenced by age, sex, exercise habits, heredity, and cardiovascular clinical status. The ventilatory threshold is another measure of relative work effort, and it represents the point at which ventilation abruptly increases, despite linear increases in oxygen uptake and work rate. In most cases, the ventilatory threshold is highly reproducible, although it may not be achieved or readily identified in some patients, particularly those with very poor exercise capacity.7

Age

Maximum values of $\dot{V}o_{2 max}$ occur between the ages of 15 and 30 years and decrease progressively with age. At 60 years, mean $\dot{V}o_{2 max}$ in men is approximately two-thirds of that at 20 years. The decline in $\dot{V}o_{2 max}$ averages 8% to 10% per decade in both sedentary and athletic populations.⁶

Sex

A lower $\dot{V}_{O_{2 max}}$ in women is attributed to their smaller muscle mass, lower hemoglobin and blood volume, and smaller stroke volume compared with men.

Exercise Habits

Physical activity has an important influence on Vo_{2 max}. After 3 weeks of bed rest, there is a 25% decrease in $\dot{V}o_{2 max}$ in healthy men. In moderately active young men, $\dot{V}o_{2 max}$ is ≈ 12 METs, whereas individuals performing aerobic training such as distance running can have a $\dot{V}o_{2 max}$ as high as 18 to 24 METs (60 to 85 mL \cdot kg⁻¹ \cdot min⁻¹).

TABLE 1.	Normal	Values	of	Maximal	Oxygen	Uptake at
Different A	Ages					

•			
Age, y	Men	Women	
20-29			
$mL \cdot kg^{-1} \cdot min^{-1}$	43±7.2	$36{\pm}6.9$	
METs	12	10	
30–39			
$mL \cdot kg^{-1} \cdot min^{-1}$	42±7.0	34±6.2	
METs	12	10	
40–49			
$mL \cdot kg^{-1} \cdot min^{-1}$	40±7.2	32±6.2	
METs	11	9	
50–59			
$mL \cdot kg^{-1} \cdot min^{-1}$	36±7.1	$29{\pm}5.4$	
METs	10	8	
60–69			
$mL \cdot kg^{-1} \cdot min^{-1}$	33±7.3	$27{\pm}4.7$	
METs	9	8	
70–79			
$mL \cdot kg^{-1} \cdot min^{-1}$	29±7.3	$27\!\pm\!5.8$	
METs	8	8	

Values are expressed as mean \pm SD. MET indicates metabolic equivalent or 3.5 mL $0_2 \cdot kg^{-1} \cdot min^{-1}$.

Heredity

There is a natural variation in $\dot{V}O_{2\ max}$ that is related to genetic factors. 8,9

Cardiovascular Clinical Status

 $\dot{V}O_{2~max}$ is affected by the degree of impairment caused by disease. It is difficult to accurately predict $\dot{V}O_{2~max}$ from its relation to exercise habits and age because of considerable scatter and correlations that are generally low. Table 1 depicts normal values for age. The nomogram shown in Figure 1 expresses the concept of maximal METs predicted from peak treadmill workload by reflecting it in terms of that expected for age in men, with 100% being normal.¹⁰

 $\dot{V}O_{2\ max}$ is equal to the product of maximum cardiac output and maximum arteriovenous oxygen difference. Because cardiac output is equal to the product of stroke volume and heart rate and because stroke volume only increases to a certain level, $\dot{V}O_2$ is directly related to heart rate. The maximum arteriovenous $\dot{V}O_2$ difference (which increases with exercise) during exercise has a physiological limit of 15% to 17% volume; hence, if maximum effort is achieved, $\dot{V}O_2$ max can be used to estimate maximum cardiac output.

Myocardial Oxygen Uptake

Myocardial oxygen uptake is primarily determined by intramyocardial wall stress (ie, the product of left ventricular [LV] pressure and volume, divided by LV wall thickness), contractility, and heart rate. Other, less important factors include external work performed by the heart, the energy necessary for activation, and the basal metabolism of the myocardium.

Accurate measurement of myocardial oxygen uptake requires cardiac catheterization to obtain coronary arterial and

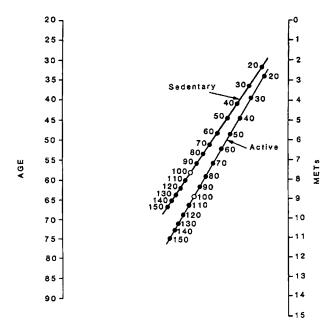


Figure 1. Nomogram based on age, METs, and activity status (sedentary vs active) that provides a percent of age-expected exercise capacity in men. For example, a 60-year-old man with a 3-MET capacity has 40% of the age-expected exercise capacity for sedentary men and 30% of that for active men.

venous oxygen content. Myocardial oxygen uptake can be estimated during clinical exercise testing by the product of heart rate and systolic blood pressure, which is called the double product or rate-pressure product. There is a linear relation between myocardial oxygen uptake and coronary blood flow. During exercise, coronary blood flow increases as much as 5-fold above the resting value. A subject with obstructive coronary artery disease (CAD) often cannot maintain adequate coronary blood flow to the affected region and supply the metabolic demands of the myocardium during exercise; consequently, myocardial ischemia occurs. Myocardial ischemia usually occurs at the same rate-pressure product rather than at the same external workload (eg, exercise test stage).

Heart Rate Response

The immediate response of the cardiovascular system to exercise is an increase in heart rate due to a decrease in vagal tone. This increase is followed by an increase in sympathetic outflow to the heart and systemic blood vessels. During dynamic exercise, heart rate increases linearly with workload and $\dot{V}o_2$. Heart rate will reach a steady state within minutes during low levels of exercise and at a constant work rate. As workload increases, the time necessary for the heart rate to stabilize will progressively lengthen.

The heart rate response to exercise is influenced by several factors. There is a decline in mean maximum heart rate with age¹¹ that seems to be related to neural influences. Dynamic exercise increases heart rate more than isometric or resistance exercise. An accelerated heart rate response to standardized workloads is observed after prolonged bed rest, indicating a deconditioning response. Other factors that influence heart rate include body position, type of dynamic exercise, certain

physical conditions, state of health, blood volume, sinus node function, medications, and environment.

Arterial Blood Pressure Response

Systolic blood pressure rises with increasing dynamic work as a result of increasing cardiac output, whereas diastolic pressure usually remains about the same or moderately lower, and it may be heard to zero in some normal subjects. Normal values of maximum systolic blood pressure for men have been defined and are directly related to age.

After maximum exercise, there is usually a decline in systolic blood pressure, which normally reaches resting levels within 6 minutes and often remains lower than pre-exercise levels for several hours. When exercise is terminated abruptly, some healthy persons have precipitous drops in systolic blood pressure due to venous pooling and a delayed immediate postexercise increase in systemic vascular resistance to match the reduction in cardiac output. Figure 2 shows the physiological response to submaximal and maximum treadmill exercise on the basis of tests of >700 apparently healthy men aged 25 to 54 years. Maximum rate-pressure product (heart rate×systolic blood pressure) ranges from a tenth percentile value of 25 000 to a 90th percentile value of 40 000.

Testing Procedures

Subject Preparation

Preparations for exercise testing include the following.

- The subject should be instructed not to eat or smoke for 3 hours before the test. Water may be taken as needed at any time. Subjects should dress appropriately for exercise, especially with regard to footwear. No unusual physical efforts should be performed for at least 12 hours before testing.
- When exercise testing is performed for diagnostic purposes, withdrawal of medications may be considered because some drugs (especially β -blockers) attenuate the exercise responses and limit the test interpretation. There are no formal guidelines for tapering medications, but rebound phenomena may occur with abrupt discontinuation of β -blockers in patients with a recent acute coronary syndrome. However, most subjects are tested while taking their usual medications. Specific questioning is important to determine which drugs have been taken so that the physician can be aware of possible electrolyte abnormalities and hemodynamic effects of cardioactive drugs.
- A brief history and physical examination should be performed to rule out contraindications (Table 2) to testing or to detect important clinical signs such as a cardiac murmur, gallop sounds, pulmonary "wheezing," or rales. Subjects with a history of worsening unstable angina or decompensated heart failure should not undergo exercise testing until their condition stabilizes. A cardiac physical examination should indicate which subjects have valvular or congenital heart disease. Because hemodynamic responses to exercise may be abnormal in such subjects, such subjects always warrant careful monitoring and, at times, may require early termination of testing. Special considerations should be

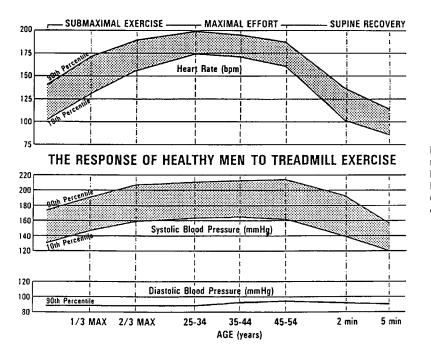


Figure 2. Normal response to progressive treadmill exercise in healthy subjects. bpm indicates beats per minute. Reprinted with permission from Froelicher VF. *Exercise and the Heart: Clinical Concepts*. Chicago, III: Yearbook Medical Publishers, Inc; 1987:102.

made for those with elevated blood pressure and aortic stenosis.

• If the indication for the testing is not clear, the subject should be questioned and the referring physician contacted.

TABLE 2. Absolute and Relative Contraindications to Exercise Testing

Absolute

- · Acute MI (within 2 days)
- · High-risk unstable angina
- Uncontrolled cardiac arrhythmias causing symptoms of hemodynamic compromise
- Active endocarditis
- Symptomatic severe aortic stenosis
- Decompensated symptomatic heart failure
- · Acute pulmonary embolus or pulmonary infarction
- Acute noncardiac disorder that may affect exercise performance or be aggravated by exercise (eg, infection, renal failure, thyrotoxicosis)
- Acute myocarditis or pericarditis
- Physical disability that would preclude safe and adequate test performance
- Inability to obtain consent

Relative*

- · Left main coronary stenosis or its equivalent
- Moderate stenotic valvular heart disease
- · Electrolyte abnormalities
- Tachyarrhythmias or bradyarrhythmias
- Atrial fibrillation with uncontrolled ventricular rate
- Hypertrophic cardiomyopathy
- · Mental impairment leading to inability to cooperate
- · High-degree AV block

*Relative contraindications can be superseded if benefits outweigh risks of exercise.

- A resting standard 12-lead electrocardiogram (ECG) should be obtained because it may differ from the resting pre-exercise ECG. The "torso" ECG distorts the standard ECG by shifting the axis to the right, increasing voltage in the inferior lead group. This may cause a disappearance of Q waves in a patient with a documented previous Q-wave inferior myocardial infarction (MI).
- Standing ECG and blood pressure should be recorded (in the sitting position with cycle ergometry) to determine vasoregulatory abnormalities and positional changes, especially ST-segment depression.
- A detailed explanation of the testing procedure should be given that outlines risks and possible complications. The subject should be instructed on how to perform the test, and these instructions should include a demonstration. If musculoskeletal or certain orthopedic limitations are a concern, the testing protocol should be modified.

Electrocardiographic Recording

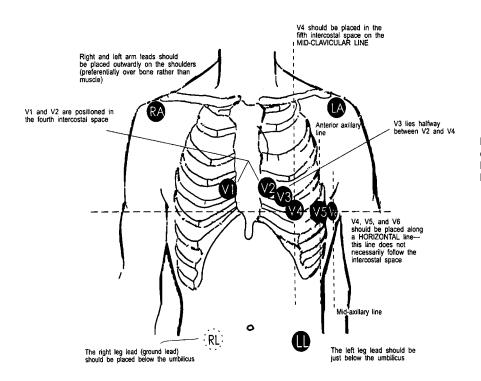
Skin Preparation

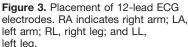
The most critical point of the electrode-amplifier recording system is the interface between electrode and skin. Removal of the superficial layer of skin significantly lowers its resistance, thus decreasing the signal-to-noise ratio. The areas for electrode application are first shaved and then rubbed with alcohol-saturated gauze. After the skin dries, it is marked with a felt-tipped pen and rubbed with a fine sandpaper or rough material. With these procedures, skin resistance should be reduced to 5000 Ω or less.

Electrodes and Cables

Many electrodes are available for performing exercise testing. Silver plate or silver chloride crystal pellets are preferred because they have the lowest offset voltage. Care should be taken to assure that the electrode gel is moist.

12-lead ECG Electrode Placement





Connecting cables between the electrodes and recorder should be light, flexible, and properly shielded. Most available commercial exercise cables are constructed to lessen motion artifact. Cables generally have a life span of ≈ 1 year. They eventually become a source of both electrical interference and discontinuity and must be replaced.

Multiple Leads

Because a high-quality standard 12-lead ECG with electrodes placed on the limbs cannot be obtained during exercise, other electrode placements have been used. Electrode placement affects ST segment slope and amplitude. Various placements do not result in comparable waveforms for analysis. For comparison with the standard resting 12-lead recording, arm and leg electrodes should be moved to the wrists and ankles, with the subject in the supine position. Differences can be minimized by placing the arm electrodes as close to the shoulders as possible, placing the leg electrodes below the umbilicus, and recording the resting ECG with the subject supine (Figure 3). Any modification of lead placement should be recorded on the tracing.

Relative Sensitivity of Leads

The lateral precordial leads (V_4 through V_6) are capable of detecting 90% of all ST depression observed in multiple lead systems. ST elevation (over non–Q-wave areas) is a rare but critical change due to transmural ischemia that occurs as frequently in lead V_2 and aVF as in V_5 .

Recorders

There are many good recorders designed to capture highquality ECG data during exercise. Many use microprocessors to generate average waveforms and make ECG measurements. The physician must compare the raw analog data with computer-generated output to validate its accuracy. Computer processing is not completely reliable because of software limitations in handling noise and inadequacy of the available algorithms.

Equipment and Protocols

For details regarding exercise testing equipment and exercise testing laboratories, the reader should refer to the AHA's "Guidelines for Clinical Exercise Testing Laboratories."¹² Figure 4 illustrates the relation of METs to stages in the various testing protocols. The treadmill and cycle ergometer are now the most commonly used dynamic exercise testing devices.

Cycle

Electrically braked cycles vary the resistance to the pedaling speed (rate-independent ergometers), thereby permitting better power output control, because it is common for subjects who are fatigued or unable to cooperate to decrease their pedaling speed. The highest values of $\dot{V}o_2$ and heart rate are obtained with pedaling speeds of 50 to 80 rpm. Cycles are calibrated in kiloponds (kp) or watts (W); 1 W is equivalent to ≈ 6 kp-meters per minute (kpm/min). Because exercise on a cycle ergometer is non-weight-bearing, kiloponds or watts can be converted to oxygen uptake in milliliters per minute. METs are obtained by dividing $\dot{V}o_2$ in milliliters per minute by the product of body weight (in kg)×3.5. The number 3.5 is the accepted value assigned to oxygen uptake while at rest and is expressed as milliliters of O_2 per kilogram of body

FUNCTIONAL CLASS			IICA TUS		O ₂ COST ml/kg/min	METS	BICYCLE ERGOMETER		TREADMILL PROTOCOLS		LS	METS			
	×						1 WATT = 6.1 Kpm/min	MOD 3 r Stages	BRUCE MODIFIED 3 min Stages MPH %GR		MODIFIED BRUCE 3 min 3 min Stages MPH Stages		NAUG	HTON	
	ACTIVITY						FOR 70 KG BODY	6.0	22	6.0	22	-			
							WEIGHT Kpm/min	5.5	20	5.5	20				
NORMAL	В							5.0	18	5.0	18	_			
	ON AGE,				56.0	16								16	
AND	0 E				52.5	15								15	
I	Ш				49.0	14	1500		T			-		14	
I	N N N				45.5	13		4.2	16	4.2	16	_		13	
	Ë				42.0	12	1350							12	
	÷				38.5	11	1200	3.4	14	3.4	14	-	min Iges	11	
	HEAL THY, DEPENDENT	7			35.0	10	1050	3.4	14	3.4	14	1	%GR	10	
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II		SEDENTARY HEALTHY	LIMITED	SYMPTOMATIC	17.5	5	450	1.7	10	1.7	10	2	7.0	5	
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111			1	PT	10.5	З		- 1.7	5			2	0	3	
				SYA	7.0	2	150	1.7	0			1	0	2	
IV	1			1	3.5	1						<u> </u>	Ľ	1	

Figure 4. Relation of METs to stages in the various testing protocols. Functional class refers to New York Heart Association class; kpm indicates kilopond-meters; MPH, miles per hour; and %GR, percent grade.

weight per minute. Figure 4 demonstrates the MET levels at given work rates (kpm/min) of a cycle protocol for a 70-kg person.

The cycle ergometer is usually less expensive, occupies less space, and is less noisy than a treadmill. Upper body motion is usually reduced, making it easier to obtain blood pressure measurements and to record the ECG. Care must be taken to prevent isometric or resistance exercise of the arms while grasping the handlebars.

A major limitation to cycle ergometer testing is the discomfort and fatigue of the quadriceps muscles. Leg fatigue in an inexperienced subject may cause him or her to stop before reaching a true $\dot{V}O_{2 \text{ max}}$. Thus, $\dot{V}O_{2 \text{ max}}$ is 10% to 15% lower in cycle versus treadmill testing in those not accustomed to cycling.

Treadmill

The treadmill should have front and/or side rails to aid in subject stability. However, subjects should be encouraged not to tightly grasp the front or side rails because this action supports body weight and thus reduces the workload at any given stage. It may be helpful if subjects remove their hands from the rails, close their fists, and place one finger on the rails to maintain balance after they are accustomed to walking on the treadmill. The treadmill should have both variable speed and grade capability and must be accurately calibrated.

Protocols

Protocols for clinical exercise testing include an initial warm-up (low load), progressive uninterrupted exercise with

increasing loads and an adequate time interval in each level, and a recovery period. For cycle ergometry, the initial power output is usually 10 or 25 W (150 kpm/min), usually followed by increases of 25 W every 2 or 3 minutes until end points are reached. If arm ergometry is substituted for cycle ergometry, a similar protocol may be used, except that initial power output and incremental increases are lower. Two-minute stages are most popular with arm ergometry.^{13,14}

Several different treadmill protocols are in use and are defined in Figure 4 according to treadmill speed, grade, stage duration, and estimated METs. The advantages of the Bruce protocol are its use in many published studies and the value of 3-minute stages to acquire submaximal data. Its disadvantages are large interstage increments in work that can make estimation of $\dot{V}_{O_{2 max}}$ less accurate and a fourth stage that can be either run or walked, resulting in different oxygen costs. Some subjects are forced to stop exercising prematurely because of musculoskeletal discomfort or an inability to tolerate the high workload increments. Initial zero or one-half stages (1.7 miles/hour at 0% and 5% grades) can be used for subjects with compromised exercise capacities. The optimum protocol for any test should last 6 to 12 minutes and should be adjusted to the subject's needs.

Ramp protocols start the subject at a relatively low treadmill speed, which is gradually increased until the patient has a good stride. The ramp angle of incline is progressively increased at fixed intervals (ie, 10 to 60 seconds) starting at 0 grade, with the increase in grade calculated on the patient's estimated functional capacity such that the protocol will be completed in 6 to 12 minutes. In this type of protocol, the rate of work increases continuously, and steady states are not reached. A limitation of ramp protocols is the requirement to estimate functional capacity from an activity scale and adjust the ramp accordingly. Occasionally underestimation or overestimation of functional capacity will result in an endurance test or in premature exercise termination. Exercise protocols should be individualized according to the type of subject being tested. A 9-minute targeted ramp protocol that increases in small steps has many advantages, including more accurate estimates of MET level.¹⁵

The 6-minute walk test is a functional test that can be used to evaluate exercise capacity in patients with marked LV dysfunction or peripheral arterial occlusive disease who cannot perform cycle ergometer or treadmill exercise. Patients are instructed to walk down a 100-foot corridor at their own pace, attempting to cover as much ground as possible in 6 minutes. At the end of the 6-minute interval, the total distance walked is determined and the symptoms experienced by the patient are recorded. This type of protocol uses a submaximal level of stress and thus correlates only modestly with $\dot{V}o_{2 max}$.¹⁶ ECG monitoring is not routinely done with this testing, thus limiting its diagnostic accuracy.

Exercise Test Supervision and Interpretation

Exercise testing should be conducted only by well-trained personnel with a sufficient knowledge of exercise physiology. Only technicians, physiologists, nurses, and physicians familiar with normal and abnormal responses during exercise can recognize or prevent adverse events. Equipment, medications, and personnel trained to provide advanced cardiopulmonary resuscitation (CPR) must be readily available. For details regarding supervision and interpretation of exercise tests, the reader should refer to the ACC/AHA/American College of Physicians' "Clinical Competence Statement on Stress Testing."¹⁷

Although exercise testing is considered a safe procedure, there are reports of acute MIs and deaths. Multiple surveys confirm that as many as 10 MIs or deaths or both may be expected per 10 000 tests in those with CAD.¹⁸ Risk is greater in the post-MI subject and in those being evaluated for malignant ventricular arrhythmias. A review summarizing 8 studies of estimates of sudden cardiac death during exercise testing revealed rates from 0.0 (4 studies) to 5 per 100 000 tests.¹⁸ Table 3 lists 3 classes of complications secondary to exercise tests.

Good clinical judgment should be foremost in deciding indications and contraindications for exercise testing.³ Although absolute contraindications are definitive, in selected cases with relative contraindications, even submaximal testing can provide valuable information. Table 2 lists absolute and relative contraindications to exercise testing. In any procedure with a risk of complications, the physician should be certain that the subject understands the risks and benefits of the test. Good physician-patient communication about testing is mandatory, and written informed consent should be obtained.

TABLE 3. Complications Secondary to Exercise Tests

Cardiac

- Bradyarrhythmias
- Tachyarrhythmias
- Acute coronary syndromes
- Heart failure
- Hypotension, syncope, and shock
- Death

Noncardiac

- Musculoskeletal trauma
- Soft-tissue injury

Miscellaneous

 Severe fatigue (malaise), sometimes persisting for days; dizziness; fainting; body aches; delayed feelings of illness

Exercise testing should be performed under the supervision of a physician who is appropriately trained to administer exercise tests. The physician should be responsible for ensuring that the exercise laboratory is properly equipped and that exercise testing personnel are appropriately trained. The degree of subject supervision needed during a test can be determined by the clinical status of the subject being tested. This determination is made by the physician or physician's designated staff member, who asks pertinent questions about the subject's medical history, performs a brief physical examination, and reviews the standard 12-lead ECG performed immediately before testing. The physician should interpret data derived from testing and suggest further evaluation or therapy. The physician or senior medical professional conducting the test must be trained in advanced CPR. A defibrillator and appropriate medications should also be immediately available.

The degree of supervision can be assigned to a properly trained nonphysician (ie, a nurse, physician assistant, or exercise physiologist or specialist) for testing apparently healthy younger persons (<40 years of age) and those with stable chest pain syndromes. A physician should be immediately available during all exercise tests.

Perceived Exertion

The subjective rating of the intensity of exertion perceived by the person exercising is generally a sound indicator of relative fatigue. Rather than using heart rate alone to clinically determine intensity of exercise, the 6 to 20 Borg scale of perceived exertion¹⁹ is useful (Table 4). Special verbal and written explanations about the rating of perceived exertion are available for subjects. Although there is some variation among subjects in their actual rating of fatigue, they seem to rate consistently from test to test. Thus, the Borg scale can assist the clinician in judging the degree of fatigue reached from one test to another and in correlating the level of fatigue during testing with that experienced during daily activities. In general, a Borg scale >18 indicates the patient has performed maximal exercise, and values >15 to 16 suggest that the anaerobic threshold has been exceeded.

0	0
20-Grade Scale	
6	
7	Very, very light
8	
9	Very light
10	
11	Fairly light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Very, very hard
20	

 TABLE 4.
 Borg Scale for Rating Perceived Exertion

The rating of perceived exertion scale. Reprinted with permission from $\operatorname{Borg.}^{\operatorname{19}}$

Anginal Scale

Levels of anginal discomfort in those with known or suspected CAD are also excellent subjective end points. Table 5 details the 1 to 4 scale that is recommended.

Indications for Terminating Exercise Testing

Absolute Indications

- ST-segment elevation (>1.0 mm) in leads without Q waves (other than V₁ or aVR).
- Drop in systolic blood pressure >10 mm Hg (persistently below baseline), despite an increase in workload, when accompanied by any other evidence of ischemia.
- Moderate-to-severe angina (grade 3 to 4); Table 5 details descriptions and grades for angina scale.
- Central nervous system symptoms (eg, ataxia, dizziness, or near syncope).
- Signs of poor perfusion (cyanosis or pallor).

TABLE 5. Four-Level Angina Scale for Exercise Tolerance Testing*

Description	Level
Onset of angina, mild but recognized as the usual angina-of-effort pain or discomfort with which the subject is familiar	1
Same pain, moderately severe and definitely uncomfortable but still tolerable	2
Severe anginal pain at a level that the subject will wish to stop exercising	3
Unbearable chest pain; the most severe pain the subject has felt	4

*Angina criteria for stopping a symptom-limited exercise test is level 2 angina, approaching level 3. Data in Table are from Allred EN, Bleecker ER, Chaitman BR, et al. Effects of carbon monoxide on myocardial ischemia. *Environ Health Perspect.* 1991;91:89–132 and Allred EN, Bleecker ER, Chaitman BR, et al. Short-term effects of carbon monoxide exposure on the exercise performance of subjects with coronary artery disease. *N Engl J Med.* 1989;321:1426–1432.

- Sustained ventricular tachycardia.
- Technical difficulties monitoring the ECG or systolic blood pressure.
- Subject's request to stop.

Relative Indications

- ST or QRS changes such as excessive ST displacement (horizontal or downsloping of >2 mm) or marked axis shift.
- Drop in systolic blood pressure >10 mm Hg (persistently below baseline). despite an increase in workload, in the absence of other evidence of ischemia.
- Increasing chest pain.
- Fatigue, shortness of breath, wheezing, leg cramps, or claudication.
- Arrhythmias other than sustained ventricular tachycardia, including multifocal ectopic, ventricular triplets, supraventricular tachycardia, heart block, or bradyarrhythmias.
- General appearance (see below).
- Hypertensive response (systolic blood pressure >250 mm Hg and/or diastolic blood pressure >115 mm Hg).
- Development of bundle-branch block that cannot be distinguished from ventricular tachycardia.

Postexercise Period

Some abnormal responses occur only in recovery. If maximum sensitivity is to be achieved with an exercise test, subjects should be supine in the postexercise period; however, for subject comfort, many health professionals prefer the sitting position. A cool-down walk after the test can delay or eliminate the appearance of ST-segment depression; however, the cool down may be indicated in some subjects, whereas abrupt cessation of exercise is the norm for exercise ECG studies. Monitoring should continue for 6 to 8 minutes after exercise or until blood pressure, heart rate, and ST segments are approximate to baseline values. Approximately 85% of subjects with abnormal responses manifest the abnormality during exercise or within 5 to 6 minutes of recovery. An abnormal ECG response occurring only in the recovery period is not unusual. Mechanical dysfunction and electrophysiological abnormalities in the ischemic ventricle after exercise can persist for minutes to hours. Monitoring of blood pressure should continue during recovery because abnormal responses may occur, particularly hypotension.

Interpretation

Clinical Responses

Symptoms

Typical anginal symptoms induced by the exercise test are predictive of CAD and are even more predictive with associated ST-segment depression. It is important to obtain a careful description of the discomfort from the subject to ascertain that it is typical angina rather than nonischemic chest pain.

Subject's Appearance

The subject's general appearance is helpful in the clinical assessment. A decrease in skin temperature, cool and light perspiration, and peripheral cyanosis during exercise can indicate poor tissue perfusion due to inadequate cardiac output with secondary vasoconstriction. Such subjects should not be encouraged to attempt greater workloads.

Physical Examination

Cardiac auscultation immediately after exercise can provide information about ischemia-induced LV dysfunction. Gallop sounds or a precordial bulge can result from LV dysfunction. A new mitral regurgitant murmur suggests papillary muscle dysfunction, which may be related to transitory myocardial ischemia. It is preferable to have subjects lie supine after exercise testing and allow those who develop orthopnea to sit up. In addition, severe angina or ominous arrhythmias after exercise may be lessened by allowing the subject to sit up, because ischemia may be decreased due to lower LV wall tension.

Exercise Capacity

Maximal work capacity in normal individuals is influenced by familiarization with the exercise test equipment, level of training, and environmental conditions at the time of testing. In estimating exercise capacity, the amount of work performed in METs (or exercise stage achieved) should be the index measured and not the number of minutes of exercise. Serial comparison of exercise capacity in individual patients to assess significant interval change requires a careful examination of the exercise protocol used during both tests, cardioactive drug therapy and time of ingestion, systemic blood pressure, and other conditions that might influence test performance. Each of these factors must be considered before attributing changes in functional capacity to progression of coronary heart disease or worsening of LV function.

A normal exercise capacity does not exclude severe LV systolic dysfunction. Mechanisms proposed to explain a normal work performance in these subjects include increased peripheral oxygen extraction, preservation of stroke volume and chronotropic reserve, ability to tolerate elevated pulmonary wedge pressures without dyspnea, ventricular dilation, and increased levels of plasma norepinephrine at rest and during exercise. Many subjects with decreased ejection fractions at rest can perform relatively normal levels of exercise, some without side effects, whereas others report increased fatigue for some time after the test.

