



**AMERICAN COLLEGE  
of SPORTS MEDICINE®**

POSITION STAND

# Exercise and Hypertension

This pronouncement was written for the American College of Sports Medicine by Linda S. Pescatello, Ph.D., FACSM, (Co-Chair), Barry A. Franklin, Ph.D., FACSM, (Co-Chair), Robert Fagard, M.D., Ph.D., FACSM, William B. Farquhar, Ph.D., George A. Kelley, D.A., FACSM, and Chester A. Ray, Ph.D., FACSM

## SUMMARY

Hypertension (HTN), one of the most common medical disorders, is associated with an increased incidence of all-cause and cardiovascular disease (CVD) mortality. Lifestyle modifications are advocated for the prevention, treatment, and control of HTN, with exercise being an integral component. Exercise programs that primarily involve endurance activity prevent the development of HTN and lower blood pressure (BP) in adults with normal BP and those with HTN. The BP lowering effects of exercise are most pronounced in people with HTN who engage in endurance exercise with BP decreasing approximately 5–7 mm Hg after an isolated exercise session (acute) or following exercise training (chronic). Moreover, BP is reduced for up to 22 h after an endurance exercise bout (e.g., postexercise hypotension), with the greatest decreases among those with the highest baseline BP.

The proposed mechanisms for the BP lowering effects of exercise include neurohumoral, vascular, and structural adaptations. Decreases in catecholamines and total peripheral resistance, improved insulin sensitivity, and alterations in vasodilators and vasoconstrictors are some of the postulated explanations for the antihypertensive effects of exercise. Emerging data suggest genetic links to the BP reductions associated with acute and chronic endurance exercise. Nonetheless, definitive conclusions regarding the mechanisms for the BP reductions following endurance exercise cannot be made at this time.

Individuals with controlled HTN and no CVD or renal complications may participate in an exercise program or competitive athletics, but should be evaluated, treated, and monitored closely. Preliminary peak or symptom-limited exercise testing may be warranted, especially for men over 45 and women over 55 yr planning a vigorous exercise program (i.e.,  $\geq 60\%$   $\dot{V}O_{2R}$ , oxygen uptake reserve). In the interim, while formal evaluation and management are taking place, it is reasonable for the majority of patients to begin moderate intensity exercise training ( $40\text{--}<60\%$   $\dot{V}O_{2R}$ ) such as walking. When pharmacologic therapy is indicated in physically active people it should, ideally: a) lower BP at rest and during exertion; b) decrease total peripheral resistance; and, c) not adversely affect exercise capacity. For these reasons, angiotensin converting enzyme (ACE) inhibitors (or angiotensin II receptor blockers in case of ACE inhibitor intolerance) and calcium channel blockers are currently the drugs of choice for recreational exercisers and athletes who have HTN.

Exercise remains a cornerstone therapy for the primary prevention, treatment, and control of HTN. The optimal training frequency, intensity, time, and type (FITT) need to be better defined to optimize the BP lowering capacities of exercise, particularly in children, women, older adults, and

certain ethnic groups. Based upon the current evidence, the following exercise prescription is recommended for those with high BP:

*Frequency:* on most, preferably all, days of the week

*Intensity:* moderate-intensity ( $40\text{--}<60\%$  of  $\dot{V}O_{2R}$ )

*Time:*  $\geq 30$  min of continuous or accumulated physical activity per day

*Type:* primarily endurance physical activity supplemented by resistance exercise

## INTRODUCTION

Since the 1970s, significant technological and pharmacotherapeutic advances have been made in the treatment and control of cardiovascular disease (CVD) and its associated risk factors. Yet, hypertension (HTN) remains a major public health problem in the United States, with 58.4 million (28.7%) Americans aged 18 yr or older having HTN (systolic blood pressure [SBP]  $\geq 140$  and/or diastolic blood pressure [DBP]  $\geq 90$  mm Hg) (11,39,107,137). HTN prevalence is increasing whereas awareness of the condition and control rates is suboptimal (39,107,137). The positive relationship between CVD risk and blood pressure (BP) occurs with a BP as low as 115/75 mm Hg and doubles for each 20/10-mm Hg increase. A person with normal BP at 55 yr of age has a 90% lifetime risk of developing HTN (264). The BP classification of “prehypertension” (SBP 120–139 or DBP 80–89 mm Hg) has been introduced to stress the public health importance of reducing BP and preventing HTN via healthy lifestyle interventions for all people (39).

There are minimal cost and side effects associated with lifestyle interventions, and they interact favorably with other CVD risk factors. For these reasons, the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (39,137), the World Health Organization (WHO) (283), the European Society of Hypertension (61), and the National High Blood Pressure Education Program (271) recommend approaches such as regular physical activity for the prevention and treatment of HTN. Table 1 lists the WHO blood pressure classification scheme and Table 2 the treatment guidelines for HTN (61,137,283).

The purpose of this Position Stand is to present an evidence-based review of the current state of knowledge on

0195-9131/04/3603-0533

MEDICINE & SCIENCE IN SPORTS & EXERCISE®

Copyright © 2004 by the American College of Sports Medicine

DOI: 10.1249/01.MSS.0000115224.88514.3A

TABLE 1. Blood pressure classification for adults aged 18 and older.\*, †, ‡, §, \*\* (61, 137, 283).

Blood Pressure Category	Systolic Blood Pressure (mm Hg)	Diastolic Blood Pressure (mm Hg)
Optimal	<120	and <80
Normal	120–129	and 80–84
High normal	130–139	or 85–89
Stage 1 hypertension	140–159	or 90–99
Stage 2 hypertension	160–179	or 100–109
Stage 3 hypertension	≥180	or ≥110

\* Produced from the National Heart, Lung, and Blood Institute publication titled, Sixth Report of the Joint Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI), Public Health Service, National Institutes of Health, National Heart, Lung, and Blood Institute, NIH Publication No. 98-4080, November 1997 (137).

† Not taking antihypertensive drugs and not acutely ill. When the systolic and diastolic blood pressure categories vary, the higher reading determines the blood pressure classification. For example, a reading of 152/82 mm Hg should be classified as Stage 1 hypertension and 170/116 mm Hg should be classified as Stage 3 hypertension. In addition to classifying stages of hypertension on the basis of average blood pressure levels, clinicians should specify presence or absence of target organ disease and additional risk factors. This specificity is important for risk classification and treatment (see Table 2).

‡ Optimal blood pressure with respect to cardiovascular risk is below 120/80 mm Hg. However, unusually low readings should be evaluated for clinical significance.

§ Based on the average of two or more readings at each of two or more visits after an initial screen.