Hemodynamic Responses

Blood Pressure During Exercise

Blood pressure is dependent on cardiac output and peripheral resistance. An inadequate rise or a fall in systolic blood pressure during exercise can occur. An inadequate rise in systolic blood pressure (<20 to 30 mm Hg) or a drop can result from aortic outflow obstruction, severe LV dysfunction, myocardial ischemia, and certain types of drug therapy (ie, β -blockers). In some subjects with CAD, higher levels of systolic blood pressure exceeding peak exercise values have been observed during the recovery phase.²¹ In most studies, exercise-induced hypotension in association with other measures of ischemia predicts a poor prognosis, with a positive predictive value of 50% for left main or triple-vessel disease.²² Exercise-induced hypotension is also associated with

cardiac complications during exercise testing (for example, serious arrhythmias), seems to be alleviated by coronary artery bypass grafting (CABG), and can occur in subjects with CAD, valvular heart disease, or cardiomyopathy. Occasionally, subjects without clinically significant heart disease will exhibit exercise-induced hypotension during exercise related to dehydration, antihypertensive therapy, or prolonged strenuous exercise.

Heart Rate During Exercise

Relatively rapid heart rate during submaximal exercise or recovery could be due to deconditioning, prolonged bed rest, anemia, metabolic disorders, or any other condition that decreases vascular volume or peripheral resistance. This finding is relatively frequent soon after MI and CABG. Relatively low heart rate at any point during submaximal exercise could be due to exercise training, enhanced stroke volume, or drugs. The common use of β -blockers, which lower heart rate, limits the interpretation of the heart rate response to exercise. Conditions that affect the sinus node can attenuate the normal response of heart rate during exercise testing. Chronotropic incompetence, which is defined as either failure to achieve 85% of the age-predicted maximal heart rate or a low chronotropic index (heart rate adjusted to MET level), is associated with an increased mortality risk in patients with known cardiovascular disease.23

Responses in Subjects With Normal Resting ECGs *P Wave*

During exercise, P wave magnitude increases significantly in inferior leads. There should be no significant changes in P wave duration.

PR Segment

The PR segment shortens and slopes downward in the inferior leads during exercise. The decreasing slope has been attributed to atrial repolarization (the Ta wave) and can cause false-positive ST depression in the inferior leads.

QRS Complex

The Q wave shows very small changes from the resting values; however, it does become slightly more negative at maximum exercise. Changes in median R wave amplitude are noted near maximum effort. A sharp decrease in the R wave is observed in the lateral leads (V_5) at maximum exercise and into the first minute of recovery. In the lateral and vertical leads (V_5 and aVF), the S wave becomes greater in depth (more negative), showing a greater deflection at maximum exercise, and then gradually returns to resting values in recovery. As the R wave decreases in amplitude, the S wave increases in depth.

J-Junction (J-Point) Depression

The J junction (QRS end/ST beginning) is depressed in lateral leads to a maximum depression at maximum exercise, then gradually returns toward pre-exercise values in recovery. J-junction depression is more common in older patients. Subjects with resting J-junction elevation (early repolarization) may develop an isoelectric J junction with exercise; this is a normal finding. The normal ST segment vector response

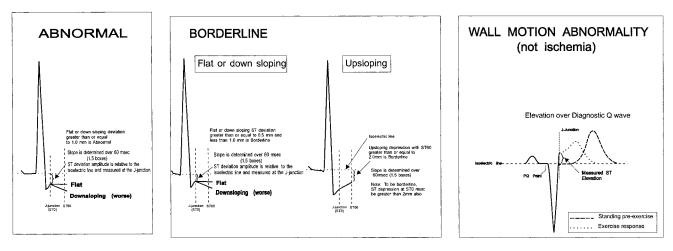


Figure 5. ST deviation assessment.

both to tachycardia and exercise is a shift rightward and upward.

T Wave

A gradual decrease in T wave amplitude is observed in all leads during early exercise. At maximum exercise, the T wave begins to increase, and at 1 minute into recovery, the amplitude is equivalent to resting values in the lateral leads.

U Wave

No significant changes are noted with exercise; however, U waves may be difficult to identify at ventricular rates >130 beats/min because of the close approximation of the T and P waves with the increased heart rate of exercise.

Abnormal Responses

ST Segment Changes

The ST level is measured relative to the P-Q junction because the U-P segment during exercise is difficult to measure. Ideally, 3 consecutive beats in the same lead with a stable baseline should be identified and the average displacement determined. The 3 key measurements are identification of the P-Q junction (isoelectric line), the J point (ie, J junction, QRS end, and ST segment beginning), and 60 or 80 ms after the J point. At ventricular rates >130 beats/min, 60 ms after the J junction is optimal to determine the extent of ST segment displacement in patients with an upsloping ST segment slope. When the J point relative to the P-Q junction is depressed at baseline, the net difference from the J junction determines the magnitude of exercise-induced displacement. When the J junction is elevated at rest (early repolarization) and progressively becomes more depressed during exercise, the magnitude of ST-segment displacement is determined from the P-Q junction and not from the resting elevated J junction. Exercise-induced myocardial ischemia can result in 1 of the following 3 ST segment manifestations on the surface ECG: depression, elevation, or normalization (Figure 5).

ST Segment Depression

ST segment depression is the most common manifestation of exercise-induced myocardial ischemia. The ST segment depression represents electrical gradients caused by myocardial ischemic segments, the extent of the ischemic zone, previous areas of myocardial necrosis, and location of the recording electrodes (Figure 5). The standard criterion for this abnormal response is horizontal or downsloping ST segment depression of ≥ 0.10 mV (1 mm) for 80 ms. However, as shown in Figure 5, other criteria have been considered. Downsloping ST segment depression is a more specific change than horizontal or upsloping depression. In the presence of marked baseline abnormalities, exercise-induced ST segment depression is less specific for myocardial ischemia. Other factors related to the probability and severity of CAD include the degree, time of appearance, duration, and number of leads with ST segment depression.

Severity of CAD is also related to the time of appearance of ischemic ST segment shifts. The lower the workload and rate-pressure product at which it occurs, the worse the prognosis and the more likely the presence of multivessel disease. The duration of ST depression in the recovery phase is also related to the severity of CAD.

ST Segment Elevation

Exercise-induced elevation may occur in an infarct territory where Q waves are present or in a noninfarct territory. The development of >0.10 mV of J-point elevation that is persistently elevated (>0.10 mV) at 60 ms after the J point in 3 consecutive beats with a stable baseline is considered an abnormal response.

ST Segment Elevation in Post-MI Patients With Q Waves

Prior MI is the most frequent cause of ST-segment elevation during exercise and seems to be related to the presence of severe hypokinetic or akinetic LV segmental wall motion. Approximately 30% of subjects with anterior MI and 15% of subjects with inferior MI tested early after MI demonstrate exercise-induced ST segment elevation in Q-wave leads. The changes may result in reciprocal ST-segment depression that simulates myocardial ischemia in other leads. However, ST-segment elevation and depression in the same test may indicate multivessel CAD. Myocardial imaging techniques will help distinguish the concomitant presence of a new myocardial ischemic zone from reciprocal changes induced by ST-segment elevation in Q-wave leads.

ST Segment Elevation in Subjects Without Prior Infarction

In subjects without previous MI (absence of Q waves on the resting ECG), ST segment elevation during exercise frequently localizes the site of severe transient ischemia resulting from significant proximal disease or spasm. In patients with active variant angina (>2 spontaneous episodes per week), exercise-induced ST segment elevation induced by coronary vasospasm has been reported in \approx 30% of subjects. A reversible thallium-201 perfusion defect usually corresponds to the site of exercise-induced ST elevation. Ventricular arrhythmias during the test are more frequent in patients who demonstrate this response.

ST Segment Normalization or Absence of Change

Another manifestation of ischemia may be normalization of or no change in the ST segment related to cancellation effects, but this is nonspecific. ECG abnormalities at rest, including T wave inversion and ST segment depression, reportedly return to normal during attacks of angina and during exercise in some subjects with ischemic heart disease, but these findings can also be observed in subjects with a persistent juvenile pattern on the resting ECG. This cancellation effect is rare but should be considered as a cause of exercise-induced ST segment "normalization."

Diagnostic Value of R Wave Changes

Many within-subject estimates of the variability of R wave amplitude changes during exercise in normal subjects have been reported. However, the average response in normal subjects is an increase in R wave amplitude during submaximal exercise, with a decline at maximum exercise. Exerciseinduced changes in R wave amplitude have not improved diagnostic accuracy, despite use of several lead systems, clinical subsets of subjects, and different criteria for an abnormal response.

T Wave Changes

The morphology of the T wave is influenced by body position, respiration, hyperventilation, drug therapy, and myocardial ischemia/necrosis. In patient populations with a low CAD prevalence, normalization of inverted T waves with exercise is a nondiagnostic finding.

U Wave Changes

U wave inversion is associated with LV hypertrophy, CAD, and aortic and mitral regurgitation. These conditions are associated with abnormal LV distensibility. Exercise-induced U wave inversion in subjects with a normal resting ECG seems to be a marker of myocardial ischemia and suggests left anterior descending CAD.

ST/Heart Rate Index and Slope

Heart rate adjustment of ST segment depression seems to improve the sensitivity of the exercise test, particularly the prediction of multivessel coronary disease, but this has not been consistently observed.^{24,25} Calculation of maximal ST/ heart rate slope in millivolts per beats per minute is performed by linear regression analysis relating the measured amount of ST segment depression in individual leads to the heart rate at the end of each stage of exercise, starting at end exercise. An

ST/heart rate slope >2.4 mV · beats⁻¹ · min⁻¹ is considered abnormal, and values >6 mV · beats⁻¹ · min⁻¹ are suggestive of 3-vessel coronary disease. The use of this measurement requires modification of the exercise protocol such that increments in heart rate are gradual, as opposed to more abrupt increases in heart rate between stages, which limit the ability to calculate statistically valid ST segment/heart rate slopes. The measurement is not accurate in the early phase after infarction. A modification of the ST segment/heart rate slope method is the ST segment/heart rate index calculation, which represents the average changes of ST segment depression with heart rate throughout the course of the exercise test. The ST/heart rate index measurements are less than the ST/heart rate slope measurements, and an ST/heart rate index of 1.6 is defined as abnormal.²⁵

Stress Testing With Imaging Modalities

The addition of various imaging techniques to exercise is particularly useful when the resting ECG has baseline abnormalities (eg, left bundle-branch block or resting ST depression >1 mm) that limit the accurate interpretation of the exercise ECG. Imaging also provides information regarding the location of ischemic myocardium and the size of the "territory" at risk. Exercise or pharmacological stress imaging studies provide greater diagnostic accuracy than exercise ECG alone and are particularly useful when the results of the exercise ECG are equivocal or indeterminate. Details regarding stress testing using imaging modalities are found in the "ACC/AHA Guidelines for the Clinical Application of Echocardiography"²⁶ and the "ACC/AHA Guidelines for Clinical Use of Cardiac Radionuclide Imaging."²⁷

Exercise Echocardiography

Echocardiographic images at rest are compared with those obtained while the patient performs stationary cycling or those obtained immediately after treadmill exercise. Images must be obtained within 1 to 2 minutes (preferably <1minute) after exercise, because abnormal wall motion begins to normalize after this point. Rest and stress images are compared side by side in a cineloop display that is gated (ie, synchronized) to the QRS complex. Myocardial contractility normally increases with exercise, whereas ischemia causes hypokinesis, akinesis, or dyskinesis of the affected segments. A test is considered positive if wall motion abnormalities develop with exercise in previously normal territories or worsen in an already abnormal segment.26 The overall sensitivity of exercise echocardiography for detecting CAD ranges from 71% to 97%, with greater sensitivities in multivessel disease. The specificity ranges from 64% to 100%.²⁶ Patients with a normal exercise echocardiogram have a low risk for future cardiac events, including revascularization procedures, MI, or cardiac death. Complications during exercise echocardiography are no different from those during exercise ECG testing, because the echocardiography procedure itself has no known risks.

Exercise Nuclear Imaging

Exercise tests with nuclear imaging use myocardial perfusion imaging agents, such as thallium-201, technetium (Tc)-99m

sestamibi, or tetrofosmin, which are injected 1 minute before the end of exercise. Images are subsequently obtained at 15 minutes for thallium 201 and at 15 to 60 minutes after exercise for Tc-99m sestamibi. Tc agents can be used with a rest/stress protocol or a stress/rest protocol. Tc-99m sestamibi offers several advantages over thallium. Sestamibi has a half-life of 6 hours, compared with 73 hours for thallium, and it also has a higher photon energy. This shorter half-life enables the injection of a higher isotope dose, resulting in improved image quality with greater resolution and less attenuation. Another unique property of Tc-based agents is the lack of redistribution. As such, images may be obtained for up to 4 hours after injection. Cardiac images are usually displayed in tomographic slices from 3 different axes: the short axis, vertical long axis, and horizontal long axis. This allows visualization of the heart in 3 dimensions so that multiple myocardial segments can be viewed individually, without the overlap of segments that occurs with planar imaging.²⁷ Perfusion defects that are present during exercise but not seen at rest indicate myocardial ischemia. Perfusion defects that are present during exercise and persist at rest suggest previous MI.

Exercise with Tc99m sestamibi imaging has shown an accuracy similar to that of exercise with thallium-201 imaging in the detection of myocardial ischemia. For planar imaging, the sensitivity and specificity of Tc99m sestamibi have been measured at 84% and 83%, compared with 83% and 88% for thallium; for single photon emission computerized tomography (SPECT) imaging, they were 90% and 93%, respectively, compared with 89% and 76% for thallium. The greater specificity of Tc99m perfusion imaging is primarily due to less soft-tissue and diaphragmatic attenuation. The overall segment agreement with Tc99m sestamibi and thallium-201 is 88% with planar and 92% with SPECT imaging.²⁷

Pharmacological Stress Testing

Pharmacological agents can be used to increase cardiac work in lieu of exercise or cause coronary arterial vasodilation to increase myocardial blood flow. Patients unable to undergo exercise stress testing for reasons such as deconditioning, peripheral vascular disease, orthopedic disabilities, neurological disease, and concomitant illness can often benefit from pharmacological stress imaging procedures. Indications for these tests include establishing a diagnosis of CAD, determining myocardial viability before revascularization, assessing prognosis after MI or in chronic angina, and evaluating cardiac risk before noncardiac surgery.

Adrenergic agents such as dobutamine increase myocardial contractility, heart rate, and blood pressure. Dobutamine is infused intravenously starting at 5 μ g · kg⁻¹ · min⁻¹, increasing to 10 μ g · kg⁻¹ · min⁻¹ and, if tolerated, increased every 3 minutes thereafter by 10 μ g · kg⁻¹ · min⁻¹ until a maximal dose of 40 to 50 μ g · kg⁻¹ · min⁻¹ is reached or an end point is achieved. Target heart rate is 85% of the age-predicted maximal value or 70% for submaximal stress. For myocardial perfusion imaging, the radioisotope is injected at peak dobutamine dose. Dobutamine infusion is then continued for 1 minute after injection. End points include new or worsening

wall motion abnormalities, adequate heart rate response, worsening arrhythmia, moderate angina, intolerable side effects, and significant increase or decrease in blood pressure. Up to 1 mg of intravenous atropine may be given if an adequate heart rate is not achieved or other end points have not been reached with dobutamine infusion. ECG, heart rate, and blood pressure are monitored during each stage.^{26,28} Echocardiographic images are obtained throughout with ECG gating and are then displayed in a quad-screen format allowing side-by-side comparison of baseline, low-dose dobutamine, peak dobutamine, and recovery images. A new or worsening wall motion abnormality constitutes a positive test.²⁶ Dobutamine stress echocardiography has a reported sensitivity of 67% to 97%²⁶ (average, 80%)²⁹ and specificity of 65% to 100%²⁶ (average, 84%)²⁹ for the detection of CAD. Complications during dobutamine infusion include nausea, headache, tremor, anxiety, angina and atypical chest pain, atrial and ventricular arrhythmias, and hypertension or hypotension.28

Vasodilators such as adenosine or dipyridamole can also be used to assess coronary perfusion during nuclear imaging or, less often, during echocardiography. These agents cause maximal coronary vasodilation in normal epicardial arteries. Due to autoregulation, arteries with stenoses recruit vasodilator reserve to maintain flow at rest and may even be maximally dilated at rest; therefore, they do not increase flow normally when vasodilators are given. The radioisotope is injected at peak vasodilator infusion and images are obtained at 15 minutes for thallium-201 and 1 hour later for Tc99m agents. Side effects include flushing, chest pain, headache, nausea, dyspnea, and atrioventricular (AV) block, which can be reversed with aminophylline.27 Due to the short half-life of adenosine, side effects usually resolve with termination of the infusion. Vasodilator agents should not be used in patients with second- or third-degree AV block (without permanent pacemakers) and in patients with a bronchospastic disease such as asthma or severe chronic obstructive lung disease.27 Notably, nuclear perfusion imaging with vasodilator agents is preferred over exercise perfusion imaging for the diagnosis of CAD in patients with left bundle branch block on resting ECG,27,30 because septal perfusion defects can occur in patients with normal coronary arteries and left bundle branch block.

Diagnostic Value of the Exercise Test

Sensitivity and Specificity

Sensitivity and specificity define how effectively a test separates subjects with disease from healthy individuals (ie, how well a test diagnoses disease). Sensitivity is the percentage of those individuals with a disease who will have abnormal tests. Sensitivity is influenced by disease severity, effort level, and anti-ischemic drugs. Specificity is the percentage of those without the disease who will have normal test results, and it may be affected by drugs such as digoxin, baseline ECG patterns, and LV hypertrophy. Sensitivity and specificity are inversely related; when sensitivity is the highest, specificity is lowest and vice versa. All tests have a range of inversely related sensitivities and specificities that can be selected by specifying a discriminant or diagnostic cut point.

The choice of a discriminant value is further complicated by the fact that some exercise test responses do not have established values that separate normal subjects from those with disease. Once a discriminant value that determines a test's specificity and sensitivity is chosen, the population tested must be considered. If the population is skewed toward individuals with a greater severity of disease, the test will have a higher sensitivity. For instance, the exercise test has a higher sensitivity in individuals with triple-vessel disease than in those with single-vessel disease. A test can also have a lower specificity if it is used in individuals who are more likely to have false-positive results. Sensitivity and specificity of exercise-induced ST segment depression can be determined by comparing the results of exercise testing and coronary angiography.³¹ From these studies, it can be seen that the exercise test cut point of 0.1 mV (1 mm) of horizontal or downsloping ST segment depression has \approx 84% specificity for angiographically significant CAD; ie, 84% of those without significant angiographic disease had a normal exercise test. These studies had a mean sensitivity of 66% for significant angiographic CAD, with a range of 40% to 90% for 1-vessel disease to 3-vessel disease.

Relative Risk and Predictive Value

Relative risk and predictive value help define the diagnostic value of a test (Glossary). The predictive value of a test is greatly influenced by the prevalence of disease in the group (or individual) being tested. Bayes' theorem states that the probability of a person having the disease after the test is performed is the product of the probability of disease before testing and the probability that the test provided a true result. For example, an exercise ECG that demonstrates ST depression in a young asymptomatic person without cardiac risk factors is most likely a false-positive result. Conversely, exercise-induced ST depression in an elderly person with typical anginal symptoms is most likely a true positive result.

Women

Exercise testing has the same characteristics in women with an intermediate probability of CAD as it does for men. In calculating the probability of CAD as determined by age and symptoms, women usually reach intermediate probability 10 years later than men.³ In a series of 976 symptomatic women referred for exercise testing and coronary angiography, a low, moderate, and high risk Duke treadmill score (a method of estimating cardiovascular prognosis) was associated with CAD (>75% luminal narrowing) in 19.1%, 34.9%, and 89.2% of women, respectively.32 The frequency of 3-vessel disease or left main coronary disease was 3.5%, 12.4%, and 46%, respectively. In a retrospective population-based cohort study of 741 women, exercise-induced angina, ischemic ECG changes, and workload were strongly associated with allcause mortality and cardiac events. The 2-year cardiac mortality rates in 976 women with low, moderate, and high risk Duke treadmill scores were 1%, 2.2%, and 3.6%, respectively.32 Thus, in women with established CAD, exercise testing provides diagnostic and prognostic information, particularly when scores are used.³

Intracardiac Conduction Blocks

Intraventricular Blocks

Intracardiac conduction blocks can exist before exercise or develop or disappear during exercise. Rate-dependent intraventricular blocks that develop during exercise often precede the appearance of chronic blocks that develop later at rest.33-35 Diagnosis of myocardial ischemia from the exercise ECG is usually impossible when left bundle branch block is present. There can be a marked degree of exercise-induced ST segment depression in addition to that found at rest in normal subjects with left bundle branch block. There is no difference in ST segment response to exercise between those with and those without myocardial ischemia. Left bundle branch block that occurs at a heart rate <125 beats/min in subjects with typical angina is frequently associated with CAD, whereas left bundle branch block occurring at a heart rate \geq 125 beats/min occurs more frequently in subjects with normal coronary arteries. The presence of intraventricular blocks at rest that disappear during exercise is rare. Subjects with left bundle branch block who develop a normal QRS pattern during exercise have been reported. Preexisting right bundle branch block35-39 does not influence interpretation of the exercise test, except in the anterior precordial leads $(V_1,$ V₂, and V₃), where ST depression is frequently present at baseline.

Intraventricular Blocks During Exercise

In addition to left or right bundle branch block, left anterior or posterior hemiblock and bifascicular block (a combination of right bundle branch block and left anterior or posterior hemiblock) may be induced with exercise. The presence of such blocks is primarily a rate-related phenomenon that occurs during exercise as the sinus rate increases beyond a critical point. Intraventricular blocks may be difficult to distinguish from ventricular tachycardia.

Conduction Abnormalities

AV Conduction

Shortening of the PR interval (by as much as 0.10 or 0.11 seconds) during exercise as the sinus rate increases is normal, probably because of increased sympathetic tone and vagal withdrawal, such as usually occurs in young, healthy individuals.

First-Degree AV Block

First-degree AV block occurs occasionally at the end of exercise or during the recovery phase. Medications or conditions that may produce prolonged AV conduction time (eg, digitalis, propranolol, verapamil, and myocarditis) predispose the individual to lengthening of the PR interval.

Second-Degree AV Block

The occurrence of Wenckebach-Mobitz type I AV block during exercise is rare. The clinical significance of exerciseinduced Mobitz type II AV block is not known, but the type II block may also be a rate-related phenomenon that appears as the sinus rate is accelerated beyond a critical level. However, it may reflect more critical underlying conduction system disease, and if second-degree AV block develops with testing, the test should be terminated.

Complete AV Block

Acquired complete AV block at rest is a relative contraindication to exercise testing. Exercise testing can be conducted in subjects with congenital complete AV block if there are no coexisting significant congenital anomalies.

Sinus Arrest

Rarely, subjects develop long periods of sinus arrest immediately after exercise. Sinus arrest usually occurs in subjects with severe ischemic heart disease.

Preexcitation Syndromes

Exercise may provoke, abolish, or have no effect on anomalous AV conduction in individuals with known Wolff-Parkinson-White (WPW) syndrome.40 When exercise does not interfere with preexisting anomalous AV conduction, significant ST depression can be observed during exercise testing. In the presence of WPW syndrome, the ST depression may not be due to ischemia but may instead be a falsepositive (indeterminate) occurrence. Although exercise has been considered a predisposing factor to initiate tachyarrhythmia in WPW syndrome, there is a low prevalence of tachyarrhythmias during or after exercise in WPW subjects.

Cardiac Arrhythmias

Exercise may induce cardiac arrhythmias under several conditions, including diuretic and digitalis therapy.41-43 Recent ingestion of alcohol or caffeine may exacerbate arrhythmias. Because exercise increases myocardial oxygen demand, in the presence of CAD, exercise-induced myocardial ischemia could predispose the subject to ectopic activity. It seems that ischemia with ST depression is not as arrhythmogenic as ischemia with ST elevation. Exercise-induced arrhythmias are generated by enhanced sympathetic tone, increased myocardial oxygen demand, or both. The period immediately after exercise is particularly dangerous because of the high catecholamine levels that are associated with generalized vasodilation. Peripheral arterial dilation induced by exercise and reduced cardiac output, resulting from diminished venous return secondary to sudden termination of muscular activity, may lead to a reduction in coronary perfusion in early recovery while the heart rate is still elevated. The increased sympathetic tone in the myocardium may stimulate ectopic Purkinje pacemaker activity by accelerating phase 4 of the action potential, which provokes spontaneous discharge and leads to increased automaticity.

Exercise can suppress cardiac arrhythmias present at rest. This phenomenon has been attributed to the overdrive suppression of the ectopic impulse formation by sinus tachycardia that is caused by exercise-induced vagal withdrawal and increased sympathetic stimulation. Exerciseinduced sinus tachycardia may inhibit automaticity of an ectopic focus because it "overrides" automaticity of the Purkinje tissue.

Ectopic ventricular beats are the most frequent cardiac arrhythmia during exercise, followed by supraventricular arrhythmias and fusion beats. Their prevalence is directly related to age and cardiac abnormalities. In general, ectopic ventricular beats are of concern in subjects with a family history of sudden death or a personal history of cardiomyopathy, valvular heart disease, or severe myocardial ischemia.

Sinus arrhythmias with periods of sinus bradycardia and wandering atrial pacemaker are relatively common during early exercise and the immediate recovery phase. Atrial ectopic contractions and atrial "group" beats can occur in either normal or diseased hearts. Exercise-induced transient atrial fibrillation and flutter occur in <1% of individuals who undergo exercise testing.44 These arrhythmias may be induced by exercise in healthy individuals or subjects with rheumatic heart disease, hyperthyroidism, WPW syndrome, or cardiomyopathy. Paroxysmal AV junctional tachycardia is observed during exercise only rarely. Exercise-induced supraventricular arrhythmias alone are not usually related to CAD but are more often related to older age, pulmonary disease, recent alcohol ingestion, or excessive caffeine intake.

Special Cases of Exercise Testing Interpretation

Heart Failure

Recent studies have proven that exercise testing is not only safe in the population of patients with heart failure, but also adds significant clinical information to the care of these patients.⁴⁵ Although exercise capacity correlates poorly with indices of resting ventricular function, the inability to perform aerobic activity is a powerful prognostic indicator. In fact, impaired exercise capacity, as measured using gas analysis (expired air), has revealed that a peak $\dot{V}o_2 \leq 14 \text{ mL} \cdot \text{kg}^{-1}$. min⁻¹ is associated with high mortality. In contrast, patients with peak $\dot{V}o_2$ values >14 mL \cdot kg⁻¹ \cdot min⁻¹ or >50% of the predicted value have a 1-year survival which is similar to that of the post-transplant population.46

Thus, gas exchange analysis is recommended when exercise testing is being used to measure exercise capacity in patients with heart failure. The protocol chosen for testing is less important when gas exchange measurements are coupled with exercise electrocardiography because these measurements are protocol-independent. Protocols for testing can be chosen depending on the physical status and capacity of the patient being tested. In general, protocols should be chosen that last ≈ 8 to 12 minutes to the peak of exercise. Shorter but more aggressive protocols may not allow sufficient time during exercise to adequately measure the full physiological response to exercise. In patients with chronic heart failure, the exercise testing procedures are similar to those of the other populations mentioned above.

Hypertension

There is evidence that an exaggerated blood pressure response with exercise testing is predictive of future hypertension,⁴⁷⁻⁴⁹ may be predictive of future mortality from MI,⁵⁰ and is associated with angiographic CAD.²¹

Cardiomyopathies

Exercise testing has been used in subjects with dilated cardiomyopathy to determine exercise capacity, assess pulmonary response to LV dysfunction, determine the grade of ventricular ectopy, and evaluate the effectiveness of treatment.⁵¹ Subjects with LV dysfunction may have reduced exercise capacity. There is often an inadequate increase in cardiac output during exercise, which limits $\dot{V}O_{2~max}$ and exercise tolerance. Stroke volume at times may increase normally during upright exercise, despite a decrease in LV ejection fraction. Ventricular dilation facilitates use of the Frank-Starling mechanism. However, with increasing exercise, stroke volume and cardiac output often cannot continue to meet the increased demands.

Several compensatory mechanisms have been proposed to explain the poor correlation between LV function and exercise capacity, including normal heart rate reserve and increased peripheral oxygen extraction.

Cardiac Transplantation Recipients

These subjects often undergo exercise testing to establish the exercise prescription for a supervised exercise program. Heart rate and blood pressure responses in these individuals are often blunted during the initial phase of exercise, and thus other end points such as perceived exertion or workload are best used as end points for exercise training. Exercise test protocols should be selected to provide slow increases in intensity of workload to allow time for the denervated heart to respond to circulating catecholamines, which are the mechanism for the increased heart rate response. Due to vagal denervation, these subjects have baseline tachycardia and a prolonged heart rate recovery period.

Hypertrophic Cardiomyopathy

In addition to dynamic outflow tract obstruction, exercise can precipitate sudden death due to arrhythmias as a result of this condition.^{52,53} Chest pain, an abnormal resting ECG, and exercise-induced ST segment depression are frequent. Exercise testing under careful supervision may be especially helpful to demonstrate the level at which significant events occur, such as the presence or severity of arrhythmias, myocardial ischemia, murmurs indicating obstruction in LV outflow, and presyncopal manifestations.

Prognostic Use of the Exercise Test

Rationale

When a subject performs symptom-limited exercise testing, diagnostic and prognostic assessments can be made on the basis of hemodynamic and electrocardiographic data. There are 2 principal reasons to estimate prognosis. The first is to provide accurate answers to a subject's questions about the probable outcome of his or her illness. Although discussion of prognosis is inherently delicate and probability statements can be misunderstood, most subjects find this information useful in planning their work, recreational activities, and financial status. The second reason for determining prognosis is to identify subjects in whom cardiovascular interventions might improve outcome.

Exercise test responses secondary to myocardial ischemia include angina, ST segment depression, and ST segment elevation in ECG leads without Q waves. Predicting the extent and severity of ischemia (ie, the amount of myocardium in jeopardy) is difficult without the use of imaging modalities. It is inversely related to the rate-pressure product at the onset of signs or symptoms of ischemia. Responses related to ischemia or LV dysfunction include chronotropic incompetence, decrease in systolic blood pressure, and poor exercise capacity.

Exercise capacity correlates poorly with LV function in subjects without signs or symptoms of heart failure, and exercise testing is not beneficial in identifying subjects with moderate LV dysfunction. LV dysfunction is better recognized by a history of heart failure or physical examination; diagnosis should be confirmed by an echocardiogram or radionuclide ventriculogram.

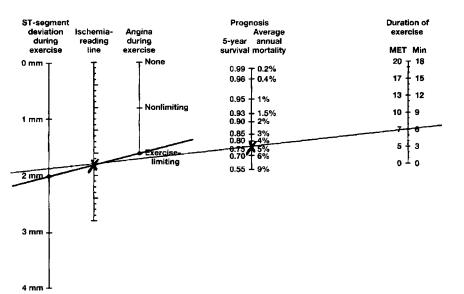
Several subject groups have been studied to determine prognosis with exercise testing, including subjects after MI, those with stable CAD (including silent ischemia and subjects after coronary revascularization), and asymptomatic individuals. Each of these topics is discussed in detail in the "ACC/AHA Guidelines for Exercise Testing."³

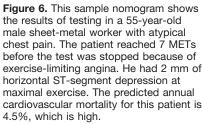
Exercise Testing After MI

Exercise testing is useful in the evaluation and management of patients after MI. As therapies and treatment strategies for MI have changed dramatically, the current role of exercise testing must be viewed in the context of the patients who present for testing. Shorter hospital stays, widespread use of thrombolytic agents, greater use of revascularization strategies, and increased use of β -adrenergic blocking agents and angiotensin-converting enzyme inhibitors continue to change the clinical presentation of the post-MI patient. Details regarding exercise ECG testing and stress testing with imaging modalities in the post-MI patient are presented in the "ACC/AHA Guidelines for the Management of Patients With Acute Myocardial Infarction."54 Exercise testing after MI yields information regarding risk stratification and assessment of prognosis; functional capacity for activity prescription after hospital discharge (this includes domestic and occupational work evaluation and exercise training as part of comprehensive cardiac risk reduction and rehabilitation); and an assessment of the adequacy of medical therapy (for example, in ischemia) and the need to use other diagnostic or treatment options.

Timing and Protocol

Exercise tests can be characterized according to the time after MI when the test is performed and the protocol that is used. The timing of the predischarge exercise test continues to shorten, as does the hospital stay for patients with an uncomplicated MI. Predischarge exercise tests in the literature range from 3 to 26 days after MI. Postdischarge tests have been performed early (14 to 21 days), at 6 weeks, or 6 months after MI.³ Exercise protocols can be either submaximal or symptom-limited. Submaximal protocols have a predetermined end point, which is often defined as a peak heart rate of 120 beats/min or 70% predicted maximum heart rate or a peak MET level of 5. Symptom-limited tests are designed to continue until the patient demonstrates abnormal signs and/or symptoms that necessitate termination of exercise.





Several studies have evaluated symptom-limited protocols at 5 to 7 days after MI and have included patients treated with thrombolytic agents. These studies demonstrate that such testing yields ischemic responses nearly twice as often as submaximal tests and are a better estimate of peak functional capacity.⁵⁵ Thus, early symptom-limited tests have the potential to be more useful in activity prescription before discharge. However, the safety and the additive prognostic value from information obtained from the performance of symptom-limited protocols within days rather than weeks after MI has not yet been established.⁵⁵

Safety

Exercise testing after MI seems to be safe. The incidence of fatal cardiac events including fatal MI and cardiac rupture is 0.03%; nonfatal MI and unsuccessfully resuscitated cardiac arrest, 0.09%, and complex arrhythmias including ventricular tachycardia, 1.4%. Symptom-limited protocols have an event rate that is 1.9 times that of submaximal tests, although the overall fatal event rate is quite low.^{55,56}

Risk Stratification and Prognosis

The prognosis among survivors of MI continues to improve as newer treatment strategies are applied. Data from large thrombolytic trials57,58 and earlier studies in patients not receiving thrombolytic therapy59 consistently demonstrate that those patients unable to perform an exercise test have the highest subsequent cardiac event rate. Uncomplicated stable patients in the era of reperfusion have a low cardiac event rate, even before undergoing further risk assessment by exercise testing. Recent studies are limited in that coronary revascularization interventions are often performed in individuals who demonstrate an ischemic response, thus reducing the predictive value of exercise-induced ischemia for cardiac death or reinfarction. Exercise test predictors of adverse outcome in the post-MI patient include ischemic ST segment depression >1 mm, particularly if at a low level of exercise or in the presence of compensated heart failure; functional capacity <5 METs; inadequate blood pressure response

(peak systolic blood pressure <110 mm Hg or <30 mm Hg rise from resting level).³

The use of β -adrenergic blocking agents after MI has increased over the past decade. Thus, the number of patients taking these agents at the time of their post-MI exercise tests continues to grow. β -Adrenergic blockers reduce the occurrence of angina and ischemic ST changes and lengthen the time to ischemia on exercise testing.60 Patients taking β -blockers after MI should continue to do so at the time of exercise testing. Because patients will be taking these medications for an indefinite period after MI, the exercise test response while on β -blockers provides information regarding the adequacy of medical therapy in preventing ischemia and arrhythmias, as well as controlling the heart rate and blood pressure response during exercise. Moreover, discontinuation of β -blockers solely for the purpose of exercise testing may expose the patient to the unnecessary risks of recurrent ischemia, arrhythmias, and adverse hemodynamic responses during exercise.3

Chronic Ischemic Heart Disease

Exercise testing provides valuable information in ambulatory patients with chronic ischemic heart disease. Patients with excellent exercise tolerance usually have a good prognosis regardless of anatomical extent of CAD. When the Duke treadmill score is used, patients are at high risk (mortality of 5% per year) if their score is -11, which generally requires both angina and significant ST depression at a low level of exercise. Similarly, patients are at low risk (mortality <1%per year) if exercise capacity is >7 METs, with normal exercise ECG and no chest discomfort.32 Exercise scoring systems that incorporate exercise-induced ECG changes, exercise-induced angina, and exercise capacity improve the prognostic estimates over what could be obtained with the exercise ECG alone. The sample nomogram in Figure 6 illustrates the use of exercise testing in predicting cardiovascular mortality. The decision to perform myocardial revascularization should consider the fact that in patients with less extensive CAD (eg, 1- to 2-vessel disease and well-preserved

LV function), a similar degree of exercise-induced myocardial ischemia does not indicate the same increased risk of cardiac events as with patients with more extensive disease (eg, 3-vessel disease or those with impaired LV function).

Cardiac Events in Subjects With Silent Myocardial Ischemia

In the presence of unstable angina, asymptomatic (silent) ischemia detected by ambulatory ECG (Holter) recording seems to confer an adverse prognosis. Subjects with silent ischemia may be at greater risk for sudden cardiac death because they do not have an intact "warning system." However, in 3 large population studies of subjects with a high prevalence of CAD who underwent exercise testing, those with ST segment depression with or without angina during testing had similar prognoses.⁶¹ Ischemia is silent in $\approx 60\%$ of subjects with ischemic ST segment depression. In patients with established CAD, silent exercise-induced ST segment depression confers increased risk of subsequent cardiac events; the magnitude of the prognostic gradient is a feature of patient selection criteria and coronary disease extent.

Exercise Testing After Coronary Revascularization Therapy

The magnitude of improvement in exercise-induced ischemic responses and aerobic capacity after CABG depends in part on the degree of revascularization achieved and LV function. CABG has been shown to improve survival in one study of subjects with cardiomegaly in whom exercise testing determined aerobic capacity <5 METs or a maximum exerciseinduced systolic blood pressure response <130 mm Hg. A second study of CABG subjects with exercise-induced ST segment depression >1.5 mm showed enhanced survival with surgery. In a third study, the greatest survival benefit after CABG was in subjects with exercise-induced ST segment depression >1 mm at workloads <5 METs. In subjects with exercise capacity >10 METs, CABG could not be shown to improve survival compared with medical therapy. The diagnostic and prognostic utility of exercise testing late after CABG (>5 years) is greater than testing performed earlier, because late after CABG an ischemic response is more likely to indicate graft occlusion, stenosis, or coronary disease progression in the native circulation. After percutaneous transluminal coronary angioplasty (PTCA), restenosis occurs in \approx 20% to 40% of patients, usually within the first 6 months (1 to 2 months after stent placement). Restenosis is more common in patients with diabetes, proximal disease in the left anterior descending artery and in those in whom the post-PTCA result is suboptimal. An abnormal exercise ECG response within a few weeks after PTCA may be secondary to a suboptimal angiographic result, impaired coronary vascular reserve in a successfully dilated vessel, or incomplete revascularization. For this reason, the predictive value of exercise electrocardiography to detect restenosis early after PTCA is suboptimal. Serial conversion of an initially normal exercise test immediately after PTCA to an abnormal test 6 months after PTCA, particularly when the latter response occurs at lower exercise workloads, is usually associated with restenosis. Exercise electrocardiography with imaging techniques early after PTCA can be used to help determine the need for a staged procedure in a patient with multivessel coronary disease who undergoes single-vessel coronary angioplasty and to provide a referenced baseline for subsequent follow-up.

Exercise-Induced Ventricular Arrhythmias

In subjects with CAD, exercise-induced ventricular arrhythmias do not usually represent an independent risk factor for subsequent mortality or coronary events.

Prognostic Scores

Scores based on the coefficients from Cox proportional hazard models seem to be the optimal way of estimating cardiovascular mortality. The Duke treadmill prognostic score is the most widely used, and it is based on the presence of angina and ischemic ST depression during the test, as well as the peak duration (or MET level achieved).³² The sample Duke nomogram in Figure 6 is an example of the use of exercise testing to predict cardiovascular mortality.

Asymptomatic Subjects

Routine screening of asymptomatic individuals with exercise tests is not recommended, and detailed guidelines for exercise testing in asymptomatic persons are presented in the "ACC/ AHA Guidelines for Exercise Testing."³ Although there is evidence that the development of an ischemic ECG response at low workloads of testing among asymptomatic men is associated with a higher relative risk of future events such as angina pectoris, MI, and sudden death, the absolute risk of cardiac events in these populations remains low.64 A study using the Ellestad protocol in asymptomatic men and women with known CAD⁶⁵ found that ECG ischemic changes and an exercise duration ≤ 5 minutes correlated with subsequent events in men >40 years but concluded the exercise ECG had limited value in women and in men ≤ 40 years. A recent study in 6100 asymptomatic men who were free of clinically detectable cardiovascular disease revealed that the occurrence of frequent premature ventricular depolarizations during exercise testing was associated with a long-term (25 year) increase in the risk of death from cardiovascular causes; no significant increase in shorter term risk was reported.⁶⁶

With regard to subjects who are asymptomatic but have risk factors for CAD, the results of exercise ECG testing are different. In the Seattle Heart Watch Study,64 men with one or more risk factors (positive family history, smoking, hypertension [blood pressure >140/90 mm Hg], and hypercholesterolemia [total cholesterol >240 mg/dL]) and 2 abnormalities on exercise testing (chest pain, exercise <6 minutes, ST depression >1.0 mm, or <90% predicted heart rate) had a 30-fold increase in 5-year cardiac risk. Exercise testing was of no predictive value in the group with no risk factors. In the Lipid Research Clinics Coronary Primary Prevention Trial,67 hypercholesterolemic men with >1 mm of ST depression on exercise testing had a 5.7 times greater risk of death from CAD than those with a negative test. Interestingly, a positive test was not significantly associated with nonfatal MI. The Multiple Risk Factor Intervention Trial⁶⁸ reported a nearly 4-fold increase in 7-year CAD mortality among men with an abnormal exercise ECG and suggested that the exercise ECG might serve to identify high-risk men who could benefit from risk factor reduction. Similar data regarding the use of the exercise ECG in women and the elderly (age >75 years) are lacking. In fact, studies have reported a lower specificity for ST segment depression in women⁶⁹ and the elderly.^{70,71}

Therefore, in asymptomatic men >40 years of age with one or more risk factors, exercise testing may provide useful information as a guide to aggressive risk factor intervention.⁷² The role of exercise testing in asymptomatic women and among the elderly (age >75 years) as a guide to identifying the high-risk patient for primary prevention requires further study.

Other Uses of the Exercise Test

Assessment of Valvular Heart Disease

The utility of exercise testing in clinical decision-making for patients with valvular heart disease is discussed in detail in the ACC/AHA's "Guidelines for the Management of Patients With Valvular Heart Disease."⁷³ Exercise testing has been used in subjects with valvular heart disease to quantify disability, to reproduce exercise-induced symptoms, and to evaluate responses to medical and surgical interventions.⁷⁴ The exercise test has also been used to identify concurrent CAD, but there is a high prevalence of false-positive responses (ST depression not due to ischemia) because of frequent baseline ECG abnormalities and LV hypertrophy. Some physicians use exercise testing to help determine when surgery is indicated on the basis of a reduction in functional capacity or abnormal hemodynamic response.⁷⁴

Aortic Stenosis

Effort syncope in subjects with aortic stenosis75,76 is an important and well-appreciated symptom. Most guidelines for exercise testing list moderate to severe aortic stenosis as a relative contraindication for testing because of concern about syncope and cardiac arrest. Proposed mechanisms for exercise-induced syncope in subjects with aortic stenosis include carotid hyperactivity, LV failure, arrhythmia, and LV baroreceptor stimulation. Exercise testing is relatively safe in both the pediatric and adult subject with aortic stenosis when performed appropriately. Attention should focus on the subject's symptoms, minute-by-minute response of blood pressure, slowing heart rate, and ventricular and atrial arrhythmias. In the presence of an abnormal blood pressure response, the subject with aortic stenosis should take at least a 2-minute cool-down walk at a lower stage of exertion to avoid acute LV volume overload, which may occur when the subject lies down.

Exercise has an important role in the objective assessment of symptoms, hemodynamic response, and functional capacity, although ST segment changes are likely to be nonspecific.

Aortic Regurgitation

Subjects with aortic regurgitation⁷⁷ usually maintain a normal exercise capacity for a longer time than those with aortic stenosis. During exercise, the decreases in diastolic duration and regurgitation volume favor forward output. As the myocardium fails, heart rate tends to slow, and ejection fraction and stroke volume decrease.

Mitral Stenosis

Subjects with mitral stenosis⁷⁸ may show either a normal or excessive increase in heart rate during exercise. Because stroke volume cannot be increased, the normal rise of cardiac output is attenuated and may eventually fall during exercise; this is frequently accompanied by exercise-induced hypotension.

Mitral Regurgitation

Subjects with mild-to-moderate mitral regurgitation⁷⁹ maintain normal cardiac output during exercise. Blood pressure, heart rate, and ECG responses are usually normal. When mitral regurgitation occurs suddenly during exercise as a result of ischemic papillary muscle dysfunction, a flat response in systolic blood pressure can occur. Subjects with severe mitral regurgitation do not necessarily have a decreased cardiac output and limited exercise capacity. However, a hypotensive response can develop, and arrhythmias frequently occur.

Exercise Prescription

An exercise test is often used to evaluate the safety of exercise training at various intensities, which is useful in formulating an exercise prescription.

Functional Classification of Disability

Exercise testing is used to determine the degree of disability in subjects with various forms of heart disease. Subjects who exaggerate their symptoms or who have a psychological impairment may often be identified. Exercise testing is a more accurate measure of the degree of cardiac impairment than a physician's assessment of exercise capacity. $\dot{V}o_{2\mmmode max}$ is the best noninvasive measurement of the exercise capacity of the cardiovascular system. Inability to reach 5 METs (<18 mL \cdot kg⁻¹ \cdot min⁻¹) without signs or symptoms is a criterion of disability used by the Social Security Administration. Determination of a subject's exercise capacity affords an objective measurement of the degree of cardiac impairment and can be useful in treatment.⁸⁰

Evaluation of Perioperative Risk for Noncardiac Surgery

Details regarding the use of stress testing in the assessment of perioperative cardiovascular risk during noncardiac surgery are presented in the ACC/AHA's "Guidelines for Perioperative Cardiovascular Evaluation for Noncardiac Surgery."⁸¹ Results of exercise testing with assessment of functional capacity seem to add to the risk stratification provided by the resting ECG in subjects without known CAD who are candidates for major elective noncardiac surgery.^{3,82} However, pharmacological stress imaging techniques are preferred in patients who are unable to perform adequate exercise, particularly those patients before peripheral vascular surgery who are limited by claudication.

Assessment of Special Populations

The Elderly

The optimal use of exercise testing in the elderly requires that age-associated changes in the response to aerobic exercise and age differences in the prevalence and severity of CAD

TABLE 6. Age-Associated Alterations in Physiological Response to Aerobic Exercise

Reduced aerobic capacity: decline in $\dot{V}_{0_2\mbox{ max}}$ of 8% to 10% per decade in nontrained populations						
Reduced maximal heart rate of 1 beat/min per year						
More rapid increase in systolic blood pressure with exercise						
Attenuated rise in ejection fraction						

and comorbid conditions be considered. The physiological response to aerobic exercise undergoes important changes with aging, even in the absence of cardiovascular disease (Table 6). Maximal aerobic capacity, as indexed by $\dot{V}O_{2 \text{ max}}$, declines 8% to 10% per decade in sedentary men and women.83-85 An age-related decline in maximal heart rate of ≈ 1 beat/min per year is the major contributor to this reduction in aerobic capacity.85,86 For treadmill exercise, the formula 220 minus age provides a reasonable prediction of maximal heart rate response throughout the adult age span in unmedicated patients of either sex, although the standard deviation of 12 beats/min limits the ability to accurately predict maximal heart rate in an individual. The systolic blood pressure response to maximal aerobic exercise is increased with age.87 The age-associated rise is more pronounced in women than men, paralleling the steeper ageassociated increase in resting systolic blood pressure in women. Finally, aging is accompanied by a less complete emptying of the left ventricle during strenuous aerobic exercise, as reflected by a blunted increase in LV ejection fraction.86,88 Because the augmentation of plasma catecholamines during exercise seems to be preserved or increased in older adults,89 a unifying explanation for the age-associated reduction in heart rate and ejection fraction responses to maximal aerobic exercise is a decrease in β -adrenergic responsiveness.

Numerous noncardiac conditions that frequently occur in older adults may limit their ability to undergo aerobic exercise testing. Some disorders, such as peripheral arterial disease and chronic obstructive lung disease, frequently coexist with CAD due to shared risk factors. Degenerative arthritis of weight-bearing joints is the most prevalent chronic disorder in older adults. Finally, the unfamiliarity with vigorous exercise and exercise testing equipment may intimidate elderly patients, causing them to perform submaximally.

To understand how age per se might affect the diagnostic utility of exercise testing, it is essential to recognize how aging affects the characteristics of CAD. Large autopsy studies have demonstrated that the prevalence of CAD, as defined by a diameter stenosis >50% in one or more coronary arteries, increases dramatically with age.^{90–92} In addition, coronary angiographic data from the Coronary Artery Surgery Study, the Duke data bank, and other series have documented an age-associated increase in CAD severity.⁹³ Because more severe CAD is more readily detected by exercise or pharmacological stress testing than milder disease, an age-associated increase in the sensitivity of exercise testing for the prediction of CAD might be expected. Such a finding has been documented for the exercise ECG, with an increase in sensitivity from 56% in patients <40 years to 84%

in those >60 years.⁷¹ However, the specificity of the exercise ECG declined from 84% in patients <40 years to 70% in those >60 years.⁷¹

The most common modalities used to perform maximal aerobic exercise testing are the motorized treadmill and the electronically or mechanically braked cycle ergometer. The treadmill is preferred in older subjects without significant balance or gait disturbances, whereas cycle ergometry is preferable for older patients with gait or balance disorders but preserved muscle strength, although peak $\dot{V}o_2$ during the latter is lower.⁹⁴

Regardless of whether treadmill or cycle ergometry is used, a protocol with modest, equal increments in work rates should be employed to achieve an exercise duration of 8 to 12 minutes. The use of small, more frequent increments in work rate is preferable to larger, less frequent increases, both physiologically and psychologically. Protocols using a constant speed, with small elevations of treadmill grade every 2 minutes, provide more data points with lesser need for gait changes than the simultaneous increases of speed and elevation every 3 minutes during protocols such as the Bruce. Similarly, cycle ergometric exercise tests should start at a low resistance and progress in modest increments. For either treadmill or cycle ergometry, some laboratories use a ramp protocol with small, almost imperceptible, increments of work rate every minute or less.95 Regardless of the exercise modality or specific protocol, adequate time should be allowed to familiarize the older patient with the testing equipment and to provide a 1- to 2-minute warm-up period. These pretest maneuvers will help alleviate the anxiety of the elderly patient and reduce the risk of musculoskeletal injury.

Despite the greater prevalence and severity of CAD with age, exercise testing remains as safe a procedure in the elderly as in younger populations. National surveys of exercise laboratories have documented very low overall risks of MI or cardiac death, and age has not been identified as a risk factor for these events. However, age-associated increases in isolated ectopic beats and nonsustained supraventricular and ventricular arrhythmias, even in clinically asymptomatic subjects,^{96,97} have been observed. The supervising clinician should be aware that myocardial ischemia or MI in the elderly may present as marked dyspnea rather than chest discomfort.

Exercise testing is well established as a useful tool for assessing the progress of patients with stable CAD and those after MI. Available data in the elderly, although more limited, suggest similar prognostic value in this age group. As in the general post-MI population, inability to perform treadmill exercise after infarction confers a high risk for future mortality. In 111 infarct survivors >64 years, one group observed a 1-year mortality of 37% in the 63 patients not eligible for exercise testing versus only 4% in those able to exercise.98 In the latter group, 1-year mortality was best predicted by the magnitude of systolic blood pressure rise during exercise; mortality was 15% in patients with an increase <30 mm Hg versus 1.8% in those with an increase >30 mm Hg.99 The prognostic importance of systolic blood pressure response to exercise was confirmed in 188 post-MI patients >70 years.¹⁰⁰ In this latter study, peak cycle work rate <60 W, exercise duration <5 minutes, and increase in rate-pressure product <12 500 also predicted increased cardiovascular mortality. In contrast, ST segment depression and ventricular arrhythmia predicted recurrent MI and need for coronary revascularization but not mortality.

In older patients with stable CAD, exercise testing also has diagnostic and prognostic utility. In 419 CAD patients >65 years, one study revealed that severe ST segment depression induced by cycle ergometry predicted triple-vessel disease.¹⁰¹ Ischemic ST segment depression predicted an increased risk of cardiac death in another study of older patients with stable CAD.¹⁰² Although not recommended for routine use in apparently healthy older adults, exercise testing has also demonstrated prognostic significance in such a population.¹⁰³

In summary, the utility and safety of exercise testing in the elderly are similar to those in younger populations. However, age-associated changes in exercise physiology and the frequent presence of both cardiovascular and noncardiovascular comorbid conditions require concerted efforts to match the older patient with an appropriate exercise testing protocol.

Obese Subjects

Exercise testing is useful in the clinical evaluation of obese patients with known or suspected CAD. However, obtaining an accurate assessment of peak cardiopulmonary responses often poses a challenge in this patient population. For many obese patients, particularly the morbidly obese, this is related to gait instability, low functional capacity, coexisting orthopedic impairments, and uneven body weight distribution. In one study, 25 obese women (mean body mass index, 40 kg/m²) were assigned to various ramp and Bruce or modified Bruce protocols on the basis of a pretest activity questionnaire. Despite a longer time to reach fatigue using the ramp protocols, mean peak Vo2 was not significantly different between tests. In another study, obese subjects with CAD were assigned to 2 severe energy-deficient study groups (one with exercise and the other by diet) plus a control group. All had exercise testing with $\dot{V}O_2$ studies 6 times in a 2-year period with the Weber-Janicki protocol.104 There were no differences between groups with the testing methodology, and all completed each test with satisfactory end points. In conclusion, these 2 studies and clinical experience reveal that obese subjects can have exercise tests effectively performed using a variety of protocols. Low-impact walking protocols, however, are preferred in this patient population.

The Physically Disabled

Special protocols are available for testing¹⁰⁵ musculoskeletally disabled subjects, especially those with hemiplegia or paresis after stroke or those with lower limb amputation or spinal cord injury. Many testing protocols use arm cycle ergometry with the subject sitting to optimize the exercise load, but some protocols consist of arm-leg or leg cycle ergometry. Safe and effective testing can be performed by most of these subjects.

The exercise testing method has been derived from a clinical trial¹⁰⁵ that evaluated the effects of exercise on physically disabled subjects with CAD. The subjects had documented CAD and a physical disability with the use of at least one arm. The exercise protocol was a graded arm ergometry test, adapted from the original Schwade Arm

Ergometer Protocol.¹⁰⁶ The protocol began at a resistance of 20 W and increased by 10 W per stage. The revolutions per minute of the arm ergometer remained constant at 50, each stage lasting 2 consecutive minutes, with a 1-minute rest period before beginning the next stage.

Subjects in the Emergency Room

Detailed recommendations regarding exercise testing among patients who present to the emergency room or chest pain centers are presented in the "AHA Advisory on Safety and Efficacy of Exercise Testing in Chest Pain Units"¹⁰⁷ and the "ACC/AHA Guidelines for Exercise Testing."3 Patients who present to the emergency department are a heterogeneous population with a large range of pretest risks for coronary disease. The accuracy of exercise testing in the emergency department setting follows Bayesian principles, with the greatest diagnostic and prognostic estimates in intermediaterisk clinical patient subsets. Exercise treadmill testing should be considered in patients who present to the emergency department with symptoms such as chest discomfort when they are classified as "low risk," which includes the following: 2 sets of cardiac enzymes at 4-hour intervals are normal; ECG at the time of presentation and pre-exercise test shows no significant changes; the rest ECG has no abnormalities that preclude accurate assessment of the exercise ECG; and the patient is asymptomatic or has minimal atypical chest pain from admission to the time results are available from the second enzyme set.108

Early exercise testing has been applied in patients with chest pain who are identified as low risk by clinical assessment, which may include a predictive instrument such as the Goldman computer protocol.¹⁰⁹ Exercise testing has been implemented using 2 approaches. In the majority of studies, it is performed soon after presentation after an acute coronary syndrome has been excluded. Acute coronary syndromes are ruled out by an accelerated diagnostic protocol, which is usually performed within a 6- to 12-hour interval with serial cardiac serum markers and electrocardiograms. In the second, less common strategy, selected low-risk patients undergo "immediate" exercise testing to stratify the group into those who can be discharged directly from the emergency department and those who require admission. Both methods have thus far been shown to be safe, informative, and costeffective, although experience with the latter is considerably more limited than with the former.

The feasibility of "early" exercise testing after excluding an acute coronary syndrome has been demonstrated by a number of recent studies involving from 100 to >400 patients presenting with chest pain and negative results on an accelerated diagnostic protocol.^{110–115} Patients with negative exercise tests were discharged, and those with positive results were admitted. No adverse effects of exercise testing have been reported. Direct discharge of patients after a negative exercise test reduced hospital admissions for the initial presentation by \approx 50%.^{110,111} A negative exercise test was associated with no cardiac events at 30 days¹¹² and at 5-month¹¹³ follow-up. Compared with patients with a positive test, those with negative tests had equivalent^{110,111} or fewer readmissions¹¹⁴ at 1 to 6 months. Substantial cost savings have also been demonstrated with an accelerated management protocol that included exercise testing.^{110,112}

Clinical experience with immediate exercise testing (serial cardiac markers not measured) is limited, and this was initially evaluated in small pilot studies of 28 patients¹¹⁶ and 32 patients.¹¹⁷ In the former study, all patients were admitted after the exercise test (23 negatives, 5 positives) for a full inpatient evaluation, which was uniformly negative. There were no adverse effects of exercise testing and no cardiac events in any patients in either of the trials at the 6-month follow-up.

These preliminary studies were extended in a series of recent investigations in which immediate exercise testing was applied in low-risk patients presenting with chest pain and normal, near-normal, or unchanged electrocardiograms.^{108,118–120} The initial study included 93 patients with no prior history of CAD, and exercise testing was performed by cardiologists.¹¹⁸ Subsequent reports included 212 patients (a small number of whom had CAD), with exercise testing performed by internists,¹¹⁹ and a series of 100 patients, all with known CAD.¹⁰⁸ This method has been applied in >1000patients¹²⁰ during the past 5 years, and there have been no reported adverse effects of exercise testing. All of those in the group with negative exercise tests were discharged directly from the emergency department, and follow-up at 30 days revealed a cardiac event in <1%. However, this approach has been associated with a small risk (<1.0%) of inadvertent exercise testing of patients with evolving, non-Q-wave infarction (but it has been associated with no complications).120

In summary, the feasibility and potential cost-effectiveness of early exercise testing to facilitate management of low-risk patients presenting to the emergency department with chest pain has been demonstrated. The majority of these protocols have used exercise testing after an accelerated diagnostic protocol of 6 to 12 hours to rule out an acute coronary syndrome. When performed after ruling out MI, exercise testing seems to be safe, accurate and cost-effective.

Drugs and Exercise Testing

β-Blockers

Subjects with angina who receive β -blockers may achieve a higher exercise capacity with less ST segment depression and less angina if the drugs prevent them from reaching their ischemic rate-pressure product and therefore translate into a reduction in diagnostic accuracy. Maximum heart rate and systolic blood pressure product may be reduced. The time of ingestion and the dosage of these medications before testing should be recorded. Whether to discontinue β -blockers before testing was discussed under "Subject Preparation."

Vasodilators

These agents can increase exercise capacity in subjects with angina pectoris.¹²¹ There has been no scientific validation that long-acting nitrates increase exercise capacity in subjects with angina when they are tested after long-term administration.

Digitalis

ST-segment depression can be induced or accentuated during exercise in individuals who are taking digitalis, including both normal subjects and subjects with CAD.¹²² A normal QT interval is associated with digitalis-induced ST changes, whereas prolonged QT intervals occur with ischemia, other type 1 antiarrhythmic drugs, electrolyte imbalance, and other medical problems. Exercise-induced ST segment depression may persist for 2 weeks after digitalis is discontinued.