\*\* This BP classification scheme has been altered in the Seventh Report of the Joint Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VII) to define normal blood pressure as <120 and <80 mm Hg, prehypertension 120–139 or 80–89 mm Hg, Stage 1 hypertension 140–159 or 90–99 mm Hg, and Stage 2 hypertension ≥160 or ≥100 mm Hg with lifestyle recommendations encouraged for those with normal BP and recommended for those with prehypertension and Stage 1 and 2 hypertension (39).

exercise and HTN with specific reference to human studies and essential HTN. The categories of evidence presented are those outlined by the National Heart, Lung, and Blood Institute (191) (Table 3). Because of the wealth of information on the cardioprotective effects of regular physical activity, we focus on the BP benefits of exercise in this Position Stand. Other benefits such as those on blood lipids-lipoproteins, insulin sensitivity, and body composition are not addressed in this document. Additional objectives of this Position Stand are to: 1) discuss the value and limitations of graded exercise testing in predicting future HTN and CVD morbidity and mortality, 2) address the role of acute (immediate effects of one bout of exercise) and chronic (long-term effects of a training program) endurance and resistance exercise on BP, 3) present exercise prescription recommen-

dations and special considerations for individuals with HTN, 4) describe potential physiologic mechanisms for the BP-lowering effects of acute and chronic exercise, and 5) summarize the current state of knowledge on exercise and HTN via an evidence based approach.

## EPIDEMIOLOGY OF HYPERTENSION

### Demographics

BP increases with age. SBP continues to increase throughout adult life, secondary to progressive arterial stiffening, whereas DBP plateaus in the sixth decade and decreases thereafter. Consequently, pulse pressure becomes increasingly greater with advancing age (142). In recent epidemiological studies, HTN was defined as SBP ≥ 140 and/or DBP ≥ 90 mm Hg, or being on antihypertensive treatment. The prevalence of HTN is estimated to be between 24 (30) and 29% (107) in the United States adult population. HTN increases with age and is higher among men than women at younger ages, but the reverse is true in older individuals. Isolated systolic HTN is rare before the age of 50 yr and becomes increasingly prevalent thereafter (142,245).

### HTN and CVD Morbidity and Mortality

HTN is associated with an increased incidence of all-cause and CVD mortality, stroke, coronary heart disease, heart failure, peripheral arterial disease, and renal insufficiency. More recently, Framingham Heart Study investigators (263) reported participants with high normal BP (SBP 130–139 or DBP 85–89 mm Hg) had higher rates of cardiovascular events as compared with those with optimal levels (SBP < 120 and DBP < 80 mm Hg). An important recent finding is the escalating evidence pulse pressure is an independent predictor of CVD morbidity and mortality, particularly in older subjects (15,90,93). Older patients with isolated systolic HTN appear to be at particularly high CVD risk (246).

Early randomized controlled trials have demonstrated salutary effects of antihypertensive drug therapy in patients

TABLE 2. Risk stratification and treatment.\*, † (137).

Blood Pressure Stages (mm Hg)	Risk Group A (No Risk Factors, No TOD/CCD)‡	Risk Group B (At Least 1 Risk Factor not Including Diabetes; No TOD/CCD)	Risk Group C (TOD/CCD and/or Diabetes, with or without Other Risk Factors)
High normal (130–139/85–89)	Lifestyle modification	Lifestyle modification	Drug therapyII
Stage 1 (140–159/90–99)	Lifestyle modification (up to 12 months)	Lifestyle modification§ (up to 6 months)	Drug therapy
Stage 2 and 3 (≥160/≥100)	Drug therapy	Drug therapy	Drug therapy

For example, a patient with diabetes and a blood pressure of 142/94 mm Hg plus left ventricular hypertrophy should be classified as having Stage 1 hypertension with target organ disease (left ventricular hypertrophy) and with another major risk factor (diabetes). This patient would be categorized as Stage 1, Risk Group C, and recommended for immediate initiation of pharmacologic treatment.

\* Produced from the National Heart, Lung, and Blood Institute publication titled, Sixth Report of the Joint Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI), Public Health Service, National Institutes of Health, National Heart, Lung, and Blood Institute, NIH Publication No. 98-4080, November 1997 (137).

† Lifestyle modification would be adjunctive therapy for all patients recommended for pharmacologic therapy.

‡ TOD/CCD indicates target organ disease/clinical cardiovascular disease including heart disease (left ventricular hypertrophy, angina/prior myocardial infarction, prior coronary revascularization and heart failure), stroke or transient ischemic attack, neuropathy, peripheral arterial disease, and retinopathy.

§ For patients with multiple risk factors, clinicians should consider drugs as initial therapy plus lifestyle modification. Major risk factors include smoking, dyslipidemia, diabetes mellitus, age >60 yr, gender (men and postmenopausal women), and family history of cardiovascular disease (women <65 yr or men <55 yr).

II For those with heart failure, renal insufficiency or diabetes.

TABLE 3. Evidence categories (191).

Evidence Category	Source of Evidence	Definition
A	Randomized controlled trials (overwhelming data)	Provides a consistent pattern of findings with substantial studies
B	Randomized controlled trials (limited data)	Few randomized trials exist which are small in size and results inconsistent
C	Nonrandomized trials, observational studies	Outcomes are from uncontrolled, nonrandomized and/or observational studies
D	Panel consensus judgment	Panel's expert opinion when the evidence is insufficient to place it in categories A through C

with elevated SBP and DBP (44). More recent trials have shown older patients with isolated systolic HTN also benefit from treatment (246). Drug therapy significantly reduced CVD mortality by 21% in patients with systolic-diastolic hypertension and by 18% in patients with isolated systolic hypertension. Risk reduction amounted to, respectively, 42% and 30% for fatal and nonfatal stroke, and 14% and 23% for coronary artery disease. It is not known whether BP reduction by lifestyle interventions would yield similar benefits regarding CVD events.

About 25% of patients with HTN in the clinic have normal BP by ambulatory monitoring or home assessment. The prognosis of white-coat HTN (or isolated clinic HTN) is better than that of sustained ambulatory HTN; however, it remains unclear whether the risk of white-coat HTN is similar to the risk of HTN in persons with normal BP in the clinic or with ambulatory monitoring (265).

Despite conclusive evidence antihypertensive therapy reduces the complications of HTN, only about half of all patients with HTN are under pharmacological treatment, and only a fraction of these have normal BP due to the insufficient implementation of contemporary guidelines (72,137,283). SBP appears to be more difficult to control than DBP.

## EXERCISE BLOOD PRESSURE AND THE PREDICTION OF HYPERTENSION AND CVD MORBIDITY AND MORTALITY

### Prediction of Future HTN

Accurate prediction of future HTN in persons with normal BP is important so that early preventive measures can be taken to potentially alter this outcome. Resting BP, family history of HTN, body mass index, and physical activity and fitness are generally accepted predictors of future HTN. Future HTN has also been reported to be associated with an exaggerated BP response during and/or after exercise.