Diuretics

Most diuretics have little influence on heart rate and cardiac performance but do decrease plasma volume, peripheral resistance, and blood pressure. Diuretics can cause hypokalemia, which results in muscle fatigue, ventricular ectopy and, rarely, ST-segment depression.

Obtaining Informed Consent for Exercise Testing

Although obtaining written consent from a subject does not protect a physician from legal action, a signed consent form is nonetheless desirable to provide a written record that documents the informed consent process. A sample consent form is shown below.

Informed Consent for Exercise Testing

To determine my cardiovascular response to exercise, I voluntarily agree to engage in an exercise test. The information obtained about my heart and circulation will be used to help my doctor advise me about activities in which I may engage.

I have been told that before I undergo the test, I will be interviewed and examined by a physician in an attempt to determine if I have a condition indicating that I should not engage in this test. I am told that the test I will undergo will be performed on a _____ (description), with gradually increasing effort until symptoms such as fatigue, shortness of breath, or chest discomfort may appear, indicating to me that I should stop. I have been told certain changes may occur during the test, including abnormal blood pressure, fainting, abnormal ECG showing heart "strain," disorders of heart beat (too rapid, too low, or ineffective), and, possibly, heart attack and death.

I have read the above and understand it, and my questions have been answered to my satisfaction.

Subject:		
	supervising the test:	
•	1 8	
Date:		

Exercise Training

Recent physical activity recommendations from the Centers for Disease Control and Prevention and the American College of Sports Medicine (ACSM),¹²³ the US Surgeon General,¹²⁴ and the AHA¹ have expanded the traditional emphasis on formal exercise prescription methodology to include a broader public health perspective with regard to physical activity. These reports have increased both professional and public awareness of the health benefits associated with daily participation in physical activity, even at moderate-intensity activity; that is, activities that include both leisure time and those of a vocational and avocational nature. However, more intense activities, including activities of longer duration and more vigorous intensity, are likely to provide additional health benefits.

Care must be taken to ensure that apparently healthy individuals who are beginning an exercise training program do not have detectable disease and that persons with known disease are stable, with no evidence of new or changing symptoms. Accordingly, medical evaluation should be obtained before entry into an exercise training program unless the anticipated activity is of light to moderate intensity, eg, brisk walking. Use of the risk stratification schema outlined in the section on "Medical Evaluation and Exercise Prescription for Individuals With CAD" can help determine the need for exercise testing and the level of subsequent supervision required during exercise training. Exercise testing should be routinely performed in persons with known or suspected cardiovascular disease before beginning an exercise training program. Care should be taken to exclude individuals from training who have evidence of unstable heart disease, such as unstable angina, uncontrolled heart failure, severe aortic stenosis, or complex arrhythmias. Training programs for persons with cardiovascular disease should be medically supervised until safe levels of activity have been established. The extent of medical supervision is discussed under the section titled "Types of Exercise Programming and Monitoring."

Exercise Training Responses

Apparently Healthy Individuals

Exercise training in apparently healthy persons impacts on several areas, including maximal oxygen uptake, central hemodynamic function, autonomic nervous system function, peripheral vascular and muscular function, and submaximal exercise capacity. Collectively, these adaptations result in an exercise training effect, which allows an individual to exercise to higher peak workloads with lower heart rates at each submaximal level of exercise.

Maximal Oxygen Uptake

 $\dot{V}O_{2\mmmode max}$ is the peak oxygen uptake achieved by muscular exercise. By strictest definition, $\dot{V}O_{2\mmmode max}$ cannot be exceeded, despite an increase in power output. Although demonstration of the $\dot{V}O_2$ plateau against work rate is a valid demonstration of $\dot{V}O_2\mmmode max}$, patients often cannot achieve the plateau because of leg fatigue, lack of necessary motivation, and general discomfort. Hence, it is customary to refer to $\dot{V}O_2\mmmode max}$ as the peak $\dot{V}O_2$ attained during volitional incremental exercise. In clinical practice, $\dot{V}O_2\mmmode max}$ is not usually measured during an exercise tolerance test but is estimated from the peak work intensity achieved.

 $\dot{V}o_2$ is the product of cardiac output and systemic arteriovenous oxygen difference. Increased $\dot{V}o_2_{max}$ after training is associated with an increase in the capacity of the cardiovascular system to deliver oxygen (increased cardiac output) and of the muscles to use that oxygen (greater arteriovenous $\dot{V}o_2$ difference). Higher cardiac output after training is achieved

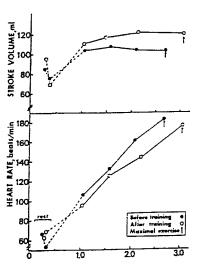


Figure 7. Mean values for stroke volume and heart rate in 15 middle-aged subjects at rest (prone and upright position) and during submaximal and maximum exercise in upright position before and after physical conditioning. Reprinted with permission from Hartley et al.¹²⁵

solely by an increase in stroke volume, because maximal heart rate is not usually increased after training in normal individuals.¹²⁵ On the basis of data in healthy subjects,¹²⁶ a training effect can be achieved in a subject in the presence of selective or nonselective β -adrenergic blockade. However, such changes may be attenuated¹²⁷ and/or may not be detected by metabolic exercise studies until after the drug is withdrawn.¹²⁶

Central Hemodynamic Changes

Although a greater maximal cardiac output can be achieved after training, submaximal values are usually unchanged.¹²⁸ Submaximal heart rate is reduced after training, with a concomitant increase in stroke volume.^{125,128} The mechanism of these changes is not known, although exercise training has resulted in an increase in myocardial contractility in animals.¹²⁹ Figure 7 depicts relations of heart rate and stroke volume before and after training.¹²⁵

Autonomic Nervous System Changes

Blood and urinary catecholamine levels are lower at rest and during submaximal exercise after training, presumably because of less sympathetic nervous system activity.¹³⁰ Parasympathetic tone may also be increased and, with sympathetic adjustments, may account for the slower heart rate and lower arterial blood pressures seen after training.

Peripheral Changes

Skeletal muscle changes after exercise training include increases in oxidative enzyme concentration, capillary density, myoglobin concentration, muscle glycogen, and adaptation of muscle fiber type to a higher percentage of type I fibers. All potentially contribute to greater capacity to use oxygen and to better endurance.¹³¹

Submaximal Endurance Capacity

Endurance training enhances the individual's ability to perform exercise at both submaximal and maximal intensities, as demonstrated either by the ability to exercise longer at a similar workload or by increasing the workload attained at a given heart rate.¹³² Improvements in endurance capacity are due to several factors, including greater availability of oxygen to exercising muscles (increased myoglobin concentration and capillary density), greater use of aerobic processes (greater concentration of oxidative enzymes), and increased muscle glycogen.¹³³ Furthermore, the results of these adaptations lower blood lactate levels and increase the anaerobic threshold. Adaptation to submaximal exercise is also associated with a lower rate-pressure product for a given exercise task, suggesting reduced myocardial oxygen demand for that level of work.

Individuals With Cardiovascular Disease

Although exercise capacity increases with training when heart disease is present, the reported physiological changes seem to differ somewhat from those found in apparently healthy individuals. These are outlined below.

Peak Oxygen Uptake

Subjects with CAD have an increase in $\dot{V}o_{2 \text{ max}}$ with training. Although the absolute magnitude of the change is less in subjects with heart disease than that observed in apparently healthy individuals, the proportional increase is similar and may favorably impact on activities of daily living. The peak heart rate may be the same or slightly greater after training in those with heart disease.¹³⁴ The smallest absolute increments in $\dot{V}o_{2 \text{ max}}$ with training are seen in individuals with heart failure, but even in those subjects the improvement is of great rehabilitative value for restoring ability to perform daily activities.

Cardiac Output

The increase in peak cardiac output is due to an increase in both stroke volume and peak heart rate, which differs from normal subjects, whose peak heart rate usually does not change. Changes in peak heart rate may reflect a greater level of effort applied during follow-up testing. In subjects with cardiac disease, the submaximal cardiac output may be lower at a given workload, with maintenance of $\dot{V}o_2$ by widening the arteriovenous Vo₂ difference after training.¹³⁵ Such a result suggests improved overall efficiency for delivery of oxygen to the tissues. Studies have found that participation in a home exercise training group (compared with a control group) by physically disabled men with CAD significantly improved peak exercise LV ejection fraction and fractional shortening between baseline and 6 months.¹⁰⁵ Another study revealed that in men with CAD, the increment in rest to peak LV ejection fraction improved with 1 year of training only in those performing high-intensity training (85% $\dot{V}O_{2 max}$) but not in those performing low-intensity training (50% $\dot{V}O_{2 max}$).¹³⁶ This improvement occurred in subjects with both depressed $(\leq 50\%)$ and those with normal (>50%) LV ejection fractions. The increase in stroke volume that occurs with shortterm training is likely attributed to augmentation of blood volume and, hence, ventricular preload.137 However, most studies involving patients with severe impairment of LV systolic function attribute the training effect to peripheral rather than central changes.138-140

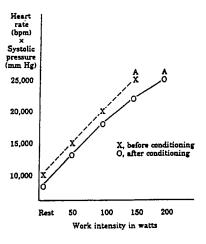


Figure 8. Change in rate-pressure product before and after exercise rehabilitation. Rate-pressure product (heart rate×systolic pressure) is shown at rest, during exercise, and during angina (A). After conditioning, more work can be tolerated because rate-pressure product (and hence myocardial oxygen uptake) is lower at rest and at each level of work intensity. Reprinted with permission from Redwood et al.¹⁴¹

Decreased Myocardial Oxygen Demand

Exercise training has special significance for individuals with CAD because the changes promote lower myocardial oxygen demand at any given workload. These include lower heart rate, lower systolic blood pressure, and lower circulating catecholamines. The benefits of these adjustments can be demonstrated by the greater amount of work that can be done before angina and/or ischemic ST depression occurs.¹⁴¹ Moreover, several provocative studies suggest that there is an improvement in myocardial oxygen supply (ie, coronary blood flow) at a given level of myocardial oxygen demand after training.^{142–144} There are many mechanisms, or combinations thereof, that may explain these findings, which are discussed in the section below. Figure 8 demonstrates the positive effects of conditioning exercise on angina.¹⁴¹

Preventive Value of Regular Physical Activity

Effects of Exercise

There is now general agreement among public health and medical authorities that reduced physical activity on the job and during leisure time, which is commonly associated with modern lifestyles, increases the risk of fatal and nonfatal CAD events, as well as all-cause mortality. National surveys during the past decade have consistently reported that $\approx 80\%$ of American adults have insufficient physical activity for health benefits.^{123,124} Thus, the AHA,¹ the ACC,¹⁴⁵ the Centers for Disease Control and Prevention, the ACSM,123 the National Institutes of Health,146,147 and the US Surgeon General¹²⁴ have declared a sedentary lifestyle a major modifiable coronary risk factor. Other risk factors for which interventions have been proven or judged likely to reduce CAD risk are cigarette smoking, hypertension, elevated plasma low density lipoprotein (LDL) and reduced high density lipoprotein (HDL) cholesterol, elevated plasma triglycerides, obesity, diabetes mellitus, thrombogenic factors, and postmenopausal status.145 Regular aerobic exercise has a favorable impact on a number of these risk factors, as well as an independent effect on other factors described below.

More than 40 epidemiological and observational studies provide the primary basis documenting the inverse relationship between physical activity and risk of CAD. There have been >100 published reports from such studies, with nearly 75% of them supporting an inverse relationship between physical activity and/or fitness and risk of an initial fatal or nonfatal MI.148-150 The populations studied consisted predominantly of initially healthy, middle-aged or older white men; fewer than 10 studies included women. There are few studies involving blacks and other racial and ethnic minorities. Meta-analyses reveal that the sedentary participants in these studies generally had about twice the incidence of death from CAD compared with their more active counterparts.151,152 Longitudinal studies that assessed cardiorespiratory fitness by exercise testing have almost unanimously shown an inverse relationship between fitness and risk of CAD in both men and women. The least fit men and women demonstrated a >5-fold increased risk of death from CAD or cardiovascular disease than the most fit individuals.¹⁵³ Accordingly, on the basis of these data, a consensus has been reached that a minimum of 30 minutes of moderate intensity physical activity (continuous or in 10-minute increments) is required on most (preferably all) days of the week to reduce the risk of CAD events.^{123,146} This is equivalent to ≈ 1.5 miles per day of brisk walking at an energy cost of 150 kcal per day for an average-sized person.

Epidemiological and experimental studies have also identified multiple biological mechanisms that help to explain the apparent effects of physical activity and cardiorespiratory fitness "against" CAD. These mechanisms are reviewed in detail elsewhere^{148,154,155} and may be classified as follows:

- Antiatherogenic effects
- Antithrombotic effects
- Endothelial function alteration
- Autonomic functional changes
- Anti-ischemic effects
- Antiarrhythmic effects

Antiatherogenic Effects

Regular exercise has both direct and indirect beneficial effects on the severity of coronary atherosclerosis. Physical activity is associated with less severe CAD, larger coronary artery luminal diameters, and reduced progression of atherosclerosis.^{155–157} Most of these beneficial effects seem to be due to the attenuation of coexisting risk factors by exercise. These include the following:

- Reduction of adiposity, particularly in those with excess upper body and abdominal fat
- Reduction of elevated blood pressure
- Reduction of elevated plasma triglycerides (and associated small dense LDL particles)
- Increase in HDL cholesterol levels
- Improvement in insulin sensitivity and glucose use and reduction in risk of type 2 diabetes¹⁵⁸

Antithrombotic Effects

Most major CAD clinical events are accompanied by coronary thrombosis. Emerging evidence suggests that exercise training favorably affects this process, in particular, the fibrinolytic system.148,155,157,159-161 In one study, strenuous endurance exercise for 6 months in healthy older patients resulted in a significant improvement in hemostatic indices, with a reduction in plasma fibrinogen levels, an increase in mean tissue plasminogen activator, an increase in active tissue plasminogen activator, and a reduction of plasminogen activator inhibitor.¹⁶¹ Short- and long-term exercise affect platelet activation. Platelet activation is important in the pathophysiological mechanisms of unstable coronary syndromes and acute MI. Available data suggest that short-term exercise can lead to increased platelet activity, especially in sedentary individuals, but regular, long-term exercise may abolish or reduce this response.162

Endothelial Function

The vascular endothelium plays an important role in the regulation of arterial tone and local platelet aggregation, in part through the release of endothelium-derived relaxing factors such as nitric oxide. This release is stimulated by various mechanisms, including the rise in shear stress associated with short- and long-term increases in blood flow.¹⁶³ Endothelium-dependent dilation is impaired in patients with coronary atherosclerosis and in patients with coronary risk factors, including hypercholesterolemia, diabetes mellitus, cigarette smoking, and hypertension.¹⁶⁴ Emerging evidence suggests that aerobic exercise improves endothelial function.^{165,166}

Autonomic Function

The balance between sympathetic and parasympathetic activity modulates cardiovascular activity. Enhanced sympathetic nervous system activity seems to be associated with an increased risk of cardiac events, particularly in those patients with known heart disease. Using measures of heart rate variability, cross-sectional studies of healthy men reported higher parasympathetic activity among those who were physically trained and fit compared with those who were not.¹⁶⁷ Whether exercise affects autonomic tone among patients with cardiovascular disease is unclear. However, improved measures of heart rate variability with exercise training have been shown in patients with chronic heart failure and in patients after MI.^{168,169}

Anti-Ischemic Effects

There are a number of mechanisms by which endurance exercise training may improve the relative balance between myocardial oxygen supply and demand and thereby result in an anti-ischemic effect. Increased metabolic capacity and improved mechanical performance of the myocardium are well-substantiated adaptations to endurance exercise training.^{155,157,159} Lowered heart rate and systolic blood pressure during submaximal exertion reduce myocardial work, thereby reducing myocardial oxygen demands and coronary blood flow requirements. Among patients with CAD, this allows a greater absolute workload to be accomplished before reaching the ischemic threshold. In addition, heart rate slowing

with training allows more time during diastole for coronary blood flow to perfuse the myocardium.

Antiarrhythmic Effects

Increased risk of ventricular fibrillation during strenuous exercise in the presence of CAD is well documented. Exercise training–induced improvement in the myocardial oxygen supply-demand balance and concomitant reduction in sympathetic tone and catecholamine release is postulated to attenuate the risk of ventricular fibrillation. This may explain the lower rate of sudden cardiac death observed in physically active men with known or suspected CAD or a high risk of CAD.^{155,157,159,170}

Hypertension

Two cohort studies have demonstrated that regular exercise reduces the incidence of hypertension.^{171,172} In addition to preventing hypertension, regular exercise has been found to lower blood pressure in hypertensive subjects. In mildly hypertensive men, short-term physical activity decreased blood pressure for 8 to 12 hours after exercise, and average blood pressure was lower on exercise days than on nonexercise days.¹⁷³ In hypertensive black men, moderate physical activity performed for 16 to 32 weeks resulted in a decrease in diastolic blood pressure, which was sustained after a reduction in antihypertensive medication.174 Randomized controlled trials of exercise and blood pressure have revealed that regular exercise reduces both systolic and diastolic blood pressures.^{175–177} The average reduction in blood pressure is 10 mm Hg for systolic and 7.5 mm Hg for diastolic pressures.178

Diabetes Mellitus

Physical activity has beneficial effects on both glucose metabolism and insulin sensitivity. These include increased sensitivity to insulin, decreased production of glucose by the liver, larger number of muscle cells that use more glucose than adipose tissue, and reduced obesity.¹⁷⁹ The effect of physical activity is an independent effect, but this is further increased with weight reduction.

Obesity

Exercise training is an important contributor to weight loss, although the effect of exercise is quite variable. It is not clear how much exercise is required to prevent weight gain or "repeat" weight gain, although it has been suggested that the levels may be much higher than the currently recommended doses of physical activity.^{180,181} Most controlled exercise training studies show only modest weight loss (≈ 2 to 3 kg) in the exercise group. However, when diet is added to the exercise program, the average weight loss is 8.5 kg, most of which is body fat, whereas a diet-only program results in a lesser weight loss (5.1 kg). Over the same study period, those undergoing neither diet nor exercise programming increased weight by an average of 1.7 kg.182,183 These data strongly support the role of both exercise and diet in weight loss programs. Body composition and fat distribution are linked to cardiovascular mortality¹⁸² and are improved by exercise. Physically active men and women have a more favorable waist-to-hip ratio (ie, less central obesity) than do sedentary individuals.¹⁸⁴ In general, the goal is caloric expenditure, which is best achieved in most people by exercise that is moderate in intensity and low impact, such as brisk walking or cycling, and used for a longer duration and frequency. Such exercise must involve a long-term commitment by the individual to achieve and maintain the weight loss.

Lipids

There is much variability in the results of exercise/lipid lowering studies, at least in part due to the heterogeneity of the study methods, study duration, populations, exercise interventions, and the use of adjuvant interventions such as diet or pharmacological lipid-lowering agents. A meta-analysis of 95 studies, most of which were not randomized controlled trials, concluded that exercise leads to a reduction of 6.3% in total cholesterol, 10.1% in LDL cholesterol, and 13.4% in total/HDL cholesterol ratio and a 5% increase in HDL.185 It seems that the training intensities required to yield modest improvements in lipids are not as high as those that lead to improvements in fitness levels, because HDL seems to increase across a broad spectrum of exercise intensities.186,187 A recent randomized controlled trial of moderate-intensity exercise (equivalent to brisk walking of 10 miles per week), Step 2 AHA diet, and the combination of diet plus exercise revealed that those in the diet plus exercise group demonstrated an 8% to 12% reduction in LDL and a -2% to 2% change in HDL level after 1 year. In this study, the addition of exercise to diet produced significant reductions in LDL that diet alone did not. Triglyceride levels were normal in these patients and did not change with exercise.¹⁸⁸ However, in patients with hypertriglyceridemia, a decrease of 15% to 30% can occur, particularly in those with insulin resistance.189

Because estrogen causes an increase in HDL, studies regarding women are confounded by menopausal status and estrogen use, which are frequently not reported. A recent study examined the effects of vigorous exercise on HDL in women runners, demonstrating increased HDL levels with increasing amounts of exercise, which continued to rise in women who ran >64 km per week.¹⁸⁷ This dose-response relationship persisted in premenopausal and postmenopausal women and in those on oral contraceptives and estrogen replacement therapy. Although these studies suggest an improvement in lipid profile with exercise training, the effects are quite modest. These improvements may have a favorable effect on cardiovascular risk; however, exercise is unlikely to normalize cholesterol levels in persons with genetically based lipid disorders.

Quality and Quantity of Exercise Needed for a Beneficial Effect

Any activity performed for training should be assessed in terms of intensity, frequency, duration, mode, and progression. Dose refers to the total amount of energy expended in physical activities that require repetitive muscular movement (usually expressed in kilojoules or kilocalories). Intensity can be defined in absolute or relative terms. Absolute intensity reflects the rate of energy expenditure during exercise and is usually expressed in METs. Relative intensity refers to the relative percentage of maximal aerobic power that is main-

	Endurance-Type Activity									
		Relative Intensity			Absolute Intensity in Healthy Adults (Age), METs					
Intensity	Vo _{2 max} , %	Maximum Heart Rate, %	RPE†	Young (20–39)	Middle-Aged (40–64)	Old (65–79)	Very Old (80+)	RPE†	Maximum Voluntary Contraction, %	
Very light	<20	<35	<10	<2.4	<2.0	<1.6	<1.0	<10	<30	
Light	20–39	35–54	10–11	2.4-4.7	2.0-3.9	1.6–3.1	1.1–1.9	10–11	30–49	
Moderate	40–59	55–69	12–13	4.8-7.1	4.0-5.9	3.2-4.7	2.0-2.9	12–13	50-69	
Hard	60–84	70–89	14–16	7.2–10.1	6.0-8.4	4.8-6.7	3.0-4.25	14–16	70–84	
Very hard	≥85	≥90	17–19	≥10.2	≥8.5	≥6.8	≥4.25	17–19	≥85	
Maximum‡	100	100	20	12.0	10.0	8.0	5.0	20	100	

TABLE 7. Classification of Physical Activity Intensity*

*Based on 8 to 12 repetitions for persons <50-60 years old and 10 to 15 repetitions for persons aged \geq 50-60 years.

†Borg rating of Relative Perceived Exertion (RPE), 6-20 scale.

\$Maximum values are mean values achieved during maximum exercise by healthy adults. Absolute intensity values are approximate mean values for men. Mean values for women are ~1 to 2 METs lower than those for men.

Adapted from Reference 190.

tained during exercise and is expressed as a percentage of maximal heart rate or a percentage of $\dot{V}_{0_2 max}$. For example, brisk walking at 4.8 km/hour (3 miles/hour) has an absolute intensity of ~4 METs. In relative terms, this intensity is considered light for a 20-year-old healthy person but represents a hard intensity for an 80-year-old.¹⁹⁰ Activities that are 40% to 60% of $\dot{V}_{0_2 max}$ are generally categorized as moderate intensity. This concept is illustrated and further defined in Table 7. Table 8 lists the energy requirements of various activities. Body weight must be used to calculate calories because METs are weight-adjusted. The following conversion formula can be used: kilocalories per minute=[(METs×3.5×body weight in kilograms)/200].

The intensity of activity needed to improve physical conditioning varies among individuals and may be as low as 40% of VO2 max for 20 minutes 3 times per week.191 However, the relationship of exercise intensity to duration suggests that lower intensity exercise requires more time to increase functional capacity than higher intensity exercise. From a health and conditioning standpoint, the major advantage of moderate-intensity exercise is the decreased likelihood of complications, whereas more vigorous exercise has the advantage of accomplishing the goal in less time and in further increasing cardiovascular conditioning. Experience with normal populations suggests that activity \geq 700 kcal (2940 kJ) per week is associated with higher peak exercise capacities. In studies of male college alumni, the risk of death became progressively lower as physical activity dose levels increased from an expenditure of 2.1 to 14.7 kJ/week (500 to 3500 kcal/week). There was a 24% reduction in cardiovascular mortality in subjects whose energy expenditure was >8.4kJ/week (2000 kcal/week). Alumni who were initially inactive and later increased their activity levels demonstrated significantly reduced cardiovascular risk compared with those who remained inactive.¹⁹² The data regarding exercise intensity are much less clear than those addressing total dose. There is a growing body of evidence that regular, moderateintensity activity (17 to 29 kJ/min; 4 to 7 kcal/min), performed by men and women of a broad age range, reduces cardiovascular mortality.^{124,146,193–195} A recent report involving 802 men (aged 64 to 84 years) concludes that more intense activity (>4 METs) is more strongly associated with lower cardiovascular mortality than is less intense activity.¹⁹³ Another report¹⁹⁶ noted that only energy expended during vigorous activity (>6 METs) was associated with a reduction of mortality among male Harvard alumni. However, at least 2 studies in older adults have demonstrated reduced mortality in walkers compared with in sedentary subjects.^{195,197}

A threshold of intensity is probably required to achieve benefit, although the exact value is not known and may vary from one person to another. Although a threshold cannot be defined from available information, much of the exercise described in published reports that is associated with good health is at least moderate in intensity, such as brisk walking. Thus, it seems that beneficial exercise does not need to be of high intensity; the total amount of activity is more important for health than the performance of high-intensity exercise. Although somewhat greater benefits may accrue from vigorous exercise, more orthopedic injuries and higher dropout rates are associated with high-intensity exercise compared with low- to moderate-intensity programs.¹⁹⁸ This is not to suggest, however, that better guidelines could not reduce the risk of vigorous exercise, providing an even greater overall benefit. Hence, current recommendations are directed toward minimizing risk and maximizing benefit.

Occupational Activity

Early studies of occupational activity suggest that it can also provide protection from CAD.^{199,200} Although standing had no protective value, studies revealed that individuals who walked for long periods of time (such as postal employees) and those who engaged in heavy activity (longshoremen) obtained protection. Unless individuals walk for one hour or more each day as a part of their occupation (low to moderate intensity), they should supplement that activity with leisuretime exercise. As for heavy occupational activity, the level of

TABLE 8. Ener	gy Requirements	of Selected	Daily	Activities*
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Activities	METs
Leisure	
Mild	
Billiards	2.4
Canoeing (leisurely)	2.5
Dancing (ballroom)	2.9
Golf (with cart)	2.5
Horseback riding (walking)	2.3
Playing a musical instrument	
Accordion	1.8
Cello	2.3
Flute	2.0
Piano	2.3
Violin	2.5
Volleyball (noncompetitive)	2.9
Walking (2 mph)	2.5
Moderate	
Calisthenics (no weight)	4.0
Cycling (leisurely)	3.5
Golf (without cart)	4.4
Swimming (slow)	4.5
Walking (3 mph)	3.3
Walking (4 mph)	4.5
Vigorous	
Chopping wood	4.9
Climbing hills (no load)	6.9
Climbing hills (5 kg load)	7.4
Cycling (moderately)	5.7
Dancing	
Aerobic or ballet	6.0
Ballroom (fast) or square	5.5
Jogging (10 min mile)	10.2
Rope skipping	12.0
Skating	
Ice	5.5
Roller	6.5
Skiing (water or downhill)	6.8
Squash	12.1
Surfing	6.0
Swimming	7.0
Tennis (doubles)	5.0
Walking (5 mph)	8.0
Activities of daily living	
Gardening (no lifting)	4.4
Household tasks, moderate effort	3.5
Lifting items continuously	4.0
Loading/unloading car	3.0
Lying quietly	1.0
Mopping	3.5
Mowing lawn (power mower)	4.5

Activities	METs
Raking lawn	4.0
Riding in a vehicle	1.0
Sitting; light activity	1.5
Taking out trash	3.0
Vacuuming	3.5
Walking the dog	3.0
Walking from house to car or bus	2.5
Watering plants	2.5

*These activities can often be done at variable intensities, assuming that the intensity is not excessive and that the courses are flat (no hills) unless so specified. Categories are based on experience or tolerance; if an activity is perceived to be more than indicated, it should be judged accordingly.

MET indicates metabolic equivalent or a unit of sitting, resting oxygen uptake.

physical effort required is rarely achieved in the work setting. An operational definition of heavy occupational activity is a job that requires lifting loads of \geq 20 pounds at least once an hour throughout the day or constantly moving loads of any size from one place to another without mechanized transportation.

Leisure-Time Activity

Significant health benefits can be obtained by including a moderate amount of physical activity (eg, 30 minutes per day of brisk walking or raking leaves or 45 minutes of recreational games such as volleyball or tennis) on most, if not all, days of the week.¹²³ Leisure-time activity to achieve health benefits should aim for a minimum total of 700 to 1000 kcal/week.^{124,201}

Risks of Exercise

Exercise has both risks and benefits, and the challenge to the physician and other healthcare professionals is to provide guidelines that minimize risks and maximize benefits. Although many factors affect the risk of exercise, 3 of the most important are age, presence of heart disease, and intensity of exercise. Screening procedures can be used that identify an individual who is at risk for an exercise-related cardiac event, which may be helpful in reducing these occurrences.

The results of selected studies reporting the risks of sudden cardiac arrest during exercise training are summarized in Table 9. These studies indicate that the risk of sudden cardiac death during vigorous exercise is low, even in those persons with cardiac disease. However, because these were not randomized controlled trials, the contribution of all potential variables to sudden cardiac arrest or death cannot be determined. Nonetheless, it is generally believed that the benefits of exercise greatly exceed the risks; thus, individuals should be encouraged to exercise prudently.

Sudden Cardiac Death

Sudden cardiac death is rare in apparently healthy individuals. In individuals under the age of 40 years, sudden cardiac death is usually attributed to congenital heart disease, whereas CAD is a more likely cause for those over age 40.

Study	Activity		Supervision	Sudden Cardiac Arrests, Events per 100 000 Person-hours	
In the general population/those without	ıt known heart disease				
Vuori et al ²⁸⁶	Cross-country skiing	None	None	1/600 000	
Gibbons et al ²⁸⁷	Jogging, swimming, tennis	None	None	1/375 000	
Thompson et al ²⁸⁸	Jogging	None	None	1/396 000	
Vander ²⁸⁹	Jogging, court games	None	None	1/888 000	
Average				1/565 000	
Individuals with known heart disease					
Fletcher and Cantwell ²⁹⁰	Jogging	Intermittent	Present	1/6000	
Leach et al ²⁹¹	Jogging	Intermittent		1/12 000	
Mead et al ²⁹²	Jogging	Intermittent	Present	1/6000	
Hartley et al ¹³⁴	Jogging	Intermittent	Present	1/6000	
Hossack and Hartwig ²⁹³	Jogging	None	Present	1/65 185	
Haskell ²⁰²	Mixed	Intermittent	Present	1/22 028	
Van Camp and Peterson ²⁰³	Mixed	Continuous	Present	1/117 333	
Hartley*	Mixed	Continuous	Present	1/98 717	
Van Camp*	Mixed	Intermittent	Present	1/121 955	
Hartley*	Bicycling, walking	Intermittent	None	1/70 000	
Fletcher*	Mixed	Intermittent	Present	0/70 200	
Franklin et al ²⁰⁹	Mixed	Continuous	Present	1/146 127	
Average				1/61 795	

TABLE 9. Risk of Sudden Cardiac Arrest During Exercise Training

*Unpublished data.

Individuals with cardiac disease seem to be at an increased risk for sudden cardiac arrest during vigorous exercise (such as jogging) than are healthy individuals.²⁰²⁻²⁰⁴ However, with judicious programs, activity is clearly beneficial in lowering mortality in groups that exercise compared with sedentary groups.151,205,206 Most recently, the incidence of major cardiovascular complications during outpatient cardiac exercise programs has been estimated to be 1 in 60 000 participanthours.207-209 The type and intensity of activity and the use of monitoring apparently affect the incidence of sudden cardiac arrest. Table 9 shows that in cardiac subjects, incidence is lowest during activities that are largely controlled, such as walking, cycling, or treadmill walking. Table 9 also suggests that activities performed with continuous ECG monitoring have the lowest rates of sudden cardiac arrest compared with those that are unmonitored or only intermittently monitored.

Unfortunately, these studies do not answer questions regarding the relative contributions of various other factors to sudden cardiac arrest. These studies strongly suggest, however, that the incidence of sudden cardiac arrest across a variety of activities, with the exception of jogging, is similar to that expected by chance alone. In subjects with heart disease, jogging seems to be associated with a greater incidence of sudden cardiac arrest compared with other activities. This is probably related to exercise intensity. Jogging at even the slowest pace may generate a $\dot{V}o_2$ that exceeds 80% of maximum for many untrained individuals.

Myocardial Infarction

MI is another risk associated with participation in exercise. It has been reported that MI during exercise is 7 times more

likely to occur than sudden cardiac death. Exercise can be a potent trigger of MI. Approximately 4% to 20% of MIs occur during or soon after exertion.210-212 Physical exertion at a level of ≥ 6 METs has been reported within 1 hour of acute MI in 4% to 7% of patients. However, the adjusted relative risk has been found to be greater in persons who do not regularly participate in physical activity.210,211 Among sedentary persons, the relative risk of MI during exercise was 107 times that of baseline, whereas among individuals who regularly exercise 5 times per week, the relative risk of infarction during exercise is only 2.4 times greater than that of baseline.²¹⁰ This inverse relationship between regular physical activity and MI is of clinical importance because healthcare providers must consider a subject's functional capacity when considering the risk/benefit ratio of exercise. It is clear that the least active subjects are at greatest risk for MI during exercise and that both leisure-time physical activity and cardiorespiratory fitness have a strong inverse relationship with the risk of acute MI during exercise.²¹³

Musculoskeletal Injuries

Musculoskeletal injuries are common and include direct injuries such as bruises, sprains, and strains, and indirect problems such as arthritis and back pain. Low-impact exercises (walking, cycling, and swimming) cause less stress on bones and joints, whereas high-impact exercises (running and aerobic dancing) cause repeated impact on the knees, ankles, and feet. Studies indicate that the intensity and nature of impact of physical activity are the 2 most important factors in determining the frequency of injuries.

	•	•	
Frequency	Intensity	Duration	Modality
Endurance training			
3–5 days/week	50%–70% max HR	20-60 min	Lower extremity: walking, jogging/running, stairclimber
	40%–60% $\dot{V}o_{2\mbox{ max}}$ or HRR		Upper extremity: arm ergometry
			Combined: rowing, cross-country ski machines, combined arm/leg cycling, swimming, aerobics
Resistance training			
2–3 days/week	1–3 sets of 8–15 RM for each muscle group		Lower extremity: leg extensions, leg curls, leg press, adductor/abductor
			Upper extremity: biceps curl, triceps extension, bench/overhead press, lateral pull-down/raises, bench-over/seated row

TABLE 10.	Exercise Prescription	for Endurance	and Resistance	Training
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Modalities listed above are not all-inclusive.

HR indicates heart rate; max, maximum; HRR, peak minus rest heart rate multiplied by percent intensity plus rest heart rate; RM, maximum number of times a load can be lifted before fatigue. Maximum heart rate equals 220 minus age or peak heart rate on exercise test.

Adapted from Shephard RJ, Balady GJ. Exercise as cardiovascular therapy. *Circulation*. 1999;99:963–972.

Pre-Exercise Training Medical Evaluation and Exercise Prescription

The following pre-exercise screening procedures and activity classifications (Tables 10 through 14) are presented as a means of beginning exercise with the lowest possible risk. They do not consider accompanying morbidities (eg, morbid obesity, severe pulmonary disease, or debilitating neurological or orthopedic conditions) that may necessitate closer supervision during training sessions. As the individual gains experience, the decision may be made to place the subject in another category.

Pre-Exercise Screening

Before initiating an exercise program, the following recommendations should be applied to all potential exercise participants:

- 1. A recent medical history and limited physical examination should be performed.
 - a. If the history or physical examination indicates significant cardiovascular disease, the person should be treated as noted in the section "Medical Evaluation and Exercise Prescription for Individuals With CAD." Examples of cardiovascular disease include previous MI, CABG, angina pectoris, valvular heart disease, heart failure, and congenital heart disease.
 - b. If the individual knows of no cardiovascular disease but has symptoms or signs that suggest the presence of significant disease or has major coronary risk factors, an exercise test is needed before beginning an exercise program. Further evaluation should follow accordingly. If an exercise test cannot be performed, activity should be limited as outlined in the next section or pharmacological testing with dobutamine combined with an imaging modality should be performed.
- 2. Age should be considered.
 - a. Among men <45 years and women <55 years without known or suspected cardiovascular disease, no further

cardiovascular workup is needed, provided the issues outlined in section 1 are normal.

- b. Among men ≥45 years and women ≥55 years, particularly those with diabetes or 2 other risk factors for cardiovascular disease, the following should occur:
 - (1) An exercise test is recommended if vigorous exercise is planned. If the test is normal, no further restrictions are needed, although diabetics require special consideration. If the test is abnormal, further workup should follow accordingly and, for the purposes of exercise, the individual should be managed as if he or she has CAD.
 - (2) If the individual chooses not to undergo an exercise test, he or she should follow the activity guidelines outlined in Table 12.
- 3. Setting: If the individual presents to a health/fitness facility as the initial step toward beginning an exercise program, screening procedures should take place as detailed in the "Recommendations for Screening, Staffing, and Emergency Policies at Health/Fitness Facilities."²¹⁴ This involves the use of screening questionnaires such as the AHA/ACSM Preparticipation Screening Questionnaire.²¹⁴ These will prompt referral for medical evaluation by a healthcare professional when indicated.

Classification for Exercise Risk

After the medical evaluation is complete, subjects can be classified by risk on the basis of their characteristics. This classification is provided in detail in Tables 11 through 14, which are used to determine the need for subsequent supervision and the level of monitoring required.

Medical Evaluation and Exercise Prescription for Apparently Healthy Individuals

Although individuals may seem healthy, medical evaluation is important because of the potential for underlying medical problems, particularly those of a cardiovascular nature. In the healthcare setting, the evaluation should include a review of the individual's medical history and any current symptoms,

TABLE 11. Risk Classification for Exercise Training: Class A: Apparently Healthy Individuals

This classification includes:

- Children, adolescents, men <45 years, and women <55 years who have no symptoms or known presence of heart disease or major coronary risk factors.
- Men ≥45 years and women ≥55 years who have no symptoms or known presence of heart disease and with <2 major cardiovascular risk factors.
- Men ≥45 years and women ≥55 years who have no symptoms or known presence of heart disease and with ≥2 major cardiovascular risk factors.

Activity guidelines: No restrictions other than basic guidelines.

Supervision required: None*.

ECG and blood pressure monitoring: Not required.

*It is suggested that persons classified as Class A-2 and particularly Class A-3 undergo a medical examination and possibly a medically supervised exercise test before engaging in vigorous exercise.

limited physical examination, and consideration of an exercise test. In the health/fitness facility setting, the initial evaluation will primarily be the use of a screening questionnaire, which may prompt referral to a healthcare provider for further work-up.

Medical History

Of particular interest are data in the history that indicate unsupervised exercise may be hazardous. This includes CAD, significant valvular heart disease, heart failure, and congenital heart disease. If any of these heart conditions are present, the individual should follow the guidelines for individuals with heart disease in the next section. Persons taking cardiovascular medications should also follow the guidelines found in the next section. Obesity and neuromuscular disease tend to increase the risk of orthopedic injury and thus would suggest the use of lower intensity, low-impact exercise of longer duration in such persons (see subsequent sections).

Symptoms

Symptoms suggesting cardiovascular or pulmonary disease should be evaluated to exclude the presence of such disease. These include chest discomfort, dizziness, shortness of breath (at rest or with activities of daily living), and leg discomfort consistent with claudication.

Physical Examination

Hypertension requires assessment and management. Murmurs or sounds suggesting significant valvular heart disease or other signs of cardiac disease (eg, heart failure) should be regarded as indicating the presence of cardiovascular disease until proven otherwise.

Detection of Occult Disease

One of the most difficult challenges a physician may undertake is the detection of occult CAD. It is well known that individuals can have significant CAD in the absence of symptoms or signs and in the presence of a normal ECG and a normal exercise test. However, in the asymptomatic patient in whom CAD is strongly suspected, an exercise test may be useful in further evaluation.

TABLE 12. Risk Classification for Exercise Training: Class B: Presence of Known, Stable Cardiovascular Disease With Low Risk for Complications With Vigorous Exercise, but Slightly Greater Than for Apparently Healthy Individuals

This classification includes individuals with any of the following diagnoses:

- 1. CAD (MI, CABG, PTCA, angina pectoris, abnormal exercise test, and abnormal coronary angiograms) whose condition is stable and who have the clinical characteristics outlined below
- Valvular heart disease, excluding severe valvular stenosis or regurgitation with the clinical characteristics as outlined below
- Congenital heart disease; risk stratification for patients with congenital heart disease should be guided by the 27th Bethesda Conference recommendations¹⁴⁵
- Cardiomyopathy: ejection fraction ≤30%; includes stable patients with heart failure with clinical characteristics as outlined below but not hypertrophic cardiomyopathy or recent myocarditis
- 5. Exercise test abnormalities that do not meet any of the high risk criteria outlined in class C below

Clinical characteristics (must include all of the following)

- 1. New York Heart Association class 1 or 2
- 2. Exercise capacity \leq 6 METs
- 3. No evidence of congestive heart failure
- 4. No evidence of myocardial ischemia or angina at rest or on the exercise test at or below 6 METs
- 5. Appropriate rise in systolic blood pressure during exercise
- 6. Absence of sustained or nonsustained ventricular tachycardia at rest or with exercise
- 7. Ability to satisfactorily self-monitor intensity of activity

Activity guidelines: Activity should be individualized, with exercise prescription provided by qualified individuals and approved by primary healthcare provider.

Supervision required: Medical supervision during initial prescription session is beneficial.

Supervision by appropriate trained nonmedical personnel for other exercise sessions should occur until the individual understands how to monitor his or her activity. Medical personnel should be trained and certified in Advanced Cardiac Life Support. Nonmedical personnel should be trained and certified in Basic Life Support (which includes cardiopulmonary resuscitation).

ECG and blood pressure monitoring: Useful during the early prescription phase of training, usually 6 to 12 sessions.

Exercise Training Techniques

Training should consist of periods of warm-up and cooldown, endurance exercise, flexibility exercise, and resistance training (Table 10). Such activities are performed to reduce the risk of injury or cardiovascular events associated with sudden onset of activity, increase functional capacity and muscular strength, improve the ability to sustain activities of daily living, and promote personal independence and positive self image.

Warm-Up and Cool-Down

Exercising at a low intensity for 5 to 10 minutes before (warm-up) and after (cool-down) the training session is a routine recommendation. Such activities help stretch and warm up muscles and ligaments in preparation for the activity session. The cool-down period also prevents hypotension, which may occur with the sudden cessation of exercise.²¹⁵

TABLE 13.Risk Classification for Exercise Training: Class C:Those at Moderate-to-High Risk for Cardiac ComplicationsDuring Exercise and/or Unable to Self-Regulate Activity or toUnderstand Recommended Activity Level

This classification includes individuals with any of the following diagnoses:

- 1. CAD with the clinical characteristics outlined below.
- Valvular heart disease, excluding severe valvular stenosis or regurgitation with the clinical characteristics as outlined below.
- Congenital heart disease; risk stratification for patients with congenital heart disease should be guided by the 27th Bethesda Conference recommendations.¹⁴⁵
- Cardiomyopathy: ejection fraction <30%; includes stable patients with heart failure with clinical characteristics as outlined below but not hypertrophic cardiomyopathy or recent myocarditis.
- 5. Complex ventricular arrhythmias not well controlled.
- Clinical characteristics (any of the following):
 - 1. NYHA class 3 or 4.
 - 2. Exercise test results
 - Exercise capacity <6 METs
 - Angina or ischemic ST depression at a workload <6 METs
 - Fall in systolic blood pressure below resting levels during exercise
 - Nonsustained ventricular tachycardia with exercise
 - Previous episode of primary cardiac arrest (ie, cardiac arrest that did not occur in the presence of an acute myocardial infarction or during a cardiac procedure).
 - 4. A medical problem that the physician believes may be life-threatening

Activity guidelines: Activity should be individualized, with exercise prescription provided by qualified individuals and approved by primary healthcare provider

Supervision: Medical supervision during all exercise sessions until safety is established.

ECG and blood pressure monitoring: Continuous during exercise sessions until safety is established, usually \geq 12 sessions.

NYHA indicates New York Heart Association.

*Class C patients who have successfully completed a series of supervised exercise sessions may be reclassified to Class B providing that the safety of exercise at the prescribed intensity is satisfactorily established by appropriate medical personnel and that the patient has demonstrated the ability to self-monitor.

Endurance Exercise

Activities that cause the greatest increase in $\dot{V}o_{2 \text{ max}}$ have certain characteristics which, when present, are said to qualify the exercise as endurance (cardiovascular) activities. These characteristics include dynamic exercise, alternately contracting and relaxing the muscles (as opposed to isometric or resistance exercise), in large muscle groups, as in walking or running. Exercise should be performed 3 to 6 times per week for a minimum of 30 minutes per session at a minimum intensity of 40% to 60% $\dot{V}o_{2 max,}$ and up to 85% to 90% $\dot{V}o_{2}$ max for those who have appropriately progressed to this level. In addition to brisk walking and running, other examples of endurance or cardiovascular activities are swimming, cycling, stair-stepping, and cross-country skiing. A useful approach to activity prescription is to identify the desirable rating of perceived exertion and instruct individuals to adhere to that intensity. A suggested rating of perceived exertion for most healthy individuals is 12 to 16 ("somewhat hard to hard") on

TABLE 14. Risk Classification for Exercise Training: Class D: Unstable Disease With Activity Restriction*

This classification includes individuals with any of the following:

- 1. Unstable ischemia.
- 2. Severe and symptomatic valvular stenosis or regurgitation.
- Congenital heart disease; criteria for risk that would prohibit exercise conditioning in patients with congenital heart disease should be guided by the 27th Bethesda Conference recommendations.¹⁴⁵
- 4. Heart failure that is not compensated.
- 5. Uncontrolled arrhythmias.
- 6. Other medical conditions that could be aggravated by exercise.

Activity guidelines: No activity is recommended for conditioning purposes. Attention should be directed to treating the patient and restoring the patient to Class C or better. Daily activities must be prescribed on the basis of individual assessment by the patient's personal physician.

*Exercise for conditioning purposes is not recommended.

a Borg scale of 6 to 20, an approach that is both effective and acceptable.²¹⁶ See Table 4 for more details on rating of perceived exertion.

Flexibility Exercise

Properly selected stretching exercises are helpful for promoting flexibility. Flexibility activities should focus on improving range of motion in a joint or series of joints. Particular attention should be focused on the lower back and posterior thigh regions in an attempt to reduce the risk of chronic lower back pain.¹⁹⁰

Resistance Training

Resistance exercise training which involves activities that use low or moderate repetition movements against resistance has been accepted as a primary component of a comprehensive exercise program both for apparently healthy and, with appropriate screening and precautions, for subjects with cardiovascular disease.217 Although the effect of resistance exercise is less than traditional endurance exercise regarding its influence on risk factor modification, the increase in strength and potential for increased muscle mass may improve the individual's ability to become more physically active and raise the basal metabolic rate and may, in older persons, improve the ability to perform activities of daily living. Persons initiating a resistance training program should be carefully screened for both cardiovascular limitations and preexisting orthopedic and musculoskeletal problems. In addition, individuals should be provided with careful recommendations regarding the specific components of the resistance training program, including proper technique, number and types of exercises, and safety precautions.

Programs including a single set of 8 to 10 different exercises (eg, chest press, shoulder press, triceps extension, biceps curl, pull-down, lower back extension, abdominal crunch/curl-up, quadriceps extension or leg press, and leg curls/calf raise) that train the major muscle groups, when performed 2 to 3 days per week, will elicit favorable adaptation and improvement (or maintenance thereof). Although greater frequencies of training and more sets may be used, the additional gains among those in adult fitness programs are usually small.^{190,218} To achieve a balanced

increase in both muscular strength and endurance, a repetition range of 8 to 12 is recommended for healthy participants <50 to 60 years of age and a range of 10 to 15 repetitions at a lower relative resistance is recommended for cardiac patients and healthy participants \geq 50 to 60 years of age.¹⁹⁰ The reason for the increased repetition range at a lower relative effort for older or "more frail" subjects is for injury prevention. The single greatest cause of musculoskeletal injury with resistance training is a previous injury. Also, higher intensity efforts (fewer repetitions with heavier weights) can have adverse effects on the knee (leg extension) and shoulder (rotator cuff) areas. For detailed recommendations regarding resistance training, see the AHA Advisory, "Resistance Exercise in Individuals With and Without Cardiovascular Disease."²¹⁷

General Guidelines for Individual Exercise Programming

- 1. Exercise only when feeling physically well. Wait until symptoms and signs of a "cold or the flu" (including fever) have been absent ≥ 2 days before resuming activity.
- 2. Do not exercise vigorously soon after eating. Wait at least 2 hours. Eating increases the blood flow requirements of the intestinal tract. During vigorous exercise, the demand of the muscles for blood may exceed the ability of the circulation to supply both the bowel and the muscles, depriving organs of blood, resulting in cramps, nausea, or faintness.
- 3. Drink fluids. Water is generally the replacement fluid of choice for most individuals. Specific recommendations regarding the amount of fluid needed to replace that lost in sweat through exercise are difficult to provide, because this will vary depending on the training intensity and duration, environmental conditions, and health status of the individual. In general, water should be taken before, during, and after any moderate-to-vigorous intensity exercise >30 minutes in duration. Disease and medications may increase susceptibility to heat illness and fluid loss. Elderly persons, obese individuals, and those taking diuretics and other antihypertensive medications are particularly prone to heat illness. Alcohol consumption can precipitate heat stress due to its effects on vasomotor tone and volume status.
- 4. Adjust exercise to the weather. Exercise should be adjusted to environmental conditions. Special precautions are necessary when exercising in hot weather. It is difficult to define when it is too hot to exercise because air temperature is greatly influenced by humidity and air movement (wind), which are not easy to measure. The following guidelines are recommended for a noncompetitive workout: if air temperature is >70°F, slow the pace, be alert for signs of heat injury, and drink adequate fluids to maintain hydration. A good rule to follow is to exercise at the usual workout pace (rating of perceived exertion, 12 to 16), which may be a slower pace or lower work intensity because of environmental conditions. Acclimatization to moderate levels of heat is gradual and

may require 12 to 14 days. Accommodation to extreme heat never occurs. Symptoms or signs of heat injury may be varied at the onset; hence, any symptom should be regarded as evidence of heat overload. The following indications of heat stress are particularly likely to occur: headache, dizziness, faintness, nausea, coolness, cramps, and palpitations. If any of these symptoms are present, stop exercising immediately and go to a cooler environment. If the air temperature is $>80^\circ$ F, exercise in the early morning or late afternoon to avoid the heat. Air-conditioned shopping malls are popular for walking. Exercise is better tolerated if humidity is low and a breeze is present. Exercise in the heat causes excessive fluid loss; therefore, adequate fluid intake is important before, during, and after each session.

- 5. Slow down for hills. When ascending hills, decrease speed to avoid overexertion. Again, a useful guide is to maintain the same rating of perceived exertion as in a usual workout.
- 6. Wear proper clothing and shoes. Dress in loose-fitting, comfortable clothes made of porous material appropriate for the weather. Use sweat suits only for warmth. Never use exercise clothing made of rubberized, nonporous material. In direct sunlight, wear light-colored clothing and a cap. Wear shoes designed for exercise (eg, walking or jogging shoes).
- 7. Understand personal limitations. Everyone should have periodic medical evaluations. When under a physician's care, ask if there are limitations.
- 8. Select appropriate exercises. Endurance exercises should be a major component of activities. It is recommended that any individual >40 years should take special care to avoid high-impact activities.^{219,220} If such activities are chosen, they should be initiated at low levels and increased slowly. A day of rest between exercise periods permits the body to gradually adapt to stresses and strains. More attention should also be given to warm-up and cool-down periods with stretching, low-level calisthenics, and low-level endurance exercises. In general, fast walking is a well-tolerated, low-impact exercise that provides excellent results. Swimming, stair climbing, rowing, and stationary cycling may also be appropriate.
- 9. Be alert for symptoms. If the following symptoms occur, obtain medical consultation before continuing exercise. Although any symptom should be clarified, these are particularly important:
 - a. Discomfort in the upper body, including the chest, arm, neck, or jaw during exercise. The discomfort may be of any intensity and may be present as an aching, burning, tightness, or sensation of fullness.
 - b. Faintness accompanying the exercise. Sometimes brief light-headedness may occur after an unusually vigorous bout of exercise or a limited cool-down period. This condition generally does not indicate heart disease and may be managed by exercising at a lower intensity with a gradual cool-down at the end of the session. If fainting or a feeling of faintness occurs during exercise, discontinue the activity until after medical evaluation.

- c. Shortness of breath during exercise. During exercise, the rate and depth of breathing should increase but should not be uncomfortable. A useful guideline is that an ordinary conversation should not be an effort, wheezing should not develop, or not more than 5 minutes should be required for recovery.
- d. Discomfort in bones and joints either during or after exercise. There may be slight muscle soreness when beginning exercise, but if back or joint pain develops, discontinue exercise until after medical evaluation.
- 10. Watch for the following signs of over-exercising:
 - a. Inability to finish. Training sessions should be completed with reserve.
 - b. Inability to converse during the activity. Breathing increases during exercise but should not be uncomfortable. When a conversation cannot be conducted during exercise because of difficulty breathing, the conditioning activity is too intense.
 - c. Faintness or nausea after exercise. A feeling of faintness after exercise may occur if the activity is too intense or has been stopped too abruptly. In any event, decrease the intensity of the workout and prolong the cool-down period.
 - d. Chronic fatigue. During the remainder of the day or evening after exercise, an individual should feel stimulated, not tired. If fatigue persists during the day, intensity and/or duration of the workout should be decreased.
 - e. Sleeplessness. If unable to sleep well despite feelings of fatigue, the amount of activity should be decreased until symptoms subside. Insomnia may occur during distance training. A proper training program should make it easier, not more difficult, to have adequate sleep.
 - f. Aches and pains in the joints. Although there may be some muscle discomfort, joints should not hurt or feel stiff. Check exercise procedures, particularly stretching and warm-up exercises, to ensure that the proper technique is being used. Muscle cramping and back discomfort may also indicate poor technique. If symptoms persist, check with a physician before continuing.
- 11. Start slowly and progress gradually. Allow time to adapt.

Medical Evaluation and Exercise Prescription for Individuals With CAD

Exercise training is useful in the treatment of CAD subjects because the physiological changes that occur lessen myocardial ischemia at rest and during submaximal exercise. Physical activity is also associated with a reduction of the risk for the development or progression of CAD.^{151,205,206} However, certain precautions and guidelines are necessary to avoid cardiac events. In this section, the basis for activity programs for subjects with CAD and specific considerations are discussed, including recommendations for special populations to reduce cardiac events associated with activity.

Inpatients

While the patient remains in the inpatient setting, walking is recommended as the major mode of exercise unless the

individual can attend classes where other monitored activities can be provided. Walking near the bedside and to the bathroom are permitted initially. If symptoms develop, the patient can easily return to bed. Walking should start slowly and gradually increase as tolerated until 5 to 10 minutes of continuous movement has been achieved. Active but nonresistance range-of-motion exercise of the upper extremities is also well tolerated early after MI or CABG as long as the activities do not stress or impair the healing of incisions in CABG patients. Initial activities should be monitored, and symptoms, rating of perceived exertion, heart rate, and blood pressure should be recorded. When tolerance is documented, the activity can be performed without supervision. The basis for exercise within the period of hospitalization is avoidance of the deleterious effects of bed rest. When patients are stable as measured by ECG, vital signs, and symptoms, they can begin walking. Although this activity is well tolerated and safe, certain precautions are recommended.

Outpatients

In the outpatient setting, large-muscle group activities should be performed for at least 30 minutes, preceded by warm-up and followed by cool-down, at least 3 times weekly. The intensity of exercise should be designated by exercise prescription. Moderately intense activity (\approx 40% to 60% of $\dot{V}o_{2 \text{ max}}$) is effective for increasing both submaximal and maximal endurance if performed on a regular basis, and it is associated with a low incidence of sudden cardiac arrest. Follow-up supervised group sessions are recommended to enhance the educational process, to ensure that the participant is tolerating the program, to confirm that progress is occurring, and to provide the appropriate level of medical supervision in high-risk patients. Long-term follow-up is recommended to monitor compliance and to ensure that the program is being followed properly.

Cardiac rehabilitation sessions are typically serial in nature and emphasize patient education and risk factor modification. Core components of such programs include the following: nutrition counseling, medical assessment, lipid and weight management, smoking cessation, diabetic evaluation and monitoring, psychosocial assessment and intervention, activity counseling, and exercise training. The exercise prescription should include emphasis on appropriate levels of frequency, intensity, duration, mode, and progression. Activity is supervised during these sessions to ensure safety and may include ECG monitoring when deemed necessary for patient safety. The number of monitored sessions depends on individual patient characteristics, as outlined in Tables 12 and 13.

Exercise testing is an integral component of the rehabilitative process because it provides for the establishment of appropriate specific safety precautions, target exercise training heart rates, and initial levels of exercise training work rates. Exercise testing is also important in the risk stratification process, as outlined in Tables 12 and 13. Exercise tests should be performed on all cardiac patients entering an exercise training program and should be repeated at least annually or at any time the patient's condition warrants. Additional evaluation of the patient's cardiac status (echocardiography, nuclear studies, or coronary angiography) may also be needed before entry into an exercise program. Training intensity can be ascertained by an exercise test. If a test is not performed, the exercise prescription must be more conservative and rely on the patient's rating of perceived exertion (see below), along with signs or symptoms to provide the upper limits of activity. Patients should always avoid activity that elicits inappropriate signs or symptoms.

Steps in This Process of Prescribing Exercise Include:

- 1. The target heart rate for moderate intensity exercise may be considered as 40% to 60% of heart rate reserve, as determined from the exercise test: [(maximal heart $rate minus resting heart rate) \times (40\% to 60\%)]$ +resting heart rate. This heart rate range can be used for the initial prescription of many types of dynamic exercise and can be increased to 85% (high intensity) if tolerated.
- 2. Activities can be prescribed by designating the target workload that achieves the training heart rate after performance of 3 to 6 minutes at that workload (steady state). It may be expressed as watts on an ergometer, speed/grade on a treadmill, or in METs.
- 3. Exercise intensity may then be assessed using the calculated target heart rate based on the equation above as a guide to the counted heart rate (manually or with a cardiotachometer). Cardiotachometers are widely available and are reasonably accurate for low-to-moderate intensity exercise. Supervision assures that the instructions are understood and that the activity is well tolerated.
- 4. Individuals can also judge the intensity of exercise as the rating of perceived exertion, which can be equated to desirable heart rate range during supervised exercise and during other activities. The original scale is a 15-grade category scale ranging from 6 to 20, with a verbal description at every odd number (see Tables 4 and 7). The following rating of perceived exertion values should be followed:
 - a. <12 is light, <40% of maximal capacity (ie, $\dot{V}O_{2 max}$)
 - b. 12 to 13 is somewhat hard (moderate), 40% to 60% of maximal capacity
 - c. 14 to 16 is hard (heavy), 60% to 85% of maximal capacity

Activities can progress as tolerance is demonstrated. The appropriate initial intensity of training is 40% to 60% of $\dot{V}O_{2\,max}$ or a rating of perceived exertion of 12 to 13 on a scale of 6 to 20. After safe activity levels have been established, duration may be increased as appropriate; later, intensity may be increased as heart rate response to exercise decreases with conditioning.