In a population-based study of middle-aged normotensive men, Miyai et al. (181) reported a significant and independent threefold higher risk for incident HTN during a 4.7-yr follow-up period in those with a disproportionate exercise BP response. Contrasting results were reported by Manolio et al. (172) in a population-based sample of 18- to 30-yr-old men and women. Individuals with an exaggerated exercise BP response at baseline were 1.7 times more likely to develop HTN over the next 5 yr than were persons with a normal exercise BP response, but the association was no longer significant after multivariate regression analysis. In middle-aged normotensive subjects from the Framingham

Offspring Study (241) who were followed for 8 yr after baseline exercise testing, an exaggerated DBP but not SBP response to exercise was a significant and independent predictor of HTN in men and women, with odds ratios of 4.2 and 2.2, respectively. Matthews et al. (176) compared 151 cases of physician-diagnosed HTN with 201 age-matched controls who were normotensive. In multiple regression analysis, those who developed HTN at follow-up were three times more likely to have had an exaggerated exercise BP response. Several studies examined BP at variable time intervals in the immediate recovery period after exercise testing and found a higher BP after acute exercise significantly predicted future HTN (49,241). The prognostic power of exercise BP was also observed in studies in children (166).

Current studies do not seem to justify the widespread use of exercise testing to predict future HTN because of a number of limitations including: exercise tests and the definition of an exaggerated BP response were not standardized across the various studies; confounding variables were not always adequately accounted for in the analyses; and non-invasive BP measurements during exercise have inherent limitations, particularly with regard to DBP. However, when exercise testing is performed for other reasons, BP measurements may provide useful prognostic information.

**Evidence statement.** An abnormal or exaggerated exercise BP contributes to the prediction of future HTN in persons with normal BP. *Evidence category C.*

### Prediction of CVD Complications

Few studies assessed the significance of exercise BP for mortality or the incidence of CVD events. In healthy men, the exercise-induced increase in SBP from baseline to 164 W during cycle ergometer exercise independently and significantly predicted mortality from CVD, non-CVD and total mortality (75), and submaximal SBP at a work load of 100 W, but not maximal SBP, contributed independently to the prediction of CVD mortality and myocardial infarction (186,187). In another study, maximal SBP during a progressive exercise test to volitional fatigue predicted all-cause and CVD mortality in men and women (159).

On the other hand, exercise BP did not significantly enhance the prognostic value of resting BP in hypertensive men (65), but exercise systemic vascular resistance added prognostic precision to vascular resistance at rest, most likely due to attenuated arterial dilatation during exercise as a result of structural vascular abnormalities in those with worse prognosis. The impaired vasodilation was not expressed in an abnormal rise in BP because of a blunted rise of cardiac output. The crucial role of cardiac output and



cardiac function is highlighted by the fact exertional hypotension is associated with a worse prognosis in cardiac patients and in persons with chronic heart failure, probably due to left ventricular dysfunction (68,131,199).

In conclusion, the prognostic importance of exercise BP depends on the population studied. A worse prognosis is associated with a hypertensive response in healthy subjects and a hypotensive response in patients with CVD and/or heart failure, whereas the results may be variable in hypertensive patients depending on cardiac function and the associated cardiac output (66).

**Evidence statement.** The prognostic value of exercise BP regarding CVD complications depends on the underlying clinical status and hemodynamic response and is therefore limited. *Evidence category D.*

## EXERCISE AND BLOOD PRESSURE BENEFITS

### Endurance Training (Chronic) Effects

**Exercise training and the prevention of HTN.** The associations between various types of physical activity and the incidence of HTN have been assessed in a number of different populations. In male university alumni, Paffenbarger et al. (200,201) reported vigorous exercise in the postcollege years protected against future HTN. Total amount and intensity of baseline physical activity were inversely associated with the risk of future HTN in middle-aged Finnish men (101). In Japanese men, duration of walk-to-work and leisure-time physical activity was significantly associated with a reduction in the risk for incident HTN (119). In the Atherosclerosis Risk in Communities Study, the incidence of HTN was lower in white men in the highest quartile of leisure activity as compared with men in the least active quartile (205). In contrast, none of the studies in women observed significant and independent relationships between the level of physical activity and the risk of developing HTN (83,101,205). Moreover, physical activity was not associated with incident HTN in the only study in blacks (205).

Others have investigated relationships between measured physical fitness and incident HTN. Blair et al. (18) reported persons with low physical fitness had a relative risk of 1.5 for the development of HTN when compared with highly fit persons, after controlling for age, sex, body mass index, and BP. Sawada et al. (231) confirmed in Japanese men the relative risk of HTN, after adjusting for age, initial BP, body fat, and other confounders, was 1.9 times higher in the least fit compared with the most fit group.

**Evidence statement.** Higher levels of physical activity and greater fitness at baseline are associated with a reduced incidence of HTN in white men, and these associations persist after appropriate multivariate analysis. *Evidence category C.* The few studies in women and the one study in black subjects did not show significant relationships; however, the paucity of data

precludes definitive conclusions regarding the role of sex and ethnicity.

**Exercise training in the treatment and management of HTN.** Many longitudinal studies have assessed the effect of aerobic training on BP in adults, but essential scientific methods have not always been followed (67). Inclusion of a control group or period is mandatory, because BP may decrease over time due to the regression to the mean and habituation to measurement conditions. Allocation to the active or control group or the order of the training and nontraining phases should be randomly determined. Therefore, only randomized controlled studies are considered for the current overview. All but a few investigations reported on resting BP; however, in most studies BP was not measured by a blinded observer or an automated device. Accordingly, data on ambulatory BP are of prime importance.

**Resting BP.** After publication of the 1993 American College of Sports Medicine (ACSM) Position Stand on physical activity, fitness, and HTN (6), the effect of endurance training on resting BP has been addressed in a number of meta-analyses of randomized controlled trials (64,69–71,108,145–147,149–151,272). Most of the study participants were men, and the average age of the various study groups ranged from 18 to 79 yr (median ~45 yr). Duration of training involved 4–52 wk (median ~16 wk). Training frequency ranged from one to seven sessions per week, but about two-thirds of the training programs involved three weekly sessions. With few exceptions, each exercise session lasted from 30 to 60 min (median ~40 min). Exercises included walking, jogging, and running in about two-thirds of the studies and cycling in about half; several studies applied other aerobic exercises. Average training intensity in the various groups ranged from ~30 to 90% of maximal oxygen uptake ( $\dot{V}O_{2max}$ ) reserve ( $\dot{V}O_{2R}$ ). All meta-analyses concluded BP decreases significantly in response to exercise training. However, the effect of training on BP was quite variable among individual studies, which may reflect differences in baseline BP, demographic characteristics, characteristics of the training program, inadequate controls, and BP assessment limitations.

In recent meta-analyses, which included 29 (108), 44 (69,71), and 54 (272) randomized controlled trials irrespective of the baseline BP of the participants, the training-mediated decreases of SBP/DBP averaged 4.7/3.1 mm Hg (108), 3.4/2.4 mm Hg (69,71), and 3.8/2.8 mm Hg (272), respectively. In 16 of 68 study groups in which average baseline BP was in the hypertensive range (SBP  $\geq$  140 or DBP  $\geq$  90 mm Hg) (137,283), the weighted net BP decrease after adjustment for control observations and weighting for study size, was significant and averaged 7.4/5.8 mm Hg (71); the BP reduction was also significant and averaged 2.6/1.8 mm Hg in the 52 study groups in which baseline BP was normal, irrespective of antihypertensive therapy. Kelley et al. (149) found significantly greater absolute reductions in resting SBP/DBP in hypertensive (6/5 mm Hg) versus normotensive (2/1 mm

Hg) adults; and when normotensive and hypertensive subjects followed the same training program, the BP decrease was greatest in the hypertensives (70). With regard to the characteristics of the training program, there were only minimal or no effects of exercise frequency, type, and duration of training on the BP response in these meta-analyses (71,108,146,149,272). Finally, it was concluded there was no influence of exercise intensity (71,108,272) or that a lower intensity was associated with a larger reduction in DBP (149).

**Ambulatory BP.** Among randomized controlled trials, 11 studies applied ambulatory BP monitoring to assess the effect of exercise training (23,46,47,87,135,136,173,215,255,260,275). Six reported average 24 h BP, nine average daytime BP from early morning to late evening, and four nighttime BP. Because earlier analyses based on controlled and uncontrolled studies found nighttime BP is either not affected or much less influenced by exercise training (70,209), the current (unpublished) analysis of the randomized controlled trials is based on daytime BP from nine studies and on 24-h BP in the three studies that did not report day ambulatory BP. Baseline SBP/DBP averaged 135/86 mm Hg. The exercise-induced weighted net reduction in BP was significant and averaged 3.0/3.2 mm Hg, respectively.

**Exercise BP.** In eight randomized controlled trials (47,111,173,189,215,260,275,282), BP was measured during cycle ergometer exercise at a median work load of 100 W (range 60–140 W). BP was assessed during treadmill exercise at an energy expenditure of ~4 metabolic equivalents (METs) in two other studies (22,190). Pre-training exercise SBP averaged 180 mm Hg and heart rate 124 bpm. The weighted net training-mediated decrease in SBP and heart rate were significant, corresponding to 7.0 mm Hg and 6.0 bpm, respectively.

**Evidence statements.** Dynamic aerobic training reduces resting BP in individuals with normal BP and in those with HTN. *Evidence category A.* The decrease in BP appears to be more pronounced in hypertensive than in normotensive subjects. *Evidence category B.* Aerobic training also reduces ambulatory BP and BP measured at a fixed submaximal work load. *Evidence category B.* Response differences among individual studies are incompletely explained by the characteristics of the exercise training programs, that is, frequency, intensity, time, and type. *Evidence category B.*

**Acute Endurance Effects (Postexercise Hypotension).** Krául and colleagues (163) were the first to report an immediate reduction in BP after dynamic exercise. Some 20 yr later, Fitzgerald (76) coincidentally discovered jogging generally reduced his elevated BP into normotensive ranges for 4–10 h after completing his run. Due to accumulating evidence of the immediate and sustained reductions in BP after a bout of endurance exercise, Kenney and Seals (153) termed the decrease in arterial BP below control levels after a session of dynamic exercise as *post-exercise hypotension* (PEH).

PEH occurs in normotensive (41,77,84,85,89,112,144,211,243) and hypertensive (16,28,40,80,81,102,112,115,144,163,170,204,207,208,214,228,229,243,250,267,276) young, middle aged, and older white men and women, with the greatest BP reductions seen in those with HTN (153,209,252). When BP is taken casually in the laboratory, SBP and DBP are reduced an average of 15 and 4 mm Hg, respectively, from a mean preexercise value of 147/94 mm Hg for several hours after the exercise session (209). These acute exercise-mediated decreases in BP are clinically significant, offering many hypertensive individuals the health related benefit of having their BP transiently lowered during the day when BP is typically at its highest levels.

The initial ACSM Position Stand on exercise and HTN commented briefly on PEH due to limited information (6). At that time, the magnitude of PEH appeared to be less in the few studies using ambulatory BP monitoring compared with those using casual BP measurements, yet the smaller decreases in BP could be of greater clinical significance because ambulatory BP is a more valid prognostic indicator of CVD (265). In addition, ambulatory monitoring includes multiple, serial measurements that better reflect the BP a person maintains during activities of daily living. Moreover, this technology eliminates many of the problems associated with clinic determinations such as terminal digit preference, observer bias, and the white-coat phenomenon (232). For these reasons, scientists and clinicians were urged to integrate ambulatory monitoring into future studies examining PEH.

As suspected, more recent investigations using ambulatory BP monitoring showed the average day SBP reductions to be approximately 5 mm Hg or 40% less than those reported by casual assessment (6,106,271); however, the reductions in ambulatory DBP were similar to casual values among white adults with HTN (209). PEH has now been found to persist for up to 22 h after an exercise bout (228). The variable that makes the largest contribution to the change in BP after exercise appears to be the preexercise value (153,209,252). Apparently, exercise works best for those who are in greatest need of its BP-lowering capabilities; however, future work is required to better characterize those who benefit most from exercise as antihypertensive therapy.

Investigations designed to compare the influence of different components of the exercise prescription on PEH, i.e., type, duration, and intensity, are lacking. Limited data suggest the manifestation of PEH is independent of these factors. PEH is a low-threshold event, with BP reductions occurring with exercise durations as short as 3 min (163) and exercise intensities as low as 40%  $\dot{V}O_{2\max}$  (207). Whether longer duration and/or more vigorous-intensity exercise would elicit PEH to a greater or lesser degree is unknown at this time. The immediacy by which PEH occurs suggests the hypotensive influence of dynamic exercise ascribed to endurance-training programs may be partially an acute phenomenon with the BP reductions accumulating as the training program continues (252).

Pescatello and Kulikowich (209) reviewed acute and chronic (exercise training) endurance exercise studies involving ambulatory BP monitoring to gain insight into why exercise does not lower BP in approximately 25% of individuals with HTN (106). The ambulatory BP monitoring exercise studies revealed important methodologic considerations that should be addressed in future research on PEH. These include: a large enough sample size to detect the smaller decreases in BP seen with ambulatory than casual BP monitoring, especially in persons with normal to high normal BP; integration of a baseline period to stabilize preexercise BP and a control session of rest to adjust for placebo effects; consistency in the timing of the BP measurements to account for diurnal variation; and multiple assessments of BP before experimentation, ideally with ambulatory BP monitoring, to account for the labile nature of HTN in the determination of BP status.

**Evidence statement.** Dynamic exercise acutely reduces BP among people with HTN for a major portion of the daytime hours. *Evidence category B.* Few data exist on modulators of the acute exercise BP response including various components of the exercise prescription, age, ethnicity, and sex; thus, definitive conclusions cannot be made at this time.