Exercise Prescription in the Presence of Ischemia

The exercise test results are the basis for the exercise prescription in patients with ischemia or arrhythmias. Myocardial ischemia manifesting as horizontal or downsloping ST segment depression and/or angina pectoris requires careful review when generating the exercise prescription. The exercise prescription is developed using the previously described methodology (40% to 60% of $\dot{V}o_{2 \text{ max}}$), but with the designated heart rate and work rate below the identified threshold of ischemia (ie, angina and/or ≥ 1 mm ischemic ST segment depression on the exercise test). In general, the heart rate prescription should be a minimum of 10 beats/min below the heart rate at which the abnormality occurs.

Special Considerations in Prescribing Exercise

All individuals must be carefully screened for medical status before beginning an exercise program. They must also have adequate instruction and follow-up to lessen the likelihood of complications. Furthermore, special considerations must be made in patients with potential limitations at program entrance. The principles of surveillance for safety and expectations for improvement are largely intended for subjects with CAD but may also apply to other subjects with a variety of noncoronary cardiac, vascular, and pulmonary diseases and other conditions, as will be discussed. Safety is the major reason for establishing special guidelines for subjects with cardiovascular disease. These recommendations should be considered appropriate for any condition associated with a higher than normal risk for sudden cardiac arrest or MI during exercise.

The Elderly

Special considerations must be addressed when prescribing exercise for the elderly. In these subjects, maximal end-diastolic volume increases, whereas maximal heart rate, LV ejection fraction, and cardiac output are all lower than in younger individuals. In the presence of CAD, these factors may affect the cardiac response to a given exercise prescription. In addition, the extent of disease and increased potential for exercise-related myocardial ischemia and arrhythmias in this age group may increase the risk of adverse events. A critical factor in an elderly (>65 years) person's ability to function independently is mobility, the ability to move without assistance.186,221-226 The overall focus for exercise training should be to enhance health-related fitness components, while simultaneously assisting in the reduction of risk for various chronic diseases and improving overall quality of life. Considerable evidence exists that physical activity, both endurance and resistance-type exercise, can significantly improve these indices and provide for functional independence and overall well-being, especially in the older adult.

As with all other patients entering an exercise program, elderly persons should undergo a medical evaluation before initiating an exercise program. This assessment should include not only a "focused" physical examination but should also identify any psychosocial limitations to participation, which are prevalent in this age group.^{226–231} For older, apparently healthy persons desiring to participate in a lowto-moderate intensity activity such as walking, an exercise test may not be required. However, for more vigorous activities and for all cardiac patients, an exercise test should be performed. In addition, a determination of any dietary inadequacies that may be compounded by modest increases in caloric expenditure and a review of the individual's medication regimen for possible interactions with activity programs should be performed.²³²

Exercise prescription guidelines, as described previously in this document, are generally appropriate for older participants. As with younger persons, the combination of endurance and resistance exercise is best for achieving the health and fitness goals of the elderly.233-236 However, despite the fact that exercise recommendations are similar, some specific comments regarding intensity, frequency, duration, and mode of exercise for the elderly are required. The exercise capacity of the elderly, both before and after exercise training, is usually lower than that observed in younger persons.^{221,237} Furthermore, because many in this age group have been sedentary for years, specific muscle groups are often markedly deconditioned. In addition, musculoskeletal limitations, particularly arthritis, can be severely limiting. Thus, it is important to recommend activities that require low-level energy expenditure, particularly during the first few weeks (40% to 50% of $\dot{V}O_{2 \text{ max}}$) of the program, and prescribe mild increases at any time when progression of activity is made. In these instances, however, participants are encouraged to increase the frequency of exercise (for shorter duration), even to perhaps 3 or 4 times per day. Higher intensity exercise training must be recommended with caution in this age group because of the potential for musculoskeletal injury.

Like those whose exercise program intensity is significantly reduced, those persons whose exercise duration is limited (<15 minutes/session) because of physical or psychosocial limitations should also attempt to exercise more frequently. Conversely, a recommendation to lengthen the duration of activity as appropriate beyond 15 minutes and to as much as 45 to 60 minutes per session is valuable for increasing caloric expenditure, but in doing so, a lesser intensity should be used. This regimen is associated with the improvement of a number of risk factors, including obesity, lipid abnormalities, hypertension, and elevated blood glucose.

Many elderly persons have symptomatic concomitant medical and physical limitations (orthopedic, arthritic, and vascular) that may be exacerbated by weight-bearing exercise, especially higher impact activities such as jogging. Even walking, a light intensity exercise, may be difficult for the elderly person. Thus, even seemingly innocent activities should be carefully considered for potential adverse effects in this age group, especially when the activity requires individuals to bear their entire weight.

Resistance Training

Resistance training is generally safe in the elderly and can promote increases in and maintenance of muscular strength, neuromuscular coordination, and lean body mass while facilitating an enhanced quality of life.^{238,239} Many activities of daily living required for functional independence such as rising from a chair, climbing steps within the home, and lifting of household items require muscular strength more than muscular endurance. Increased muscle mass may also be helpful in increasing aerobic exercise tolerance in this population and, thus, should also contribute to functional capacity.

The process of increasing muscular strength begins for most elderly persons as they begin to exercise. Strength levels are often so reduced that even the aerobic exercise program will enhance strength. Conversely, debilitated patients may require resistance training before they can participate meaningfully in aerobic exercise training. However, further increases in strength will require the addition of some type of resistance component. Recommendations regarding these activities should follow the AHA's advisory on resistance training.²¹⁷ Any exercise program, especially resistance exercise, should be closely monitored for potential overuse injuries, particularly early in the program.²¹⁷ Systemic blood pressure may also increase more in response to resistance training compared with aerobic exercise and, therefore, blood pressure monitoring may be indicated in some individuals (Table 10).

A pre-exercise period of stretching and light activity involving the large muscle groups for 5 to 10 minutes is appropriate for most exercise programs involving the elderly. An extended cool-down period after physical activity is suggested because of an increase in the potential for postexercise hypotension, syncopal episodes, or arrhythmias during recovery. Increasing the older patient's range of motion and flexibility is also integral to the success of the exercise program. As a result of aging, sedentary lifestyle, and medical and physical limitations, the elderly often exhibit decreased flexibility and are thus encouraged to regularly practice range of motion and flexibility exercises. Including some flexibility exercise as part of the warm-up period is recommended, but the majority of the flexibility training program should be performed after the aerobic portion of the program, when muscles and joints have been appropriately "exercised."217 Flexibility exercise should be encouraged because increased flexibility will reduce the likelihood of injury associated with the exercise program and of injuries that may occur during activities of daily living.

The participant's footwear should be evaluated with emphasis on properly "fitted," comfortable, supportive shoes for exercise. Because of potential circulatory limitations, reduced support from the surrounding muscles, and degenerative changes in bones and joints that occur with aging, proper footwear is particularly important for the elderly. If floor exercise is used, the elderly may require exercise mats (even on carpeted surfaces) to avoid discomfort.

The thermoregulatory capacity in the elderly is of concern because some medications that these individuals may be taking (eg, β -blockers, phenothiazines) may adversely impact thermoregulation, whereas others (diuretics) increase the potential for dehydration with exercise in this age group. Loss of fluid during exercise can further reduce an already volumedependent cardiac output. Consequently, ample fluid intake before, during, and after exercise should be encouraged. The elderly should also be aware of symptoms of dehydration, including thirst and dizziness, particularly during hot or humid weather conditions.

Heart Failure

Exercise training has been shown to be effective in improving functional capacity in patients with impaired LV function.^{240,241} Because heart failure patients with abnormal exercise tolerance may have preserved hemodynamics in the presence of extreme deconditioning, it is quite appropriate to recommend exercise rehabilitation programs to this group of

patients. Currently, a growing body of research demonstrates that exercise training in patients with LV systolic dysfunction is beneficial. Accordingly, exercise activity is now recommended as a component of a comprehensive approach to the patient with heart failure.^{242,243}

Exercise training in patients with heart failure has been shown to reduce heart rate at rest and submaximal exercise and increase peak $\dot{V}O_2$. Although central hemodynamics have not consistently shown improvement, significant peripheral changes such as an increase in systemic AV O₂ difference, with improved leg blood flow and a reduction in arterial and venous lactate levels, have been reported.244-246 Neurohormonal abnormalities (prevalent in heart failure patients) have been shown to improve after training.²⁴⁶ Exercise training seems to favorably impact autonomic tone in patients with heart failure, leading to enhanced vagal tone supported by overall reductions in heart rate, increased heart rate variability, and declines in sympathetic nervous activity. Exercise training yields important changes in skeletal muscle fiber type and function, leading to enhanced oxidative capacity. This is demonstrated by improved endurance and is associated with a reduction in the ratio of inorganic phosphate to phosphocreatine, an indirect measure of improved oxidative capacity.247,248

The responses to exercise training in heart failure patients vary, although the majority of studies have demonstrated improvements in exercise capacity. Several factors may account for reported differences in the studies to date. The cause of heart failure, hemodynamic abnormalities, and peripheral limitations are heterogeneous in this patient group. Small subject groups and differences in the exercise prescription and medical course during the time of exercise training may influence the reported outcomes. Not all such patients will improve, and some may have an exacerbation of their condition while exercising. An increase in signs of mild-tomoderate hemodynamic compromise (using pulmonary capillary wedge pressure and cardiac output) with exercise training has been reported in heart failure patients with a peak $\dot{V}O_2 \leq 14 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, in contrast to those patients with a peak $\dot{V}o_2 > 14 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$.²⁴⁹ Furthermore, heart failure patients with a low peak Vo2 may also fail to demonstrate significant improvement in functional capacity after exercise training.250

In those with heart failure, a training program should be initiated at a low to moderate level (25% to 60% of $\dot{V}o_{2 max}$) of the exercise capacity, preferably measured using a metabolic exercise test. Careful supervision and monitoring are particularly important during the initial training period. Telemetry monitoring during these early sessions is also recommended. Patients may begin sessions similar to those of other cardiac patients but may be limited in duration of activity until their endurance improves. Resistance training in this patient group may be beneficial, but the safety and efficacy of this type of training have not yet been well established.

Heart Transplantation

Patients with cardiac transplantation have generally been inactive before the procedure and remain deconditioned after the operation. The denervated donor heart has altered physiological responses to exercise, which include both blunted chronotropic and inotropic responses that tend to limit exercise capacity.^{251–253} Nonetheless, several investigations have suggested that exercise training increases endurance capacity.254-257 Generally, patients may enter medically supervised outpatient exercise programs as soon as they are discharged from the hospital. Frequency of activity is dependent on physician direction and may be as little as one session per week initially, working up to at least 3 sessions per week. Because the heart rate in a denervated heart rises more slowly in response to exercise and may remain elevated longer after activity, it is more difficult to use heart rate to monitor exercise intensity. The rating of perceived exertion in combination with other descriptors of exercise tolerance such as workload can be particularly helpful with this patient group.²⁵⁸ Resistance training can be useful to offset the skeletal muscle loss and weakness due to corticosteroid use and general inactivity.

Surgical Incision After CABG

The extent of healing of surgical incisions from CABG can be the most limiting factor for exercise. Hence, the decision to begin activity is often deferred to the surgeons. Low-level activities are usually acceptable 24 to 48 hours after surgery. Chest and leg wounds usually require 4 to 6 weeks for healing. Upper body exercises that cause sternal tension should be avoided for up to 3 months after surgery.²¹⁷ In those patients who have undergone minimally invasive CABG without sternotomy, wound healing should be monitored. Such patients need less restriction of activity.

After Percutaneous Coronary Interventions

Because there are no specific studies that have evaluated the safety of exercise training within days after percutaneous coronary interventions, until such data are available, it is recommended that subjects begin or resume exercise no sooner than 5 to 7 days after the procedure. Care must be taken to assure that anginal symptoms are recorded and properly evaluated and that catheterization access sites are healed and stable. Exercise testing may be of considerable value in assessing new or different symptoms or in patients with incomplete revascularization (ie, those in whom not all stenotic lesions have been dilated).³

Pacemakers and Implantable Cardioverter Defibrillators

If performance during an exercise test is satisfactory, individuals with pacemakers have problems similar to those of other cardiac subjects. Although the paced rate of some pacemakers can be accelerated during exercise, some cannot. The type and settings of a pacemaker should be noted, and exercise should be prescribed accordingly. Physical activity intensities in fixed-rate pacemakers must be gauged by a method other than pulse counting, such as defining specific workloads that are initially $\approx 40\%$ to 60% of peak exercise capacity, as determined by the exercise test and by using the rating of perceived exertion. Exercise prescription for patients with defibrillators should be limited to a target heart rate that is at least 10 to 15 beats/min lower than the threshold discharge rate for the defibrillator.

Diabetes Mellitus

Patients with diabetes require special attention, especially if they are using exogenous insulin or oral hypoglycemic medications. Because they are prone to leg and foot wounds that may interfere with or be aggravated by exercise, initial medical evaluation should include an examination of the lower extremities. Patients should be advised to wear thick protective (preferably white cotton) socks and well-fitting supportive footwear during exercise. Patient history should include details regarding type of medication, timing and type of insulin used, and previous episodes of hypoglycemia. Patients should be counseled regarding the effect of exercise on blood glucose levels and the possibility of hypoglycemia, which may occur for several hours after the exercise session. Recognition and treatment of hypoglycemic episodes should be reviewed with diabetic patients.

Blood glucose levels should initially be obtained before and after exercise to provide an assessment of the individual's response to exercise. The type of insulin (long or short acting), time of injection, last meal, and intensity of exercise should all be recorded because each of these factors can contribute to variations in blood glucose levels after exercise. In addition, glucose recordings may provide evidence for a change in insulin prescription. Blood glucose levels <100 and >300 mg/dL should preclude exercise at that time.

Stroke Patients With Disabilities

The population of stroke subjects is increasing as our population ages. These stroke victims often have comorbidities of CAD and peripheral arterial disease. Functional impairments in this disabled group include paresis, paralysis, spasticity, and sensory perceptional dysfunction.²⁵⁹ Aerobic exercise training in the disabled stroke patient is safe^{260,261} and reduces the energy expenditure and cardiac demands of a designated activity.^{105,262} These subjects may perform a variety of aerobic activities; however, stationary cycle ergometry (arm, leg, and arm-leg) is most often used. Such activities can be modified to satisfy the needs of the individual. Limited data suggest¹⁰⁵ that LV ejection fraction improves after upper extremity training. Evidence also documents that reduction of risk of stroke in later life is conferred by exercise patterns in early years.^{263,264}

Hypertension

Exercise is recommended as a component of the initial treatment for as long as 12 months in patients with stage 1 hypertension (140 to 159/90 to 99 mm Hg) with no other coronary risk factors and no evidence of cardiovascular disease, and for as long as 6 months in those with one other risk factor, not including diabetes. For patients with diabetes or cardiovascular disease or those with stage 2 or 3 hypertension (\geq 160/100 mm Hg), drug therapy should be initiated concurrently with exercise and other lifestyle modification programs.²⁶⁵

A slight increase in systolic pressure may precede exercise training sessions due to anticipation and is generally not a

cause for concern. Incremental increases in systolic blood pressure during exercise are normal, although unusually high blood pressures (>190 mm Hg systolic), particularly during low-level activity, may warrant adjustment in medical therapy. A 10 to 15 mm Hg fall in blood pressure from resting levels during exercise is a cause for concern. Exercise must be discontinued in such instances, and the patient should be further evaluated before returning to training sessions.

Peripheral Arterial Disease

Most patients with this condition are limited by claudication during exercise that involves dynamic motion of calf and leg muscles. Details regarding exercise training in such patients can be found elsewhere.²⁵⁸ In general, exercises that promote conditioning (those not limited by claudication but that involve large muscle groups) should be combined with those that subsequently reduce claudication (eg, treadmill walking).

Special Medical Conditions

Acute Myocardial Ischemia

Individuals with unstable myocardial ischemia, as judged by anginal symptoms or a changing pattern in the ECG, should not exercise until the condition has been treated and stabilized.

Arrhythmias

Although there is some evidence that regular physical activity may be beneficial in subjects with arrhythmias, most studies have focused on benign arrhythmias. The occurrence of exercise-induced high-grade ventricular ectopy (\geq 3 sequential ventricular ectopic beats) at rest should be evaluated and/or treated before beginning an exercise program. In general, individuals with arrhythmias other than high-grade ventricular ectopy may exercise if they are asymptomatic and remain hemodynamically stable. Telemetry ECG monitoring during rehabilitative sessions may be helpful for adjusting antiarrhythmic therapy.

Systemic Infections

Acute systemic infections can be adversely affected by activity. Even individuals with chronic infections may benefit more from rest than exercise. However, as the infection responds to treatment, exercise can begin. For example, in the treatment of bronchitis, moderate exercise can begin when the individual has a normal temperature, normal white blood cell count, and negative cultures.

Endocarditis

Individuals with infective endocarditis should avoid exercise until the disease is stable. The contribution of physical activity to emboli is not known for certain, but low-tomoderate activity levels seem prudent until the course of antibiotics is completed.

Myocarditis

As with any infection, activity should be maintained at low levels until the individual has no signs of active inflammation. When such has subsided, exercise can be prescribed prudently, as previously outlined for patients with cardiovascular disease.

Thromboembolic Disease

Thrombophlebitis, arterial embolism, or pulmonary embolism should be treated with rest, even though factors that cause clot "dislodgment" are not clearly defined. Low-level walking or range-of-motion activity is probably safe as soon as the individual is in a stable treatment program and has had no recurrence of symptoms. A moderate exercise program can be started when the risk of recurrent events has stabilized.

Neuromuscular Diseases

Neuromuscular inflammation and injuries should be evaluated by a qualified healthcare professional to assess the appropriateness of exercise training and to determine the types of activities that are suitable.

New or Changing Symptoms

Chest Discomfort

Both the new occurrence and exacerbation of previous chest discomfort, whether typical angina pectoris or other forms of atypical chest discomfort, must be evaluated before initiating or continuing exercise.

Shortness of Breath

The occurrence of shortness of breath at rest may suggest pulmonary congestion, and appropriate assessment for cardiogenic pulmonary edema is needed. Some shortness of breath and fatigue may occur because of the deconditioning effect of bed rest after a cardiovascular event or surgery. Edema on the chest x-ray film, rales, or a third heart sound on examination will clarify the presence of significant pulmonary congestion.

Faintness, Dizziness, or Light-Headedness

These symptoms may occur after a prolonged period of bed rest or inactivity, and they can be due to a contracted blood volume or loss of postural reflexes caused by inactivity or surgery. Such individuals will often have an orthostatic fall in blood pressure that may be hazardous if left untreated. Cardiac arrhythmias may also lead to such symptoms and thus should also be considered as a potential cause.

Weakness and Fatigue

These complaints are common symptoms after a lengthy illness and need not necessarily be a concern. The sensation of fatigue will usually improve with time and conditioning. Weakness after cardiac surgery may occur because of low hemoglobin and may limit the early phases of cardiac rehabilitation. Restitution of hemoglobin to normal levels requires several weeks unless transfusions are administered. However, if these symptoms persist, additional evaluation and review of medication regimens should be done.

Types of Exercise Programming and Monitoring

Levels of supervision and monitoring must be considered on the basis of the type of patient, staff, facility, and resources. Details regarding administration and programming of cardiac rehabilitation are provided in the "Guidelines for Cardiac Rehabilitation and Secondary Prevention Programs" by the American Association of Cardiovascular and Pulmonary Rehabilitation.²⁵⁸ Recommendations for risk stratification are seen in Tables 11 through 14. For the apparently healthy individual, no supervision is needed (Table 11). For those with unstable disease, no activity is recommended (Table 14). Additional guidelines are provided for moderate-to-high risk and low-risk subjects.

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Medically Supervised Exercise

Moderate- to High-Risk Subjects

Activity programs are needed to provide close medical supervision for individuals who are at moderate-to-high risk for a complication associated with vigorous physical activity. Such individuals are largely from class C (Table 13). These patients require careful medical supervision and surveillance to ensure that the activity is well tolerated. A physician should be readily available for these classes, although the presence of a properly trained and experienced nurse in the exercise room is sufficient if a physician is not in the exercise area. The qualifications of the physician may vary, but experience in internal medicine and cardiovascular disease and in treatment of subjects with heart disease is recommended. Training programs should be medically supervised until the safety of the prescribed activity has been established. All individuals entering these programs should be evaluated as described in Table 13.

Low-Risk Subjects

Low-risk subjects (class B) benefit from medically supervised programs because vigorous exercise can be conducted more safely, and group dynamics often help subjects comply with good health behaviors. Medical supervision of low-risk subjects can be provided by a well-trained nurse working under a physician's standard orders. If direct medical supervision by a physician is not provided, the supervisor should have successfully completed an AHA-sponsored course in Advanced Cardiac Life Support and should be able to administer emergency medications. Well-trained cardiovascular nurses usually meet these criteria. All individuals entering these programs should be evaluated as outlined in Table 12. The program should provide the same basic requirements detailed for high-risk subjects in Table 13.

Low-risk patients can exercise in nonmedical settings, including the home or health/fitness facilities. Such patients should be properly instructed by appropriately trained health-care professionals regarding the exercise prescription and self-monitoring techniques. Details regarding exercise in nonmedical settings are provided in the AHA/ACSM's "Recommendations for Cardiovascular Screening, Staffing, and Emergency Policies at Health/Fitness Facilities."²¹⁴

In the first 1 or 2 weeks after discharge from the hospital after MI, individuals may walk at a slow, regular pace with increasing duration, starting with 10-minute periods and working up to 1 hour. Such activity need not be supervised. Unmonitored exercise²⁶⁶ can also be used for conditioning after the individual has recovered from the MI (\geq 2 weeks after hospital discharge) or in other cases of stable CAD, although medically supervised and monitored exercise is preferred. If cardiac rehabilitation facilities are not available, activity guidelines can still be provided to cardiac subjects,

and they should be encouraged to exercise. If individuals carefully watch for signs of intolerance and are attentive to heart rate and rating of perceived exertion, this activity level is considered safe. Walking is a safe, low-impact, controllable exercise that in the majority of cases generates an intensity that is 40% to 70% of $\dot{V}o_{2 \text{ max}}$. Range-of-motion exercises and light calisthenics can be performed in an unmonitored setting. Activities are considered safe and appropriate if they meet the criterion of moderate intensity, as perceived by the physician or judged by an exercise test.

Guidelines for Electrocardiographic Monitoring

Various recommendations exist regarding the number of ECG-monitored sessions that are necessary and reasonable in an exercise training program. There are no controlled clinical trials that have specifically evaluated this issue. Some programs use as few as 6 sessions, with progression in mode and intensity of the exercise during these periods,²⁶⁷ whereas others have used as many as 36 sessions of ECG monitoring. The fewest possible sessions should be used, and it is recommended that the classification as outlined in Tables 12 and 13 be used as a general guideline. Importantly, the ultimate judgment must remain with the medical supervisor of the cardiac rehabilitation program and must consider the patient, staff, and exercise setting. Individuals who are class A (apparently healthy) do not require ECG-monitored sessions because the general guidelines are adequate. Class B individuals should be monitored and supervised until they understand their desirable activity levels (usually 6 to 12 sessions). Class C individuals should be medically supervised with ECG monitoring until they understand the level of activity that is safe and the medical team determines that the exercise is well tolerated and effective. Usually ≥ 12 sessions are needed.

ECG-Monitored Cardiac Rehabilitation

Monitoring sessions should ideally be performed with continuous ECG monitoring by either hardwired apparatus or telemetry. The sessions should be conducted by personnel who understand the exercise principles involved and have a working knowledge of electrocardiography and arrhythmia detection. The sessions should also be supervised by either a physician or a nurse trained in emergency CPR, preferably with previous experience in intensive cardiac care. Such individuals should have recently completed an AHAsponsored course in Advanced Cardiac Life Support. Standing orders for the management of a complication should be immediately available. Monitored sessions should also include symptom assessment by the staff, blood pressure recording, the subject's rating of perceived exertion, and instructions to subjects about selection and proper use of exercise equipment. ECG-monitored sessions should include instruction for different modes and progressions of exercise.

Home-Monitored Programs

The use of transtelephonic ECG monitoring at home has been suggested as a substitute for outpatient visits to the clinic.^{268,269} Such programs have the disadvantage of lacking immediate emergency medical care but the advantage of not requiring a clinic visit. These programs may be particularly useful in following subjects in the event that center-based cardiac rehabilitation programs are not readily available.²⁷⁰ One program reported using both ECG and voice transtele-phonic monitoring, which supported both the efficacy and safety of home programs.²⁷¹

Counseling and Compliance

To enhance health and prevent and treat cardiovascular disease, physical activity should be a permanent lifestyle behavior. Although an exercise prescription and the physician's advice to increase physical activity can be very strong motivators to patients, behavior change is very difficult for most persons. Prescriptions and advice alone may not be sufficient. Evaluation of the individual's readiness to change their behavior can be an important component of a successful exercise counseling program, such as that used in the Physician-based Assessment and Counseling for Exercise (PACE) project.^{272,273}

A recent review regarding physical activity interventions in healthcare settings included 12 studies in apparently healthy patients and 24 randomized studies in patients with cardiovascular disease.²⁷⁴ This literature provides evidence that such interventions can be successful in both the short and long term in increasing physical activity. However, only about half of the studies were successful in increasing physical activity or cardiorespiratory conditioning in their participants. Characteristics of successful interventions included long-term continuing intervention and multiple contacts, supervised exercise, provision of exercise equipment, and behavioral approaches. Importantly, the behavioral component fostered patient selection of an enjoyable activity, setting realistic goals, identifying barriers, problem-solving, self-monitoring, providing feedback and positive reinforcement, and enhancing social support.275 Continuing intervention and behavioral approaches have been shown to increase activity or fitness levels in CAD patients for as long as 4 to 5 years.276,277

One approach to promote an increase in physical activity among patients is for exercise to begin slowly and to progress gradually to the recommended exercise prescription, with assessment of success and reinforcement provided regularly. Patients can begin at a more moderate intensity, shorter duration, and lower frequency than the ultimate goal. Gradual increases in activity are not only safer for sedentary people and for patients with CAD, but short-term successes may increase the patient's self-efficacy for being physically active.²⁷⁸ The healthcare provider can use positive outcomes for feedback and reinforcement. This approach requires repeated follow-up visits.

The most effective interventions are those with multiple components and a continued maintenance intervention; they can be delivered using a model in which physicians provide advice and other members of the healthcare team provide more in-depth behavioral counseling and follow-up.²⁷⁹ For successful implementation of physical activity counseling in a healthcare setting, a coordinated, multilevel intervention should use strategies directed toward the practice environment, patients, and providers.²⁸⁰ Systematic delivery of a

counseling program might be enhanced through the use of encounter forms²⁸¹ and case management systems.²⁸² In addition, achieving greater implementation of physical activity interventions in healthcare settings will require improved education and training of health professionals and attention to healthcare policy and reimbursement issues.

Social Service and Vocational Rehabilitation

Helping the individual return to normal activities and a healthy lifestyle is an important focus of rehabilitation and requires close cooperation between the subject, physician, employer, and social service agencies. Decisions about longterm goals should be made early. These goals include issues of personal safety, an acceptable (preferably optimal) standard of living for the subject, and productivity for the employer. Heavy labor can increase myocardial work and thus may increase the risk of myocardial ischemia, arrhythmias, and sudden cardiac arrest. It may also pose a problem for employers who are liable for workers' compensation if a complication of heart disease occurs on the job. Most occupational activities require <5 METs. In the 15% of individuals in the labor force whose work involves heavy manual labor,²⁵⁸ the exercise test data should not be used as the sole criterion for recommendations regarding return to work. Energy demands of lifting heavy objects, temperature, environmental and psychological stresses are not assessed appropriately by routine exercise tests and must be taken into consideration. In patients with low functional capacity, LV dysfunction, exercise-induced myocardial ischemia, and those who are otherwise apprehensive about returning to a physically demanding occupation, simulated work tests can be performed.258,283,284

If the subject is in a low-risk activity category and exerts reasonable precautions, the probability of a complication is very small. Hence, such subjects should be encouraged and helped to return to work. If the subject is in a high-risk category, the case must be judged on its individual merits. Even so, many of these subjects can return to work if the following guidelines are followed:

- The subject should participate in an organized, medically supervised cardiac rehabilitation program to enhance strength and endurance and to provide surveillance and education during return to activity.
- Mechanical devices should be used when possible to reduce the amount of lifting required.
- If the subject is required to perform lifting or carrying activities, this should take place in optimum environmental conditions and be spaced with rest periods to avoid cumulative effects. Arm or resistance training in cardiac rehabilitation programs may be particularly useful for individuals in this group.
- Good cardiovascular health should be maintained through risk factor reduction and regular medical follow-up.

Sexual Activity

Sexual activity in the patient after MI may be resumed at the same time as other activities, such as walking and driving, are resumed, usually ≈ 2 to 4 weeks after returning home.

However, this must be considered in the context of the patient's physical, medical, and emotional status.²⁵⁸ Details regarding these issues and appropriate counseling are discussed elsewhere. In general, sexual activity is similar to moderate-intensity exercise for most individuals with CAD. Heart rates rarely exceed 120 beats/min, systolic blood pressure is <170 mm Hg, and metabolic requirements are between 5 and 7 METs.²⁸⁵ There seems to be no particular benefit in altering positions or sexual customs. Exercise training can, however, lessen the hemodynamic stress of sexual activity. The use of β -blockers and other drugs may impair sexual performance.

Obtaining Informed Consent for Exercise Training

Obtaining informed signed consent before initiating an exercise training program helps to clarify the responsibilities and goals of both the physician and the subject. A sample consent is shown below.

Informed Consent for Exercise Training

I want to participate in the ______ exercise training program to improve my cardiovascular function. This program was recommended by my physician, Dr _____.