## Resistance Exercise

**Training effects (chronic concentric and eccentric exercise).** Randomized controlled trials examining the effects of chronic concentric and eccentric resistance training on resting BP in adults have resulted in conflicting findings (14,23,31,45,56,58,116,143,185,256,261). A recent meta-analysis that included 320 male and female subjects (182 exercise, 138 control) also examined the effects of chronic resistance training on resting SBP and DBP in normotensive and hypertensive adults (148). In general, the resistance training programs followed the guidelines recommended by the ACSM (9). Statistically significant decreases of approximately 3 mm Hg were found for both resting SBP and DBP across all BP categories as a result of progressive resistance training. These decreases were equivalent to reductions of approximately 2 and 4%, respectively, for resting SBP and DBP. Although these modest changes may not be important from a clinical standpoint, a reduction of as little as 3 mm Hg in average population SBP has been estimated to reduce coronary heart disease by 5–9%, stroke by 8–14%, and all-cause mortality by 4% (247,271). The lack of available data on the effects of resistance training on ambulatory BP warrants further investigation because this may be more indicative of future CVD morbidity and mortality (265).

**Evidence statement.** Resistance training performed according to ACSM guidelines reduces BP in normotensive and hypertensive adults. *Evidence category B.*

**Training effects (chronic static exercise).** To date, only two randomized controlled trials including adults with resting DBP between 80 and 90 mm Hg have ex-

amined the effects of static (isometric) exercise training on resting SBP and DBP (277). In one trial, resting SBP was reduced by an average of approximately 13 mm Hg and resting DBP by approximately 15 mm Hg as a result of four, 2-min static handgrip contractions performed 3 d·wk<sup>-1</sup> for 8 wk. Contractions were performed at 30% of maximal effort with a 3-min rest period between contractions. A second trial, consisting of four contractions at 50% of maximum effort and held for 45 s with a 1-min rest between contractions, was performed 5 d·wk<sup>-1</sup> for 5 wk. Statistically significant reductions of approximately 10 and 9 mm Hg were reported for resting SBP and DBP, respectively.

Two nonrandomized controlled trials have examined the effects of static exercise on resting SBP and DBP in normotensive and hypertensive adults (158,222). Kiveloff and Huber (158) reported significant reductions ranging from 16 to 43 mm Hg in resting SBP and 2 to 24 mm Hg in resting DBP as a result of 5–8 wk of static exercise (6-s contractions for all large muscle groups, 3× d<sup>-1</sup>, 5 d·wk<sup>-1</sup>) in hypertensive adults. No significant reductions were found for normotensives. Unfortunately, no control group data were reported for either normotensives or hypertensives (158). More recently, a well-controlled study by Ray and Carrasco (222) examined the effects of four, 3-min bouts of static handgrip exercise at 30% of maximal voluntary contraction performed 4× wk<sup>-1</sup> for 5 wk in normotensive adults. Statistically significant reductions of approximately 5 mm Hg were reported for resting DBP with no statistically significant reductions observed for resting SBP. Given the paucity of studies available, additional randomized controlled trials are needed to examine the effects of static exercise on both resting and ambulatory SBP and DBP in adults.

**Evidence statement.** The limited evidence available suggests static exercise reduces BP in adults with elevated BP. *Evidence category C.*

**Acute effects of resistance exercise.** We are aware of no randomized or nonrandomized controlled trials that have examined the acute effects of resistance exercise on BP in adults. However, three observational studies have examined the acute effects of resistance exercise on BP (126,196,227). Hill et al. (126) investigated the effects of 11–18 min of resistance exercise on postexercise BP. A statistically significant decrease in DBP was observed for up to 1 h after the session whereas no statistically significant reductions were found for resting SBP. O'Connor et al. (196) examined the effects of 30 min of resistance exercise on ambulatory BP in females for up to 2 h after the session. Although no statistically significant changes were found for resting DBP, a significant elevation in SBP was observed at 1 and 15 min after the 80%-intensity condition, and at 1 min after a 60%-intensity condition.

More recently, Roltsch et al. (227) examined the ambulatory BP response for up to 24 h after resistance exercise in sedentary, endurance-trained, and resistance-trained men and women. Ambulatory BP was also as-



sessed in the same subjects after 48 h of no exercise. The exercise session consisted of two sets of exercises on 12 machines with a 60-s rest period between sets. No statistically significant differences in ambulatory BP were observed among the groups.

**Evidence statement.** Limited evidence suggests that resistance exercise has little effect on BP for up to 24 h after the exercise session. *Evidence category C.*

### Acute Effects of Isometric Exercise

**Evidence statement.** There are currently no studies available to provide a recommendation regarding the acute effects of isometric exercise on BP in adults.

### Special Populations

**Older adults.** Randomized controlled trials dealing with the effects of chronic aerobic exercise on resting BP in normotensive and hypertensive adults 50 yr of age and older have led to conflicting results (21,45,48,74,96,103,111,154,197,213,223). However, a meta-analysis that focused specifically on the effects of aerobic exercise for reducing resting SBP and DBP in 802 normotensive and hypertensive male and female adults (563 exercise, 239 control) 50 yr of age and older found a statistically significant reduction of approximately 2 mm Hg (2%) for resting SBP and a nonsignificant reduction of approximately 1 mm Hg (1%) for resting DBP (151). Although these reductions in SBP may seem small from a clinical perspective, they are important for the general older adult population (247,271). Most of the studies followed the ACSM Guidelines for physical activity in older adults (10). Although few studies have examined the acute effects of endurance exercise in older adults, three reports have shown that PEH can occur for up to 22 h, regardless of the exercise intensity (102,228,250).

**Evidence statement.** Regular endurance exercise reduces BP in older adults; additionally, there is no evidence to support the notion that training-mediated BP alterations differ between younger and older adults. *Evidence category B.* Limited evidence also suggests PEH occurs in older adults. *Evidence category C.*

**Children and adolescents.** Randomized controlled trials studying the effects of chronic exercise on resting SBP and DBP in children and adolescents have led to conflicting results (20,25,59,62,73,91,92,100,113,139,169,262). A meta-analysis that included a total of 1266 normotensive and hypertensive male and female children and adolescents (649 exercise, 617 control) found nonsignificant decreases of approximately 1 (1%) and 2 (3%) mm Hg for resting SBP and DBP as a result of exercise training (152). No differences were found when data were partitioned according to whether the subjects participated primarily in aerobic versus progressive resistance training. In the few studies involving children and adolescents with HTN, no statistically significant differences were found for either resting SBP or DBP as result of training as well as when data were partitioned accord-

ing to whether the subjects were normotensive or hypertensive. Furthermore, the distinction between normotension and HTN is difficult because of the continuous increase in BP with age in children and adolescents as well as the different upper limits in various age categories. Although it is well established the prevalence of HTN is greater in adults versus children and adolescents, it is likely the escalating number of overweight and obese children and adolescents will lead to an increase in the prevalence of HTN in this cohort in the future. The former notwithstanding, defining HTN in children and adolescents is difficult given the increases in BP that occur as a result of increasing body size, particularly height (5).

Clearly, a need exists for additional research on the effects of exercise, including progressive resistance training, on resting BP in hypertensive children and adolescents. Finally, few exercise studies have utilized ambulatory BP monitoring in children and adolescents. One observational study examined ambulatory BP in black and white male and female adolescents, and reported increased fitness was associated with lower ambulatory BP, especially in black adolescents (117).

**Evidence statement.** The evidence to date does not support endurance and resistance training as a nonpharmacologic intervention for reducing BP in children and adolescents. *Evidence category B.*

**Sex.** Meta-analytic research limited to randomized controlled trials has found no significant differences between the resting BP responses of normotensive and hypertensive men and women to endurance (149,150) and resistance (148) training. Of the 24 studies examining PEH among persons with HTN, 10 involved men and women (28,40,81,102,112,170,214,229,266,267), 9 included men only (16,81,115,120,144,207,243,250,276), and 4 women only (121,204,208,210). Of these, Quinn (214) is the only investigator who directly compared the influence of sex on PEH and found men and women to be similar in their postexercise BP response. Although estrogen is known to modulate vascular reactivity (179), none of the investigations involving men and women accounted for menstrual cycle phase in their study designs. Future work comparing the BP response of women and men of all ages to acute and chronic endurance and resistance exercise with ambulatory BP monitoring is needed.

**Evidence statement.** Endurance exercise training reduces BP similarly in men and women. *Evidence category B.* Limited evidence suggests acute endurance exercise reduces BP similarly in white men and women. *Evidence category C.*

**Ethnicity.** HTN has an earlier age of onset, is most common, and is associated with greater CVD morbidity and mortality in black Americans as compared with other ethnic groups in the United States (11). Despite these alarming statistics, there are few studies examining the influence of acute (120,121,210) and chronic (2,57,162,198,272) endurance exercise on BP among

black Americans with HTN. A recent meta-analysis of randomized controlled trials examining the effects of aerobic exercise training on resting BP in normotensive and hypertensive adults reported black participants, when compared with white and Asian subjects, had greater decreases in resting SBP, whereas reductions in DBP were greater in Asians compared with whites and blacks (272). These results, however, were based on only six studies of Asians (124,125,197,230,258,270) and four studies of blacks (2,26,57,162). Headley et al. (121) and Pescatello et al. (210) directly compared the acute exercise BP response between black and white women with high normal to Stage 1 HTN and found PEH to be less in the black women. Because of the wide-reaching public health and medical treatment implications of these preliminary observations, future work is urgently needed to clarify if, how, and why ethnicity alters the BP response to endurance exercise, particularly among people of black and non-Hispanic white origin. Given the lack of research, a need also exists for studies examining ethnic differences in the BP response to acute and chronic progressive resistance exercise.

**Evidence statement.** Currently no convincing evidence exists to support the notion ethnic differences exist in the BP response to chronic (*Evidence category B*) and acute exercise (*Evidence category C*).

## EXERCISE RECOMMENDATIONS

**Evaluation.** The routine evaluation of a patient with HTN includes a thorough individual and family history, physical examination, screening tests for secondary causes, and assessment of major risk factors, target organ damage, and CVD complications (137,283). Based on the level of BP and the presence of risk factors, target organ damage, and clinical CVD, the patient is classified in risk group A, B, or C (Table 2) (137). The extent of a pretraining screening evaluation mainly depends on the intensity of the anticipated exercise (129) and on the patient's symptoms, signs, overall CVD risk, and clinical CVD.

In patients with HTN about to engage in hard or very hard exercise (intensity  $\geq 60\%$   $\dot{V}O_2R$ ), a medically supervised peak or symptom-limited exercise test with ECG monitoring may be warranted. In hypertensive men in the British Regional Heart Study (238), the long-term risk of major CVD events was reduced as patients were more active, up to moderately vigorous activity, but the risk increased again in vigorously active men, particularly in the presence of other risk factors (J-shaped curve). Peak or symptom-limited exercise testing is also indicated in patients with symptoms suggestive of CVD such as exertional dyspnea, chest discomfort, or palpitations.

In asymptomatic men or women in risk categories A or B (Table 2) and with BP  $< 180/110$  mm Hg (Table 1; Stage 1 or 2), who engage in light to moderate dynamic physical activity (intensity  $< 60\%$   $\dot{V}O_2R$ ), there is gen-

erally no need for further testing beyond the routine evaluation. Individual patients in risk category C without CVD, or with Stage 3 hypertension (BP  $\geq 180/110$  mm Hg), may benefit from exercise testing before engaging in moderate-intensity exercise ( $40\text{--}<60\%$   $\dot{V}O_2R$ ) but not for light or very light activity ( $<40\%$   $\dot{V}O_2R$ ).

In patients with documented CVD such as ischemic heart disease, heart failure or stroke, exercise testing is warranted and vigorous exercise training ( $\geq 60\%$   $\dot{V}O_2R$ ) is best initiated in dedicated rehabilitation centers under medical supervision, where according to clinical status, advice can be given on the continuation of medically supervised gymnasium or home-based exercise programs. Comorbid conditions such as diabetes, ischemic heart disease, and heart failure should be adequately controlled before the start of exercise training. In the interim, while formal evaluation and management are taking place, it is reasonable for the majority of patients to begin moderate-intensity exercise training ( $40\text{--}<60\%$   $\dot{V}O_2R$ ) such as walking. Finally, systematic follow up should be provided as outlined in the statement on Benefits and Recommendations for Physical Activity Programs for all Americans (78).

**Safety.** Although considerable epidemiological evidence suggests chronic exercise may help to protect against the development of CVD and its sequelae, cardiovascular events associated with exercise have been reported in the medical literature and the lay press, suggesting strenuous physical activity may actually precipitate acute myocardial infarction or cardiac arrest in selected individuals (33,248). Accordingly, the challenge for physicians and other healthcare professionals is to provide prescriptive guidelines that minimize risks and maximize benefits.

Pathophysiologic evidence suggests exercise, by increasing myocardial  $\dot{V}O_2$  and simultaneously shortening diastole and coronary perfusion time, may evoke a transient oxygen deficiency at the subendocardial level, which, in the presence of a compromised coronary circulation, may be highly arrhythmogenic (128). The notion strenuous physical activity can trigger acute myocardial infarction, particularly among persons with latent or known heart disease who are habitually sedentary, has also been substantiated by several studies (97,180,280). This may occur via abrupt increases in heart rate and SBP, induced coronary artery spasm in diseased artery segments, or the twisting of epicardial coronary arteries leading to disruption of vulnerable atherosclerotic plaque and thrombotic occlusion of a coronary vessel (225,251).

Factors that may affect the risk of physical exertion are age, the presence of coronary artery disease, and the intensity of exercise, which is directly related to the hemodynamic response and myocardial  $\dot{V}O_2$  (79). Several investigators have shown excellent correlations between the measured myocardial  $\dot{V}O_2$  and two of its major determinants: heart rate ( $r = 0.88$ ) and the heart rate times SBP product ( $r = 0.92$ ) (157,194). Thus, an excessive SBP response to exercise may contribute to isch-



emic cardiac events including acute myocardial infarction (128). Similarly, the potential for a hypertensive-induced cerebrovascular accident cannot be discounted.