I will have a clinical evaluation before I enter this exercise program. This evaluation will include a medical history and physical examination consisting of but not limited to ECG at rest and, in some instances, with effort, and measurements of heart rate and blood pressure. The purpose of this evaluation is to determine the safety of my participation in this exercise training program.

The program will follow an exercise prescription formulated by Dr _____.

I understand that activities are designed to place a gradually increasing workload on the circulation in an attempt to improve its function. The reaction of the cardiovascular system to such activities cannot be predicted with complete accuracy. Certain changes may occur during or after exercise, including abnormalities of blood pressure or heart rate, ineffective heart function, and, possibly, in some instances, heart attacks or cardiac arrest.

I realize that it is necessary for me to promptly report symptoms or signs indicating any abnormality or distress to the exercise supervisor. I consent to administration of immediate resuscitation measures deemed advisable by the exercise supervisor.

I have read the above and I understand it. My questions have been answered to my satisfaction.

Subject: _	
Physician:	
Witness:	
Date:	

Glossary

Testing

Arrhythmia: dysrhythmia or abnormal heart rhythm Balke-type protocol: constant speed (2.0 to 3.0 miles/ hour), variable grade treadmill exercise test

- Bruce-type protocol: variable speed and grade treadmill exercise test (incremental speed and grade increase every 3 minutes)
- CAD (coronary artery disease): coronary heart disease, MI, CABG, coronary angioplasty, and myocardial ischemia
- Calories (kilocalorie): amount of energy required to raise temperature of 1 kg of water by 1°C

Calories/min: (METs×3.5×body weight in kilograms)/200

- Exercise capacity: functional capacity, training, or conditioning level; level of fitness
- Isometric/static exercise: muscle contraction with no movement (see "resistance exercise" below)
- Isotonic/dynamic exercise: muscle contraction producing movement
- J-junctional (J-point) depression: depression at the beginning of ST segment
- Kilogram (kg): 1000 g
- Kilopond-meter (kpm): kilogram-meter of work=1 J (10 ergs)
- MET: metabolic equivalent (3.5 mL \cdot kg⁻¹ \cdot min⁻¹ of oxygen uptake)
- 0.1 mV=1 mm (provided calibration is set at 10 mm/mV)
- Predictive value: percentage of those with or without disease who are identified correctly
- PTCA: percutaneous transluminal coronary angioplasty
- Rating of perceived exertion: Borg scale of 6 to 20 or 1 to 10
- Resistance exercise: muscle contraction with limited movement
- Sensitivity: percentage of persons who have disease who will have a positive test
- Specificity: percentage of persons who do not have disease who will have a negative test
- ST depression: horizontal or downsloping (0.10 mV/ms) segment, measured from isoelectric PR level
- Training: physical activity and conditioning leading to fitness
- Ventilatory threshold: a measure of relative work effort that represents the point at which ventilation abruptly increases despite linear increases in oxygen uptake
- Vo₂: oxygen uptake
- VO_{2 max}: maximal oxygen uptake

Training

- Aerobic: exercise in which energy needed is provided by using oxygen inspired to combust metabolites
- Anaerobic: exercise in which energy needed exceeds oxidative processes and nonaerobic metabolism begins
- Cardiac output: volume of blood ejected from heart in liters per minute (normal is 4 to 6 L/min at rest, depending on body size)
- Cardiovascular exercise: predominantly dynamic exercise using large-muscle groups
- Ejection fraction: ratio of LV stroke volume to end-diastolic volume (or percentage of end-diastolic volume ejected with each cardiac contraction); normal is 60% to 75%
- Flexibility activity: activity designed to enhance range of motion of joints
- Medical supervision: physician readily available (the presence of a properly trained nurse in the exercise room is acceptable if physician is not available in the exercise room)

- NYHA class: New York Heart Association classification Class 1: heart disease without symptoms
- Class 2: heart disease with symptoms during ordinary
- activity
- Class 3: heart disease with symptoms during less than ordinary activity
- Class 4: heart disease with symptoms at rest
- Occupational activity: on-the-job activity, such as a job requiring lifting of loads ≥ 20 pounds at least hourly throughout the day or constantly moving any size load from place to place without mechanized aid
- Strength activity: muscular contraction against resistance designed to increase skeletal muscle strength
- Stroke volume: amount of blood ejected from the heart with each contraction; normal is 80 to 90 mL at rest in a 70-kg man

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References

- Fletcher GF, Balady G, Blair SN, et al. Statement on exercise: benefits and recommendations for physical activity programs for all Americans: a statement for health professionals by the Committee on Exercise and Cardiac Rehabilitation of the Council on Clinical Cardiology, American Heart Association. *Circulation*. 1996;94:857–862.
- Fletcher GF, Balady G, Froelicher VF, et al. Exercise standards: a statement for healthcare professionals from the American Heart Association Writing Group. *Circulation*. 1995;91:580–615.
- Gibbons RJ, Balady GJ, Bricker JT, et al. ACC/AHA guidelines for exercise testing update: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Exercise Testing). J Am Coll Cardiol. In press.
- Rowell LB, ed. Human Circulation: Regulation During Physical Stress. New York: Oxford University Press; 1986.
- MacDougall J. Blood pressure responses to resistive static and dynamic exercise. In: Fletcher G, ed. *Cardiovascular Response to Exercise*. Mount Kisco, NY: Futura Publishing Co, Inc; 1994:155–173.
- Cohn JN, ed. Quantitative exercise testing for the cardiac patient: the value of monitoring gas exchange: introduction. *Circulation*. 1987; 76(suppl VI):VI-1–VI-2.
- Cohen-Solal A, Zannad F, Kayanakis JG, et al. Multicentre study of the determination of peak oxygen uptake and ventilatory threshold during bicycle exercise in chronic heart failure: comparison of graphical methods, interobserver variability and influence of the exercise protocol: the VO2 French Study Group. *Eur Heart J.* 1991;12:1055–1063.
- Bouchard C, Daw EW, Rice T, et al. Familial resemblance for VO2 max in the sedentary state: the HERITAGE family study. *Med Sci Sports Exerc.* 1998;30:252–258.
- Bouchard C, An P, Rice T, et al. Familial aggregation of VO(2 max) response to exercise training: results from the HERITAGE Family Study. J Appl Physiol. 1999;87:1003–1008.
- Morris CK, Myers J, Froelicher VF, et al. Nomogram based on metabolic equivalents and age for assessing aerobic exercise capacity in men. *J Am Coll Cardiol*. 1993;22:175–182.
- 11. Londeree BR, Moeschberger ML. Influence of age and other factors on maximal heart rate. *J Cardiac Rehabil.* 1984;4:44–49.
- Pina IL, Balady GJ, Hanson P, et al. Guidelines for clinical exercise testing laboratories: a statement for healthcare professionals from the Committee on Exercise and Cardiac Rehabilitation, American Heart Association. *Circulation*. 1995;91:912–921.
- Franklin BA. Exercise testing, training and arm ergometry. Sports Med. 1985;2:100–119.
- Balady GJ, Weiner DA, McCabe CH, et al. Value of arm exercise testing in detecting coronary artery disease. Am J Cardiol. 1985;55:37–39.

- Kaminsky LA, Whaley MH. Evaluation of a new standardized ramp protocol: the BSU/Bruce Ramp protocol. *J Cardiopulm Rehabil*. 1998; 18:438–444.
- Bittner V, Weiner DH, Yusuf S, et al. Prediction of mortality and morbidity with a 6-minute walk test in patients with left ventricular dysfunction: SOLVD Investigators. *JAMA*. 1993;270:1702–1707.
- Rodgers GP, Ayanian JZ, Balady G, et al. American College of Cardiology/American Heart Association clinical competence statement on stress testing: a report of the American College of Cardiology/American Heart Association/American College of Physicians-American Society of Internal Medicine Task Force on clinical competence. *Circulation*. 2000;102:1726–1738.
- Gordon NF, Kohl HW. Exercise testing and sudden cardiac death. J Cardiopulm Rehabil. 1993;13:381–386.
- Borg GA. Psychophysical bases of perceived exertion. *Med Sci Sports Exerc*. 1982;14:377–381.
- 20. Deleted in proof.
- McHam SA, Marwick TH, Pashkow FJ, et al. Delayed systolic blood pressure recovery after graded exercise: an independent correlate of angiographic coronary disease. J Am Coll Cardiol. 1999;34:754–759.
- Dubach P, Froelicher VF, Klein J, et al. Exercise-induced hypotension in a male population: criteria, causes, and prognosis. *Circulation*. 1988;78: 1380–1387.
- Lauer MS, Francis GS, Okin PM, et al. Impaired chronotropic response to exercise stress testing as a predictor of mortality. *JAMA*. 1999;281: 524–529.
- Okin PM, Kligfield P. Gender-specific criteria and performance of the exercise electrocardiogram. *Circulation*. 1995;92:1209–1216.
- Kligfield P, Ameisen O, Okin PM. Heart rate adjustment of ST segment depression for improved detection of coronary artery disease. *Circulation*. 1989;79:245–255.
- 26. Cheitlin MD, Alpert JS, Armstrong WF, et al. ACC/AHA guidelines for the clinical application of echocardiography: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Clinical Application of Echocardiography): developed in collaboration with the American Society of Echocardiography. *Circulation*. 1997;95:1686–1744.
- 27. Ritchie JL, Bateman TM, Bonow RO, et al. Guidelines for clinical use of cardiac radionuclide imaging: report of the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Committee on Radionuclide Imaging), developed in collaboration with the American Society of Nuclear Cardiology. J Am Coll Cardiol. 1995;25:521–547.
- Secknus MA, Marwick TH. Evolution of dobutamine echocardiography protocols and indications: safety and side effects in 3011 studies over 5 years. J Am Coll Cardiol. 1997;29:1234–1240.
- Geleijnse ML, Fioretti PM, Roelandt JR. Methodology, feasibility, safety and diagnostic accuracy of dobutamine stress echocardiography. *J Am Coll Cardiol*. 1997;30:595–606.
- Vaduganathan P, He ZX, Raghavan C, et al. Detection of left anterior descending coronary artery stenosis in patients with left bundle branch block: exercise, adenosine or dobutamine imaging? *J Am Coll Cardiol*. 1996;28:543–550.
- Gianrossi R, Detrano R, Mulvihill D, et al. Exercise-induced ST depression in the diagnosis of coronary artery disease: a meta-analysis. *Circulation*. 1989;80:87–98.
- Mark DB, Shaw L, Harrell FE Jr, et al. Prognostic value of a treadmill exercise score in outpatients with suspected coronary artery disease. *N Engl J Med.* 1991;325:849–853.
- Heinsimer JA, Irwin JM, Basnight LL. Influence of underlying coronary artery disease on the natural history and prognosis of exercise-induced left bundle branch block. *Am J Cardiol.* 1987;60:1065–1067.
- Vasey C, O'Donnell J, Morris S, et al. Exercise-induced left bundle branch block and its relation to coronary artery disease. *Am J Cardiol.* 1985;56:892–895.
- Whinnery JE, Froelicher VF Jr, Longo MR Jr, et al. The electrocardiographic response to maximal treadmill exercise of asymptomatic men with right bundle branch block. *Chest.* 1977;71:335–340.
- Williams MA, Esterbrooks DJ, Nair CK, et al. Clinical significance of exercise-induced bundle branch block. *Am J Cardiol*. 1988;61:346–348.
- Wayne VS, Bishop RL, Cook L, et al. Exercise-induced bundle branch block. Am J Cardiol. 1983;52:283–286.
- Whinnery JE, Froelicher VF. Exercise testing in right bundle-branch block. *Chest.* 1977;72:684–685. Letter.

- Whinnery JE, Froelicher VF. Acquired bundle branch block and its response to exercise testing in asymptomatic air crewmen: a review with case reports. *Aviat Space Environ Med.* 1976;46:69–78.
- Sharma AD, Yee R, Guiraudon G, et al. Sensitivity and specificity of invasive and noninvasive testing for risk of sudden death in Wolff-Parkinson-White syndrome. J Am Coll Cardiol. 1987;10:373–381.
- Allen BJ, Casey TP, Brodsky MA, et al. Exercise testing in patients with life-threatening ventricular tachyarrhythmias: results and correlation with clinical and arrhythmia factors. *Am Heart J.* 1988;116:997–1002.
- Ryan M, Lown B, Horn H. Comparison of ventricular ectopic activity during 24-hour monitoring and exercise testing in patients with coronary heart disease. N Engl J Med. 1975;292:224–229.
- 43. Sami M, Chaitman B, Fisher L, et al. Significance of exercise-induced ventricular arrhythmia in stable coronary artery disease: a Coronary Artery Surgery Study project. *Am J Cardiol.* 1984;54:1182–1188.
- Atwood JE, Myers J, Sullivan M, et al. Maximal exercise testing and gas exchange in patients with chronic atrial fibrillation. J Am Coll Cardiol. 1988;11:508–513.
- Weber KT, Kinasewitz GT, Janicki JS, et al. Oxygen utilization and ventilation during exercise in patients with chronic cardiac failure. *Circulation*. 1982;65:1213–1223.
- Mancini DM, Eisen H, Kussmaul W, et al. Value of peak exercise oxygen consumption for optimal timing of cardiac transplantation in ambulatory patients with heart failure. *Circulation*. 1991;83:778–786.
- Manolio TA, Burke GL, Savage PJ, et al. Exercise blood pressure response and 5-year risk of elevated blood pressure in a cohort of young adults: the CARDIA study. *Am J Hypertens*. 1994;7:234–241.
- Matthews CE, Pate RR, Jackson KL, et al. Exaggerated blood pressure response to dynamic exercise and risk of future hypertension. *J Clin Epidemiol.* 1998;51:29–35.
- Singh JP, Larson MG, Manolio TA, et al. Blood pressure response during treadmill testing as a risk factor for new-onset hypertension: the Framingham heart study. *Circulation*. 1999;99:1831–1836.
- Mundal R, Kjeldsen SE, Sandvik L, et al. Exercise blood pressure predicts mortality from myocardial infarction. *Hypertension*. 1996;27: 324–329.
- 51. Wilson JR, Fink LI, Ferraro N, et al. Use of maximal bicycle exercise testing with respiratory gas analysis to assess exercise performance in patients with congestive heart failure secondary to coronary artery disease or to idiopathic dilated cardiomyopathy. *Am J Cardiol.* 1986; 58:601–606.
- Losse B, Kuhn H, Loogen F, et al. Exercise performance in hypertrophic cardiomyopathies. *Eur Heart J.* 1983;4:197–208.
- 53. Savage DD, Seides SF, Maron BJ, et al. Prevalence of arrhythmias during 24-hour electrocardiographic monitoring and exercise testing in patients with obstructive and nonobstructive hypertrophic cardiomyopathy. *Circulation*. 1979;59:866–875.
- 54. Ryan TJ, Anderson JL, Antman EM, et al. ACC/AHA guidelines for the management of patients with acute myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). J Am Coll Cardiol. 1996;28:1328–1428.
- Juneau M, Colles P, Theroux P, et al. Symptom-limited versus low level exercise testing before hospital discharge after myocardial infarction. *J Am Coll Cardiol*. 1992;20:927–933.
- Hamm LF, Crow RS, Stull GA, et al. Safety and characteristics of exercise testing early after acute myocardial infarction. *Am J Cardiol.* 1989;63:1193–1197.
- 57. Newby LK, Califf RM, Guerci A, et al. Early discharge in the thrombolytic era: an analysis of criteria for uncomplicated infarction from the Global Utilization of Streptokinase and t-PA for Occluded Coronary Arteries (GUSTO) trial. J Am Coll Cardiol. 1996;27:625–632.
- Chaitman BR, McMahon RP, Terrin M, et al. Impact of treatment strategy on predischarge exercise test in the Thrombolysis in Myocardial Infarction (TIMI) II trial. *Am J Cardiol.* 1993;71:131–138.
- 59. Krone RJ, Dwyer EM Jr, Greenberg H, et al. Risk stratification in patients with first non-Q wave infarction: limited value of the early low level exercise test after uncomplicated infarcts: the Multicenter Post-Infarction Research Group. *J Am Coll Cardiol.* 1989;14:31–37; discussion 38–39.
- 60. Ronnevik PK, von der Lippe G. Prognostic importance of predischarge exercise capacity for long-term mortality and non-fatal myocardial infarction in patients admitted for suspected acute myocardial infarction and treated with metoprolol. *Eur Heart J.* 1992;13:1468–1472.

- Dagenais GR, Rouleau JR, Hochart P, et al. Survival with painless strongly positive exercise electrocardiogram. *Am J Cardiol.* 1988;62: 892–895.
- 62. Deleted in proof.
- 63. Deleted in proof.
- 64. Bruce RA, DeRouen TA, Hossack KF. Value of maximal exercise tests in risk assessment of primary coronary heart disease events in healthy men: five years' experience of the Seattle heart watch study. *Am J Cardiol.* 1980;46:371–378.
- Allen WH, Aronow WS, Goodman P, et al. Five-year follow-up of maximal treadmill stress test in asymptomatic men and women. *Circulation*. 1980;62:522–527.
- Jouven X, Zureik M, Desnos M, et al. Long-term outcome in asymptomatic men with exercise-induced premature ventricular depolarizations. N Engl J Med. 2000;343:826–833.
- Ekelund LG, Suchindran CM, McMahon RP, et al. Coronary heart disease morbidity and mortality in hypercholesterolemic men predicted from an exercise test: the Lipid Research Clinics Coronary Primary Prevention Trial. J Am Coll Cardiol. 1989;14:556–563.
- Rautaharju PM, Prineas RJ, Eifler WJ, et al. Prognostic value of exercise electrocardiogram in men at high risk of future coronary heart disease: Multiple Risk Factor Intervention Trial experience. J Am Coll Cardiol. 1986;8:1–10.
- Kim C, Kwok YS, Saha S, et al. Diagnosis of suspected coronary artery disease in women: a cost-effectiveness analysis. *Am Heart J.* 1999;137: 1019–1027.
- Vaitkevicius PV, Fleg JL. An abnormal exercise treadmill test in an asymptomatic older patient. J Am Geriatr Soc. 1996;44:83–88.
- Hlatky MA, Pryor DB, Harrell FE Jr, et al. Factors affecting sensitivity and specificity of exercise electrocardiography: multivariable analysis. *Am J Med.* 1984;77:64–71.
- Smith SC, Amsterdam E, Balady GJ, et al. Prevention V: beyond secondary prevention: identifying the high risk patient for primary prevention: writing group II. *Circulation*. 2000;101:e12–e15.
- 73. Bonow RO, Carabello B, de Leon AC Jr, et al. Guidelines for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Patients with Valvular Heart Disease). *Circulation*. 1998;98: 1949–1984.
- Hochreiter C, Borer JS. Exercise testing in patients with aortic and mitral valve disease: current applications. *Cardiovasc Clin.* 1983;13: 291–300.
- Areskog NH. Exercise testing in the evaluation of patients with valvular aortic stenosis. *Clin Physiol.* 1984;4:201–208.
- Atwood JE, Kawanishi S, Myers J, et al. Exercise testing in patients with aortic stenosis. *Chest.* 1988;93:1083–1087.
- Misra M, Thakur R, Bhandari K, et al. Value of the treadmill exercise test in asymptomatic and minimally symptomatic patients with chronic severe aortic regurgitation. *Int J Cardiol.* 1987;15:309–316.
- Vacek JL, Valentin-Stone P, Wolfe M, et al. The value of standardized exercise testing in the noninvasive evaluation of mitral stenosis. *Am J Med Sci.* 1986;292:335–343.
- Weber KT, Janicki JS, McElroy PA. Cardio-pulmonary exercise testing in the evaluation of mitral and aortic valve incompetence. *Herz.* 1986; 11:88–96.
- Lee TH, Shammash JB, Ribeiro JP, et al. Estimation of maximum oxygen uptake from clinical data: performance of the Specific Activity Scale. *Am Heart J.* 1988;115:203–204.
- Eagle KA, Brundage BH, Chaitman BR, et al. ACC/AHA guidelines for perioperative cardiovascular evaluation for noncardiac surgery: report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines, Committee on Perioperative Cardiovascular Evaluation for Noncardiac Surgery. *Circulation*. 1996;93: 1278–1317.
- Carliner NH, Fisher ML, Plotnick GD, et al. Routine preoperative exercise testing in patients undergoing major noncardiac surgery. *Am J Cardiol.* 1985;56:51–58.
- Dehn MM, Bruce RA. Longitudinal variations in maximal oxygen intake with age and activity. J Appl Physiol. 1972;33:805–807.
- Fleg JL, Lakatta EG. Role of muscle loss in the age-associated reduction in VO2 max. J Appl Physiol. 1988;65:1147–1151.
- Ogawa T, Spina RJ, Martin WH 3rd, et al. Effects of aging, sex, and physical training on cardiovascular responses to exercise. *Circulation*. 1992;86:494–503.

- Fleg JL, O'Connor F, Gerstenblith G, et al. Impact of age on the cardiovascular response to dynamic upright exercise in healthy men and women. J Appl Physiol. 1995;78:890–900.
- Daida H, Allison TG, Squires RW, et al. Peak exercise blood pressure stratified by age and gender in apparently healthy subjects. *Mayo Clin Proc.* 1996;71:445–452.
- Port S, Cobb FR, Coleman RE, et al. Effect of age on the response of the left ventricular ejection fraction to exercise. *N Engl J Med.* 1980;303: 1133–1137.
- Fleg JL, Tzankoff SP, Lakatta EG. Age-related augmentation of plasma catecholamines during dynamic exercise in healthy males. J Appl Physiol. 1985;59:1033–1039.
- White NK, Edward JE, Dry TJ. The relationship of the degree of coronary atherosclerosis with age in men. *Circulation*. 1950;1:645–654.
- Ackerman RF, Dry TJ, Edwards JE. Relationship of various factors to the degree of coronary atherosclerosis in women. *Circulation*. 1950;1: 1345–1354.
- Elveback L, Lie JT. Continued high incidence of coronary artery disease at autopsy in Olmsted County, Minnesota, 1950 to 1979. *Circulation*. 1984;70:345–349.
- Gersh BJ, Kronmal RA, Frye RL, et al. Coronary arteriography and coronary artery bypass surgery: morbidity and mortality in patients ages 65 years or older: a report from the Coronary Artery Surgery Study. *Circulation*. 1983;67:483–491.
- Miyamura M, Honda Y. Oxygen intake and cardiac output during maximal treadmill and bicycle exercise. J Appl Physiol. 1972;32: 185–188.
- Myers J, Buchanan N, Walsh D, et al. Comparison of the ramp versus standard exercise protocols. J Am Coll Cardiol. 1991;17:1334–1342.
- Maurer MS, Shefrin EA, Fleg JL. Prevalence and prognostic significance of exercise-induced supraventricular tachycardia in apparently healthy volunteers. *Am J Cardiol.* 1995;75:788–792.
- Busby MJ, Shefrin EA, Fleg JL. Prevalence and long-term significance of exercise-induced frequent or repetitive ventricular ectopic beats in apparently healthy volunteers. J Am Coll Cardiol. 1989;14:1659–1665.
- Deckers JW, Fioretti P, Brower RW, et al. Ineligibility for predischarge exercise testing after myocardial infarction in the elderly: implications for prognosis. *Eur Heart J.* 1984;5:97–100.
- Fioretti P, Deckers JW, Brower RW, et al. Predischarge stress test after myocardial infarction in the old age: results and prognostic value. *Eur Heart J.* 1984;5:101–104.
- Ciaroni S, Delonca J, Righetti A. Early exercise testing after acute myocardial infarction in the elderly: clinical evaluation and prognostic significance. *Am Heart J.* 1993;126:304–311.
- Samek L, Betz P, Schnellbacher K. Exercise testing in elderly patients with coronary artery disease. *Eur Heart J.* 1984;5:69–73.
- 102. Glover DR, Robinson CS, Murray RG. Diagnostic exercise testing in 104 patients over 65 years of age. *Eur Heart J.* 1984;5:59–61.
- 103. Josephson RA, Shefrin E, Lakatta EG, et al. Can serial exercise testing improve the prediction of coronary events in asymptomatic individuals? *Circulation*. 1990;81:20–24.
- 104. Weber KT, Janicki JS. Equipment and protocol to evaluate the exercise response. In: Weber KT, Janicki JS, eds. *Cardiopulmonary Exercise Testing: Physiologic Principles and Clinical Applications*. Philadelphia: Saunders; 1986:139–150.
- Fletcher BJ, Dunbar SB, Felner JM, et al. Exercise testing and training in physically disabled men with clinical evidence of coronary artery disease. *Am J Cardiol.* 1994;73:170–174.
- Schwade J, Blomqvist CG, Shapiro W. A comparison of the response to arm and leg work in patients with ischemic heart disease. *Am Heart J*. 1977;94:203–208.
- 107. Stein R, Chaitman B, Balady GJ, et al. Safety and utility of exercise testing in emergency room chest pain centers: an advisory from the Committee on Exercise, Rehabilitation, and Prevention, Council on Clinical Cardiology, American Heart Association. *Circulation*. 2000; 102:1463–1467.
- 108. Lewis WR, Amsterdam EA, Turnipseed S, et al. Immediate exercise testing of low risk patients with known coronary artery disease presenting to the emergency department with chest pain. *J Am Coll Cardiol*. 1999;33:1843–1847.
- Goldman L, Cook EF, Brand DA, et al. A computer protocol to predict myocardial infarction in emergency department patients with chest pain. *N Engl J Med.* 1988;318:797–803.
- Roberts RR, Zalenski RJ, Mensah EK, et al. Costs of an emergency department-based accelerated diagnostic protocol vs hospitalization in

patients with chest pain: a randomized controlled trial. JAMA. 1997; 278:1670-1676.

- 111. Farkouh ME, Smars PA, Reeder GS, et al. A clinical trial of a chest-pain observation unit for patients with unstable angina: Chest Pain Evaluation in the Emergency Room (CHEER) Investigators. N Engl J Med. 1998; 339:1882–1888.
- 112. Gomez MA, Anderson JL, Karagounis LA, et al. An emergency department-based protocol for rapidly ruling out myocardial ischemia reduces hospital time and expense: results of a randomized study (ROMIO). J Am Coll Cardiol. 1996;28:25–33.
- Mikhail MG, Smith FA, Gray M, et al. Cost-effectiveness of mandatory stress testing in chest pain center patients. *Ann Emerg Med.* 1997;29: 88–98.
- 114. Polanczyk CA, Johnson PA, Hartley LH, et al. Clinical correlates and prognostic significance of early negative exercise tolerance test in patients with acute chest pain seen in the hospital emergency department. *Am J Cardiol.* 1998;81:288–292.
- 115. Zalenski RJ, McCarren M, Roberts R, et al. An evaluation of a chest pain diagnostic protocol to exclude acute cardiac ischemia in the emergency department. Arch Intern Med. 1997;157:1085–1091.
- 116. Tsakonis JS, Shesser R, Rosenthal R, et al. Safety of immediate treadmill testing in selected emergency department patients with chest pain: a preliminary report. *Am J Emerg Med.* 1991;9:557–559.
- 117. Kerns JR, Shaub TF, Fontanarosa PB. Emergency cardiac stress testing in the evaluation of emergency department patients with atypical chest pain. Ann Emerg Med. 1993;22:794–798.
- 118. Lewis WR, Amsterdam EA. Evaluation of the patient with 'rule out myocardial infarction.' *Arch Intern Med.* 1996;156:41–45.
- 119. Kirk JD, Turnipseed S, Lewis WR, et al. Evaluation of chest pain in low-risk patients presenting to the emergency department: the role of immediate exercise testing. *Ann Emerg Med.* 1998;32:1–7.
- 120. Amsterdam EA, Kirk JD, Turnipseed ST, et al. Immediate exercise testing for assessment of clinical risk in patients presenting to the emergency department with chest pain: results in over 1000 patients. *Circulation*. 1998;98:I-774. Abstract.
- 121. Sullivan M, Savvides M, Abouantoun S, et al. Failure of transdermal nitroglycerin to improve exercise capacity in patients with angina pectoris. *J Am Coll Cardiol*. 1985;5:1220–1223.
- 122. Sullivan M, Atwood JE, Myers J, et al. Increased exercise capacity after digoxin administration in patients with heart failure. *J Am Coll Cardiol*. 1989;13:1138–1143.
- 123. Pate RR, Pratt M, Blair SN, et al. Physical activity and public health: a recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *JAMA*. 1995;273: 402–407.
- 124. US Department of Health and Human Services. *Physical Activity and Health: A report of the Surgeon General*. Pittsburgh, Pa: President's Council on Physical Fitness and Sports; 1996.
- 125. Hartley LH, Grimby G, Kilbom A, et al. Physical training in sedentary middle-aged and older men: cardiac output and gas exchange during submaximal and maximal exercise. *Scand J Clin Lab Invest.* 1969;24: 335–344.
- 126. Sweeney ME, Fletcher BJ, Fletcher GF. Exercise testing and training with beta-adrenergic blockade: role of the drug washout period in "unmasking" a training effect. *Am Heart J.* 1989;118:941–946.
- 127. Pollock ML, T. LD, Foster C, et al. Acute and chronic responses to exercise in patients treated with beta blockers. J Cardiopulm Rehabil. 1991;11:132–144.
- 128. Saltin B, Blomqvist G, Mitchell JH, et al. Response to exercise after bed rest and after training. *Circulation*. 1968;38:VII-1–VII-78.
- Physical training and intrinsic cardiac adaptations. *Circulation*. 1973; 47:677–680.
- Hartley LH, Mason JW, Hogan RP, et al. Multiple hormonal responses to prolonged exercise in relation to physical training. *J Appl Physiol*. 1972;33:607–610.
- 131. Holloszy JO. Biochemical adaptations in muscle: effects of exercise on mitochondrial oxygen uptake and respiratory enzyme activity in skeletal muscle. *J Biol Chem.* 1967;242:2278–2282.
- Gleser MA, Vogel JA. Endurance exercise: effect of work-rest schedules and repeated testing. J Appl Physiol. 1971;31:735–739.
- 133. Hultman E. Studies on muscle metabolism of glycogen and active phosphate in man with special reference to exercise and diet. *Scand J Clin Lab Invest*. 1967;19:1–63.
- 134. Hartley LH. Exercise and cardiac rehabilitation. Proc N Engl Cardiovasc Soc. 1976;28:37–40.