The “normal” hemodynamic response to endurance exercise is a progressive increase in SBP, typically 8–12 mm Hg·MET<sup>-1</sup> (192). When comparing healthy men and women, men generally have higher SBP during maximal treadmill testing (~20 mm Hg), which may range from as low as 160 to more than 220 mm Hg (127), whereas DBP usually falls slightly or remains unchanged. In contrast, increased subendocardial perfusion, secondary to elevated DBP, may contribute to the lower incidence of ischemic responses when performing static-dynamic efforts versus dynamic efforts alone (17,50). Commonly used criteria for discontinuation of exercise testing include SBP and/or DBP values > 250 mm Hg and 115 mm Hg, respectively (95). It should be emphasized, however, that clinicians have arbitrarily established these values and that no data exist to support these endpoints. Moreover, there are virtually no reports of hypertensive-related cardiovascular complications that have resulted when subjects exceeded these levels. For endurance exercise training, it appears prudent to maintain even lower BP values (e.g., < 220/105 mm Hg).

**Exercise recommendations.** The optimal training frequency, intensity, time, and type (FITT) of exercise needed to lower BP remains unclear. The recommendations put forth by the Centers for Disease Control and Prevention and ACSM (202) for society at large (“*Every U.S. adult should accumulate 30 minutes or more of moderate-intensity physical activity on most, preferably all, days of the week*”) generally apply to individuals at risk for developing or those with HTN. Similarly, the recommendations promulgated in the 1996 Surgeon General’s report, *Physical Activity and Health* (257), are applicable as well. The specific recommendations outlined in the most recent ACSM Position Stand entitled “The Recommended Quantity and Quality of Exercise for Developing and Maintaining Cardiorespiratory and Muscular Fitness, and Flexibility in Healthy Adults” (9) are also generally relevant and—for the most part—suitable for those with HTN. When formulating the exercise FITT recommendations for this Position Stand, due consideration was given to the many recent thorough reviews on this topic (6,71,106,149,209,252,268,272).

**Frequency: (on most, preferably all, days of the week).** Training frequencies between 3 and 5 d·wk<sup>-1</sup> are effective in reducing BP (71,177). Although limited evidence suggests seven sessions may be more effective than three sessions per week (135,193), other data suggest that there is no association between frequency of weekly exercise and BP reductions (132). However, because a single bout of exercise can cause an acute reduction in BP that lasts many hours (e.g., PEH), augmenting or contributing to the reductions in BP resulting from exercise training (118,252), consideration should be given to daily or near-daily exercise.

**Intensity: (moderate-intensity physical activity).** Fagard (71) completed a meta-analysis of randomized controlled intervention trials in normotensive and hypertensive subjects and found the reductions in BP resulting from endurance exercise training conducted at intensities between 40% and 70% of  $\dot{V}O_{2R}$  were of similar magnitude (SBP 3.4/DBP 2.4 mm Hg). Hagberg et al. (106), in their review of human studies, concluded greater reductions in resting BP occur when training at less than 70%  $\dot{V}O_{2max}$  as compared with higher intensities among individuals with HTN. Earlier data using animal models of HTN are consistent with this conclusion (254). There are few data, however, on the effects of light (20–30% of  $\dot{V}O_{2R}$ ) and very hard ( $\geq 85\%$  of  $\dot{V}O_{2R}$ ) exercise on BP modulations (71). In summary, moderate-intensity exercise training appears effective in lowering BP acutely (85,86,102,207,209) and chronically (4,24,103,173,184, 235,272). Thus, the recommendation for those with HTN is regular participation in moderate-intensity endurance exercise, corresponding to 40–<60% of  $\dot{V}O_{2R}$  to maximize the benefits and minimize possible adverse effects of more vigorous exercise. This intensity range corresponds to approximately 12–13 on the Borg rating of perceived exertion (RPE) 6–20 scale (195). The reliance on RPE to monitor exercise intensity may be important for some, because the hemodynamic response to exercise may be altered by various antihypertensive medications, e.g., beta blockers. Although a vigorous exercise program may be appropriate in selected hypertensive patients, the risk of cardiovascular complications and orthopedic injuries is higher and adherence is lower with higher-intensity exercise programs (88).

**Time: (duration: 30 min or more continuous or intermittent exercise per day).** Randomized controlled trials to date have generally used continuous rather than intermittent exercise, with durations between 30 and 60 min per session (71). The reduction in resting BP resulting from endurance exercise training does not appear to differ for exercise durations within this range. Intermittent shorter bouts of activity may also elicit reductions in BP (118,188,252). Moreover, other health benefits may be derived when multiple bouts of physical activity are performed throughout the day (19,188). A recent study (132) suggests 30–60 min of aerobic exercise *per week* conducted at 50%  $\dot{V}O_{2max}$  in previously sedentary hypertensive adults is effective in reducing resting BP. Greater reductions in systolic BP were observed with 61–90 min of aerobic exercise *per week*, but further increases in exercise time per week did not cause additional reductions in BP. These results should be interpreted with caution, because this was a nonrandomized trial and information on exercise frequency was not provided. These data notwithstanding, the recommendation is for 30–60 min of continuous or intermittent exercise per day (minimum of 10-min intermittent bouts accumulated throughout the day to total 30–60 min of exercise).

**Type: (primarily aerobic activity supplemented by resistance exercise).** Most intervention trials (69,71,108,145–147,149,150,272) have used endurance exercises such as walking, jogging, running, or cycling as the exercise modality. However, *any* activity that uses large muscle groups, can be maintained continuously, and is rhythmical and aerobic in nature is recommended as the primary modality for those with HTN. Individual preference is an important factor to maximize long-term adherence. Resistance training is also an important component of a well-rounded exercise program. Although limited data suggest resistance training has a favorable effect on resting BP, the magnitude of the acute and chronic BP reductions are less than those reported for endurance exercise (148). The present recommendation is for resistance training to serve as an adjunct to an aerobic-based exercise program. The reader is referred to the recent American Heart Association statement on resistance training that discusses special considerations for those with and without CVD and other comorbid conditions (212). Other specific recommendations are beyond the scope of this Position Stand but can be found elsewhere (9,88,98).