- Detry JM, Rousseau M, Vandenbroucke G, et al. Increased arteriovenous oxygen difference after physical training in coronary heart disease. *Circulation*. 1971;44:109–118.
- 136. Oberman A, Fletcher GF, Lee J, et al. Efficacy of high-intensity exercise training on left ventricular ejection fraction in men with coronary artery disease (the Training Level Comparison Study). *Am J Cardiol.* 1995; 76:643–647.
- 137. Rerych SK, Scholz PM, Sabiston DC Jr, et al. Effects of exercise training on left ventricular function in normal subjects: a longitudinal study by radionuclide angiography. Am J Cardiol. 1980;45:244–252.
- 138. Sullivan MJ, Knight JD, Higginbotham MB, et al. Relation between central and peripheral hemodynamics during exercise in patients with chronic heart failure: muscle blood flow is reduced with maintenance of arterial perfusion pressure. *Circulation*. 1989;80:769–781.
- Hambrecht R, Niebauer J, Fiehn E, et al. Physical training in patients with stable chronic heart failure: effects on cardiorespiratory fitness and ultrastructural abnormalities of leg muscles. *J Am Coll Cardiol*. 1995; 25:1239–1249.
- Adamopoulos S, Coats AJ, Brunotte F, et al. Physical training improves skeletal muscle metabolism in patients with chronic heart failure. *J Am Coll Cardiol.* 1993;21:1101–1106.
- Redwood DR, Rosing DR, Epstein SE. Circulatory and symptomatic effects of physical training in patients with coronary artery disease and angina pectoris. N Engl J Med. 1972;286:959–965.
- 142. Ehsani A, Martin W, Heath G, et al. Cardiac effects of prolonged and intense exercise training in patients with coronary artery disease. *Am J Cardiol*. 1982;50:246–254.
- 143. Niebauer J, Hambrecht R, Velich T, et al. Attenuated progression of coronary artery disease after 6 years of multifactorial risk intervention: role of physical exercise. *Circulation*. 1997;96:2534–2541.
- 144. Gould KL, Ornish D, Kirkeeide R, et al. Improved stenosis geometry by quantitative coronary arteriography after vigorous risk factor modification. *Am J Cardiol.* 1992;69:845–853.
- 145. Fuster V, Gotto AM, Libby P, et al. 27th Bethesda Conference: matching the intensity of risk factor management with the hazard for coronary disease events. J Am Coll Cardiol. 1996;27:964–976.
- NIH Consensus Development Panel on Physical Activity and Cardiovascular Health. Physical activity and cardiovascular health. JAMA. 1996;276:241–246.
- 147. Leon AS, ed. *Physical Activity and Cardiovascular Health: A National Consensus.* Champaign, Ill: Human Kinetics; 1997.
- 148. Leon AS. Contribution of regular moderate-intensity physical activity. In: Leon AS, ed. *Physical Activity and Cardiovascular Health: A National Consensus.* Champaign, Ill: Human Kinetics; 1997:55–66.
- 149. Lee IM, Paffenbarger RS Jr. Is vigorous physical activity necessary to reduce the risk of cardiovascular disease? In: Leon AS, ed. *Physical Activity and Cardiovascular Health: A National Consensus*. Champaign, Ill: Human Kinetics; 1997:67–75.
- Paffenbarger RS Jr, Lee IM. Physical activity and fitness for health and longevity. *Res Q Exerc Sports*. 1996;67:S11–S28.
- Berlin JA, Colditz GA. A meta-analysis of physical activity in the prevention of coronary heart disease. Am J Epidemiol. 1990;132: 612–628.
- Powell KE, Thompson PD, Caspersen CJ, et al. Physical activity and the incidence of coronary heart disease. *Annu Rev Public Health*. 1987;8: 253–287.
- 153. Farrell SW, Kampert JB, Kohl HW 3rd, et al. Influences of cardiorespiratory fitness levels and other predictors on cardiovascular disease mortality in men. *Med Sci Sports Exerc.* 1998;30:899–905.
- Leon AS. Effects of exercise conditioning on physiologic precursors of coronary heart disease. J Cardiopulm Rehabil. 1991;11:46–57.
- 155. Leon AS, Richardson M. Exercise, health, and disease. In: Roberts SO, Robergs RA, Hanson P, eds. *Clinical Exercise Testing and Prescription: Theory and Application.* Boca Raton, Fla: CRC Press; 1997:281–302.
- 156. Kramsch DM, Aspen AJ, Abramowitz BM, et al. Reduction of coronary atherosclerosis by moderate conditioning exercise in monkeys on an atherogenic diet. N Engl J Med. 1981;305:1483–1489.
- 157. Squires RW. Mechanisms by which exercise training may improve the clinical status of cardiac patients. In: Pollock ML, Schmidt DH, eds. *Heart Disease and Rehabilitation*, 3rd ed. Champaign, Ill: Human Kinetics; 1995:147–160.
- Bouchard C, Despres JP. Physical activity and health: atherosclerotic, metabolic, and hypertensive diseases. *Res Q Exerc Sport.* 1995;66: 268–275.

- 159. Haskell WL. Sedentary lifestyle as a risk factor for coronary heart disease. In: Pearson TA, ed. *Primer in Preventive Cardiology*. Dallas, Tex: American Heart Association; 1994:173–187.
- Rao GHR. Effect of exercise on platelet physiology and pharmacology. In: Somani SM, ed. *Pharmacology and Toxicology*. Boca Raton, Fla: CRC Press; 1996:211–223.
- Stratton JR, Chandler WL, Schwartz RS, et al. Effects of physical conditioning on fibrinolytic variables and fibrinogen in young and old healthy adults. *Circulation*. 1991;83:1692–1697.
- Kestin AS, Ellis PA, Barnard MR, et al. Effect of strenuous exercise on platelet activation state and reactivity. *Circulation*. 1993;88:1502–1511.
- Miller VM, Vanhoutte PM. Enhanced release of endothelium-derived factor(s) by chronic increases in blood flow. *Am J Physiol*. 1988;255: H446–H451.
- Meredith IT, Yeung AC, Weidinger FF, et al. Role of impaired endothelium-dependent vasodilation in ischemic manifestations of coronary artery disease. *Circulation*. 1993;87:V-56–V-66.
- Charo S, Gokce N, Vita JA. Endothelial dysfunction and coronary risk reduction. J Cardiopulm Rehabil. 1998;18:60–67.
- 166. Hambrecht R, Wolf A, Gielen S, et al. Effect of exercise on coronary endothelial function in patients with coronary artery disease. N Engl J Med. 2000;342:454–460.
- Goldsmith RL, Bigger JT Jr, Steinman RC, et al. Comparison of 24-hour parasympathetic activity in endurance-trained and untrained young men. J Am Coll Cardiol. 1992;20:552–558.
- Coats AJ. Exercise rehabilitation in chronic heart failure. J Am Coll Cardiol. 1993;22:172A–177A.
- Malfatto G, Facchini M, Sala L, et al. Effects of cardiac rehabilitation and beta-blocker therapy on heart rate variability after first acute myocardial infarction. *Am J Cardiol.* 1998;81:834–840.
- Leon AS, Connett J, Jacobs DR Jr, et al. Leisure-time physical activity levels and risk of coronary heart disease and death: the Multiple Risk Factor Intervention Trial. *JAMA*. 1987;258:2388–2395.
- Paffenbarger RS Jr, Wing AL, Hyde RT, et al. Physical activity and incidence of hypertension in college alumni. *Am J Epidemiol*. 1983;117: 245–257.
- 172. Blair SN, Goodyear NN, Gibbons LW, et al. Physical fitness and incidence of hypertension in healthy normotensive men and women. *JAMA*. 1984;252:487–490.
- 173. Pescatello LS, Fargo AE, Leach CN Jr, et al. Short-term effect of dynamic exercise on arterial blood pressure. *Circulation*. 1991;83: 1557–1561.
- 174. Kokkinos PF, Narayan P, Colleran JA, et al. Effects of regular exercise on blood pressure and left ventricular hypertrophy in African-American men with severe hypertension. *N Engl J Med.* 1995;333:1462–1467.
- 175. Fagard RH. Prescription and results of physical activity. J Cardiovasc Pharmacol. 1995;25:S20–S27.
- Kelley GA, Kelley KS. Progressive resistance exercise and resting blood pressure: a meta-analysis of randomized controlled trials. *Hypertension*. 2000;35:838–843.
- 177. Kelley GA. Aerobic exercise and resting blood pressure among women: a meta-analysis. *Prev Med.* 1999;28:264–275.
- Kokkinos PF, Papademetriou V. Exercise and hypertension. Coron Artery Dis. 2000;11:99–102.
- 179. Wasserman DH, Zinman B. Fuel homeostasis. In: Ruderman N, Devlin JT, eds. *The Health Professional's Guide to Diabetes and Exercise*. Alexandria, Va: American Diabetes Association; 1995;29–47.
- Klem ML, Wing RR, McGuire MT, et al. A descriptive study of individuals successful at long-term maintenance of substantial weight loss. Am J Clin Nutr. 1997;66:239–246.
- Schoeller DA, Shay K, Kushner RF. How much physical activity is needed to minimize weight gain in previously obese women? *Am J Clin Nutr.* 1997;66:551–556.
- Blair SN. Evidence for success of exercise in weight loss and control. Ann Intern Med. 1993;119:702–706.
- 183. Wood PD, Stefanick ML, Williams PT, et al. The effects on plasma lipoproteins of a prudent weight-reducing diet, with or without exercise, in overweight men and women. *N Engl J Med.* 1991;325:461–466.
- 184. Troisi RJ, Heinold JW, Vokonas PS, et al. Cigarette smoking, dietary intake, and physical activity: effects on body fat distribution: the Normative Aging Study. Am J Clin Nutr. 1991;53:1104–1111.
- Tran ZV, Weltman A. Differential effects of exercise on serum lipid and lipoprotein levels seen with changes in body weight: a meta-analysis. *JAMA*. 1985;254:919–924.

- 186. King AC, Haskell WL, Young DR, et al. Long-term effects of varying intensities and formats of physical activity on participation rates, fitness, and lipoproteins in men and women aged 50 to 65 years. *Circulation*. 1995;91:2596–2604.
- Williams PT. High-density lipoprotein cholesterol and other risk factors for coronary heart disease in female runners. *N Engl J Med.* 1996;334: 1298–1303.
- Stefanick ML, Mackey S, Sheehan M, et al. Effects of diet and exercise in men and postmenopausal women with low levels of HDL cholesterol and high levels of LDL cholesterol. *N Engl J Med.* 1998;339:12–20.
- American Diabetes Association. Diabetes mellitus and exercise. Diabetes Care. 1997;20:1908–1912.
- 190. American College of Sports Medicine Position Stand: the recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults. *Med Sci Sports Exerc.* 1998;30:975–991.
- Kannel WB, Gordon T, Sorlie P, et al. Physical activity and coronary vulnerability: the Framingham Study. *Cardiol Dig.* 1971;6:28.
- 192. Paffenbarger RS Jr, Hyde RT, Wing AL, et al. Physical activity, all-cause mortality, and longevity of college alumni. N Engl J Med. 1986;314:605–613.
- 193. Bijnen FC, Caspersen CJ, Feskens EJ, et al. Physical activity and 10-year mortality from cardiovascular diseases and all causes: the Zutphen Elderly Study. Arch Intern Med. 1998;158:1499–1505.
- Kushi LH, Fee RM, Folsom AR, et al. Physical activity and mortality in postmenopausal women. JAMA. 1997;277:1287–1292.
- 195. Hakim AA, Petrovitch H, Burchfiel CM, et al. Effects of walking on mortality among nonsmoking retired men. N Engl J Med. 1998;338: 94–99.
- 196. Lee IM, Hsieh CC, Paffenbarger RS Jr. Exercise intensity and longevity in men: the Harvard Alumni Health Study. JAMA. 1995;273: 1179–1184.
- 197. LaCroix AZ, Leveille SG, Hecht JA, et al. Does walking decrease the risk of cardiovascular disease hospitalizations and death in older adults? *J Am Geriatr Soc.* 1996;44:113–120.
- 198. Siscovick DS, Weiss NS, Fletcher RH, et al. The incidence of primary cardiac arrest during vigorous exercise. N Engl J Med. 1984;311: 874–877.
- Paffenbarger RS Jr, Wing AL, Hyde RT. Physical activity as an index of heart attack risk in college alumni. Am J Epidemiol. 1978;108:161–175.
- Paffenbarger RS, Hale WE. Work activity and coronary heart mortality. N Engl J Med. 1975;292:545–550.
- Haskell WL. Health consequences of physical activity: understanding and challenges regarding dose-response. *Med Sci Sport Exerc.* 1994;26: 649–660.
- Haskell WL. Cardiovascular complications during exercise training of cardiac patients. *Circulation*. 1978;57:920–924.
- Van Camp SP, Peterson RA. Cardiovascular complications of outpatient cardiac rehabilitation programs. JAMA. 1986;256:1160–1163.
- Burke AP, Farb A, Malcom GT, et al. Plaque rupture and sudden death related to exertion in men with coronary artery disease. *JAMA*. 1999; 281:921–926.
- Oldridge NB, Guyatt GH, Fischer ME, et al. Cardiac rehabilitation after myocardial infarction: combined experience of randomized clinical trials. *JAMA*. 1988;260:945–950.
- O'Connor GT, Buring JE, Yusuf S, et al. An overview of randomized trials of rehabilitation with exercise after myocardial infarction. *Circulation*. 1989;80:234–244.
- 207. Haskell WL. The efficacy and safety of exercise programs in cardiac rehabilitation. *Med Sci Sports Exerc.* 1994;26:815–823.
- Vongvanich P, Paul-Labrador MJ, Merz CN. Safety of medically supervised exercise in a cardiac rehabilitation center. *Am J Cardiol.* 1996;77:1383–1385.
- Franklin BA, Bonzheim K, Gordon S, et al. Safety of medically supervised outpatient cardiac rehabilitation exercise therapy: a 16-year follow-up. *Chest.* 1998;114:902–906.
- 210. Mittleman MA, Maclure M, Tofler GH, et al. Triggering of acute myocardial infarction by heavy physical exertion: protection against triggering by regular exertion: Determinants of Myocardial Infarction Onset Study Investigators. N Engl J Med. 1993;329:1677–1683.
- 211. Willich SN, Lewis M, Lowel H, et al. Physical exertion as a trigger of acute myocardial infarction: Triggers and Mechanisms of Myocardial Infarction Study Group. N Engl J Med. 1993;329:1684–1690.
- 212. Tofler GH, Muller JE, Stone PH, et al. Modifiers of timing and possible triggers of acute myocardial infarction in the Thrombolysis in Myo-

cardial Infarction Phase II (TIMI II) Study Group. J Am Coll Cardiol. 1992;20:1049–1055.

- Lakka TA, Venalainen JM, Rauramaa R, et al. Relation of leisure-time physical activity and cardiorespiratory fitness to the risk of acute myocardial infarction. *N Engl J Med.* 1994;330:1549–1554.
- Balady GJ, Chaitman B, Driscoll D, et al. Recommendations for cardiovascular screening, staffing, and emergency policies at health/fitness facilities. *Circulation*. 1998;97:2283–2293.
- Fleg JL, Lakatta EG. Prevalence and significance of postexercise hypotension in apparently healthy subjects. *Am J Cardiol*. 1986;57: 1380–1384.
- Pollock MI, Wilmore JH. Exercise in Health and Disease: Evaluation and Prescription for Prevention and Rehabilitation. Philadelphia, Pa: Saunders; 1990.
- 217. Pollock ML, Franklin BA, Balady GJ, et al. Resistance exercise in individuals with and without cardiovascular disease: benefits, rationale, safety, and prescription: An advisory from the Committee on Exercise, Rehabilitation, and Prevention, Council on Clinical Cardiology, American Heart Association. *Circulation*. 2000;101:828–833.
- Feigenbaum MS, Pollock ML. Strength training: rationale for current guidelines for adult fitness programs. *Physician Sports Med.* 1997;25: 44-64.
- Kilbom A, Hartley LH, Saltin B, et al. Physical training in sedentary middle-aged and older men, I: medical evaluation. *Scand J Clin Lab Invest.* 1969;24:315–322.
- Pollock ML, Carroll JF, Graves JE, et al. Injuries and adherence to walk/jog and resistance training programs in the elderly. *Med Sci Sports Exerc.* 1991;23:1194–1200.
- 221. Elia EA. Exercise and the elderly. Clin Sports Med. 1991;10:141-155.
- 222. Brown M, Holloszy JO. Effects of a low intensity exercise program on selected physical performance characteristics of 60- to 71-year olds. *Aging (Milano)*. 1991;3:129–139.
- 223. King AC, Haskell WL, Taylor CB, et al. Group- vs home-based exercise training in healthy older men and women: a community-based clinical trial. *JAMA*. 1991;266:1535–1542.
- 224. Shephard RJ. Exercise and aging: extending independence in older adults. *Geriatrics*. 1993;48:61–64.
- Stewart AL, King AC, Haskell WL. Endurance exercise and healthrelated quality of life in 50–65 year-old adults. *Gerontologist.* 1993;33: 782–789.
- Emery CF, Hauck ER, Blumenthal JA. Exercise adherence or maintenance among older adults: 1-year follow-up study. *Psychol Aging*. 1992; 7:466–470.
- 227. Hassmen P, Ceci R, Backman L. Exercise for older women: a training method and its influences on physical and cognitive performance. *Eur J Appl Physiol*. 1992;64:460–466.
- King AC, Taylor CB, Haskell WL. Effects of differing intensities and formats of 12 months of exercise training on psychological outcomes in older adults. *Health Psychol.* 1993;12:292–300.
- Marcus BH, Simkin LR. The stages of exercise behavior. J Sports Med Phys Fitness. 1993;33:83–88.
- Barry HC, Eathorne SW. Exercise and aging: issues for the practitioner. Med Clin North Am. 1994;78:357–376.
- Courneya KS. Understanding readiness for regular physical activity in older individuals: an application of the theory of planned behavior. *Health Psychol.* 1995;14:80–87.
- 232. Rich MW, Palmeri S, McCluskey ER, et al. Calcium channel blockers for hypertension in older patients. *Cardiovasc Rev Rep.* 1991;12:11–14.
- Brown M, Holloszy JO. Effects of walking, jogging and cycling on strength, flexibility, speed and balance in 60-to 72-year olds. *Aging* (*Milano*). 1993;5:427–434.
- 234. McAuley E. Self-efficacy and the maintenance of exercise participation in older adults. *J Behav Med.* 1993;16:103–113.
- Rogers MA, Evans WJ. Changes in skeletal muscle with aging: effects of exercise training. *Exerc Sport Sci Rev.* 1993;21:365–379.
- 236. Franklin BA, Whaley MH, Howley ET, eds. ACSM's Guidelines for Exercise Testing and Prescription. Philadelphia, Pa: Lippincott Williams & Wilkins; 2000.
- 237. Williams MA, Maresh CM, Esterbrooks DJ, et al. Early exercise training in patients older than age 65 years compared with that in younger patients after acute myocardial infarction or coronary artery bypass grafting. *Am J Cardiol.* 1985;2000:55:263–266.
- Fiatarone MA, Marks EC, Ryan ND, et al. High-intensity strength training in nonagenarians: effects on skeletal muscle. *JAMA*. 1990;263: 3029–3034.

- Kasch FW, Boyer J, VanCamp SP, et al. The effect of physical activity and inactivity in aerobic power in older men. *Physician Sports Med.* 1990;18:73–83.
- Conn EH, Williams RS, Wallace AG. Exercise responses before and after physical conditioning in patients with severely depressed left ventricular function. *Am J Cardiol.* 1982;49:296–300.
- Gordon A, Tyni-Lenne R, Persson H, et al. Markedly improved skeletal muscle function with local muscle training in patients with chronic heart failure. *Clin Cardiol.* 1996;19:568–574.
- Wenger NK, Froehler ES, Smith LK. Cardiac Rehabilitation: Clinical Practice Guideline No. 17. Rockville, Md: Public Health Service; 1995.
- Lee AP, Ice R, Blessey R, et al. Long-term effects of physical training on coronary patients with impaired ventricular function. *Circulation*. 1979;60:1519–1526.
- Sullivan MJ, Higginbotham MB, Cobb FR. Exercise training in patients with severe left ventricular dysfunction: hemodynamic and metabolic effects. *Circulation*. 1988;78:506–515.
- 245. Coats AJ, Adamopoulos S, Radaelli A, et al. Controlled trial of physical training in chronic heart failure: exercise performance, hemodynamics, ventilation, and autonomic function. *Circulation*. 1992;85:2119–2131.
- 246. Shemesh J, Grossman E, Peleg E, et al. Norepinephrine and atrial natriuretic peptide responses to exercise testing in rehabilitated and nonrehabilitated men with ischemic cardiomyopathy after healing of anterior wall acute myocardial infarction. *Am J Cardiol.* 1995;75: 1072–1074.
- Minotti JR, Johnson EC, Hudson TL, et al. Skeletal muscle response to exercise training in congestive heart failure. J Clin Invest. 1990;86: 751–758.
- Wilson JR, Rayos G, Yeoh TK, et al. Dissociation between peak exercise oxygen consumption and hemodynamic dysfunction in potential heart transplant candidates. J Am Coll Cardiol. 1995;26: 429–435.
- Wilson JR, Graves J, Rayos G. Circulatory status and response to cardiac rehabilitation in patients with heart failure. *Circulation*. 1996; 94:1567–1572.
- Kiilavuori K, Sovijarvi A, Naveri H, et al. Effect of physical training on exercise capacity and gas exchange in patients with chronic heart failure. *Chest.* 1996;110:985–991.
- 251. Kobashigawa JA. The transplanted heart. In: Balady GJ, Pinã IL, eds. *Exercise and Heart Failure*. Armonk, NY: Futura, 1997;97–111.
- 252. von Scheidt W, Neudert J, Erdmann E, et al. Contractility of the transplanted, denervated human heart. Am Heart J. 1991;121: 1480–1488.
- Quigg RJ, Rocco MB, Gauthier DF, et al. Mechanism of the attenuated peak heart rate response to exercise after orthotopic cardiac transplantation. J Am Coll Cardiol. 1989;14:338–344.
- 254. Kobashigawa JA, Leaf DA, Lee N, et al. A controlled trial of exercise rehabilitation after heart transplantation. N Engl J Med. 1999;340: 272–277.
- 255. Savin WM, Gordon E, Green S. Comparison of exercise training effects in cardiac denervated and innervated humans. J Am Coll Cardiol. 1983; 1:772A. Abstract.
- Kavanagh T, Yacoub MH, Mertens DJ, et al. Cardiorespiratory responses to exercise training after orthotopic cardiac transplantation. *Circulation*. 1988;77:162–171.
- 257. Niset G, Hermans L, Depelchin P. Exercise and heart transplantation: a review. *Sports Med.* 1991;12:359–379.
- 258. American Association of Cardiovascular and Pulmonary Rehabilitation. Guidelines for Cardiac Rehabilitation and Secondary Prevention Programs: Promoting Health & Preventing Disease. 3rd ed. Champaign, Ill: Human Kinetics; 1999.
- Potempa K, Braun LT, Tinknell T, et al. Benefits of aerobic exercise after stroke. Sports Med. 1996;21:337–346.
- Monga TN, Deforge DA, Williams J, et al. Cardiovascular responses to acute exercise in patients with cerebrovascular accidents. *Arch Phys Med Rehabil.* 1988;69:937–940.
- Fletcher BJ, Dunbar S, Coleman J, et al. Cardiac precautions for non-acute inpatient settings. *Am J Phys Med Rehabil*. 1993;72:140–143.
- 262. Macko RF, DeSouza CA, Tretter LD, et al. Treadmill aerobic exercise training reduces the energy expenditure and cardiovascular demands of hemiparetic gait in chronic stroke patients: a preliminary report. *Stroke*. 1997;28:326–330.
- 263. Abbott RD, Rodriguez BL, Burchfiel CM, et al. Physical activity in older middle-aged men and reduced risk of stroke: the Honolulu Heart Program. *Am J Epidemiol*. 1994;139:881–893.

- 264. Shinton R, Sagar G. Lifelong exercise and stroke. BMJ. 1993;307: 231–234.
- The sixth report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure. Arch Intern Med. 1997;157:2413–2446.
- Fletcher BJ, Lloyd A, Fletcher GF. Outpatient rehabilitative training in patients with cardiovascular disease: emphasis on training method. *Heart Lung*. 1988;17:199–205.
- 267. Fletcher BJ, Thiel J, Fletcher GF. Phase II intensive monitored cardiac rehabilitation for coronary artery disease and coronary risk factors: a six-session protocol. Am J Cardiol. 1986;57:751–756.
- Fletcher GF, Chiaramida AJ, LeMay MR, et al. Telephonicallymonitored home exercise early after coronary artery bypass surgery. *Chest.* 1984;86:198–202.
- DeBusk RF, Haskell WL, Miller NH, et al. Medically directed at-home rehabilitation soon after clinically uncomplicated acute myocardial infarction: a new model for patient care. *Am J Cardiol.* 1985;55: 251–257.
- Shaw DK, Sparks KE, Jennings HS 3rd. Transtelephonic exercise monitoring: a review. J Cardiopulm Rehabil. 1998;18:263–270.
- 271. Ades PA, Pashkow FJ, Fletcher G, et al. A controlled trial of cardiac rehabilitation in the home setting using electrocardiographic and voice transtelephonic monitoring. *Am Heart J.* 2000;139:543–548.
- Prochaska JO, Velicer WF, Rossi JS, et al. Stages of change and decisional balance for 12 problem behaviors. *Health Psychol.* 1994; 13:39–46.
- 273. Calfas KJ, Long BJ, Sallis JF, et al. A controlled trial of physician counseling to promote the adoption of physical activity. *Prev Med.* 1996;25:225–233.
- Simons-Morton DG, Calfas KJ, Oldenburg B, et al. Effects of interventions in health care settings on physical activity or cardiorespiratory fitness. *Am J Prev Med.* 1998;15:413–430.
- King AC, Blair SN, Bild DE, et al. Determinants of physical activity and interventions in adults. *Med Sci Sports Exerc.* 1992;24:S221–236.
- Haskell WL, Alderman EL, Fair JM, et al. Effects of intensive multiple risk factor reduction on coronary atherosclerosis and clinical cardiac events in men and women with coronary artery disease: the Stanford Coronary Risk Intervention Project (SCRIP). *Circulation*. 1994;89: 975–990.
- 277. Niebauer J, Hambrecht R, Schlierf G, et al. Five years of physical exercise and low fat diet: effects on progression of coronary artery disease. J Cardiopulm Rehabil. 1995;15:47–64.
- McAuley E, Courneya KS, Rudolph DL, et al. Enhancing exercise adherence in middle-aged males and females. *Prev Med.* 1994;23: 498–506.

- 279. King AC, Sallis JF, Dunn AL, et al. Overview of the Activity Counseling Trial (ACT) intervention for promoting physical activity in primary health care settings: Activity Counseling Trial Research Group. *Med Sci Sports Exerc.* 1998;30:1086–1096.
- Lomas J. Diffusion, dissemination, and implementation: who should do what? Ann N Y Acad Sci. 1993;703:226–235; discussion 235–227.
- Logsdon DN, Lazaro CM, Meier RV. The feasibility of behavioral risk reduction in primary medical care. Am J Prev Med. 1989;5:249–256.
- DeBusk RF, Miller NH, Superko HR, et al. A case-management system for coronary risk factor modification after acute myocardial infarction. *Ann Intern Med.* 1994;120:721–729.
- 283. Wilke NA, Sheldahl LM, Dougherty SM, et al. Baltimore Therapeutic Equipment work simulator: energy expenditure of work activities in cardiac patients. Arch Phys Med Rehabil. 1993;74:419–424.
- Sheldahl LM, Wilke NA, Tristani FE. Exercise prescription for return to work. J Cardiopulm Rehabil. 1985;5:565–575.
- Hellerstein HK, Friedman EH. Sexual activity and the postcoronary patient. Arch Intern Med. 1970;125:987–999.
- Vuori I, Makarainen M, Jaaskelainen A. Sudden death and physical activity. *Cardiology*. 1978;63:287–304.
- Gibbons LW, Dooper KH, Meyer BM, et al. The acute cardiac risk of strenuous exercise. JAMA. 1980;244:1799–1801.
- Thompson PD, Funk EJ, Carleton RA, et al. Incidence of death during jogging in Rhode Island from 1975 through 1980. JAMA. 1982;247: 2535–2538.
- Vander L. Cardiovascular complications of recreational physical activity. *Physic Sports Med.* 1982;10:89–98.
- Fletcher GF, Cantwell JD. Ventricular fibrillation in a medically supervised cardiac exercise program: clinical, angiographic, and surgical correlations. *JAMA*. 1977;238:2627–2629.
- Leach CN Jr, Sands MJ Jr, Lachman AD, et al. Cardiac arrest during exercise training after myocardial infarction. *Conn Med.* 1982;46: 239–243.
- 292. Mead WF, Pyfer HR, Thrombold JC, et al. Successful resuscitation of two near simultaneous cases of cardiac arrest with a review of fifteen cases occurring during supervised exercise. *Circulation*. 1976;53: 187–189.
- 293. Hossack KF, Hartwig R. Cardiac arrest associated with supervised cardiac rehabilitation. J Cardiac Rehabil. 1982;2:402–408.

KEY WORDS: AHA Scientific Statements ■ exercise ■ oxygen ■ coronary disease ■ risk factors ■ stress