### Special considerations

- Antihypertensive medications such as beta blockers and diuretics impair the ability to regulate body temperature during exercise in hot and/or humid environments and provoke hypoglycemia (88,206). Thus, people using these medications should be educated on the signs/symptoms of heat illness, the role of adequate hydration, proper clothing to facilitate evaporative cooling, the optimal times of the day to exercise, the importance of decreasing the exercise dosage (time and intensity) during periods of increased heat or humidity, and methods to prevent hypoglycemia. In addition, beta blockers can substantially alter submaximal and maximal exercise capacity, particularly in those without myocardial ischemia and with nonselective agents.
- Because antihypertensive agents such as alpha blockers, calcium channel blockers, and vasodilators may provoke hypotensive episodes after abrupt cessation of activity, extending the cool-down period is generally recommended (88,98).
- Many persons with HTN are overweight (BMI 25–29.9 kg·m<sup>-2</sup>) or obese (BMI ≥ 30 kg·m<sup>-2</sup>) (133,191,257). Therefore, an exercise program that emphasizes a daily caloric expenditure of more than 300 kcal, coupled with reductions in energy intake, should be recommended. This may be accomplished best with moderate-intensity, prolonged exercise, such as walking. The combination of regular exercise and weight loss should be effective in lowering resting BP (133,191,257). More specific guidelines for overweight and obese individuals with features of the metabolic syndrome are beyond the scope of this Position Stand and can be found elsewhere (3,8,191).

- Older persons appear to demonstrate similar increases in  $\dot{V}O_{2\max}$  (103,104,161,234) and reductions in BP with exercise training as young adults (10). Additional exercise recommendations for the older adult beginning an exercise program can be found in the ACSM Position Stand on this topic (10).
- Patient education regarding the importance of regular exercise for BP control and management may increase exercise adherence. Patients may be especially responsive if this information comes from their personal physician. Anecdotal evidence suggests knowledge of the immediate BP-lowering effects of exercise (i.e., PEH) may promote exercise adherence (210).
- Individuals with severe or uncontrolled BP should add exercise training only *after* physician evaluation and initiation of drug therapy.
- Other precautions or modifications may be necessary for selected patients, particularly higher risk patients with comorbidities such as coronary artery disease or chronic heart failure. For example, in the hypertensive patient with coronary artery disease, the above-referenced guidelines are still appropriate, but the intensity of training should be set safely below ( $\geq 10$  beats·min<sup>-1</sup>) the ischemic ECG or anginal threshold. Additional prescriptive information is beyond the scope of this Position Stand. The reader is referred to other authoritative documents regarding special considerations for those with coronary artery disease (7,79,278).

**Evidence statements.** For persons with high BP, an exercise program that is primarily aerobic-based (*Evidence category A*) with adjunctive resistance training (*Evidence category B*) is recommended. The evidence is limited regarding frequency, intensity, and time recommendations (*Evidence category C*) and special considerations for those with HTN (*Evidence category D*). Nonetheless, the antihypertensive effects of exercise appear to occur at a relatively low duration and intensity.

## MECHANISMS

### Potential Mechanisms for Reductions in BP after Endurance Exercise

**Chronic exercise.** Because mean arterial pressure (MAP) is determined by cardiac output and total peripheral resistance (TPR), reductions in arterial pressure after endurance exercise training must be mediated by decreases in one or both of these variables. Reductions in resting cardiac output do not typically occur after chronic exercise; thus, decreased TPR appears to be the primary mechanism by which resting BP is reduced after exercise training.

As derived from Poiseuille's law, TPR is directly proportional to blood viscosity and length of the vessel, but inversely proportional to the fourth power of the radius. Because the former are not significantly altered by training, reductions in TPR are primarily associated with

changes in vessel diameter. Accordingly, small changes in vessel diameter have a profound impact on vascular resistance. Reductions in vascular resistance after training are mediated by neurohumoral and structural adaptations, altered vascular responsiveness to vasoactive stimuli, or both. Reductions in the vasoconstrictor state of the peripheral vasculature by less sympathetic neural influence or greater local vasodilator influence (e.g., nitric oxide) are examples of neural and local changes that would reduce peripheral resistance and lower BP. Larger lumen diameter and greater distensibility of the vasculature are structural adaptations to training permitting lower peripheral resistance. Finally, genetic factors may contribute to BP adaptations after endurance exercise training.

## Neurohumoral Adaptations

**Sympathetic nervous system.** Elevated sympathetic nerve activity is a hallmark observation in essential HTN (1). Sympathetic nerve activity (SNA) and the subsequent release of norepinephrine (NE) mediate vasoconstriction and increase vascular resistance. Reductions in central sympathetic nerve outflow or circulating NE attenuate vasoconstriction and lead to reductions in BP. Currently, direct recordings of efferent SNA in humans by microneurography do not convincingly demonstrate reductions in central SNA at rest after exercise training in normotensive subjects (220–222). However, similar data are lacking in hypertensive subjects. Muscle SNA was elevated in hypertensive subjects as compared with normotensive subjects in some (82,99,174,175) but not all studies (183,269). Therefore, training may possibly reduce SNA in hypertensive subjects. Furthermore, studies indicate baroreflex control of SNA is enhanced by exercise training (29,240,242), therefore, providing one potential mechanism of lowering SNA. However, microneurography studies are limited only to nerve activity to skeletal muscle or skin, and reductions in SNA to other vascular beds (e.g., renal and splanchnic beds) may also occur.

Despite the limited evidence to support reductions in efferent SNA after training, reductions in plasma NE have been reported after training (134,178,193,258). Meredith et al. (178) found reductions in plasma NE after endurance training were related to decreased spillover and not increased clearance, suggesting a decrease in SNA. Recently, Brown et al. (27) reported training-induced decreases in BP in older mild hypertensive subjects were associated with reduced NE release rate. Less NE at the synapse would be one mechanism that would facilitate reductions in vascular resistance after training. Interestingly, Meredith et al. (178) reported that the reduction in total plasma NE spillover could be accounted for mainly by the decrease in NE spillover in the kidneys (70%). However, the associated fall in renal vascular resistance was insufficient to account for the BP-lowering effect of exercise training. These investigators sug-

gested that other effects associated with the inhibition of the renal sympathetic outflow may be important in reducing BP after training (e.g., decreased renin release).

Hyperinsulinemia and insulin resistance are associated with HTN and activation of the sympathetic nervous system (12,13,140). Because exercise training improves insulin sensitivity (122), this may be an important mechanism in mediating reductions in sympathetic outflow and BP. A recent study in hypertensive subjects demonstrated a close association between the reduction in resting BP and plasma NE and improved insulin sensitivity after exercise training (160). Training-induced muscle adaptations appear to be important in attenuating insulin mediated sympathetic activation.

Finally, elevated SNA has been associated with increases in arterial wall thickening (54). Therefore, training-induced decreases in SNA may be beneficial in preventing vascular remodeling that is associated with HTN.

**Renin-angiotensin system.** Because angiotensin II is a powerful vasoconstrictor and regulator of blood volume, reductions in renin and angiotensin II with training would likely be a contributor to reduced BP. In normotensive subjects, reduced resting levels of renin and angiotensin II after training have been reported (94,134). However, in hypertensive subjects, exercise training does not consistently reduce plasma renin (103,193,258) and angiotensin II levels (63,123). Thus, current evidence suggests that the renin-angiotensin system does not appreciably contribute to the lowering of BP after training.

**Vascular responsiveness.** Vascular adaptations are likely to contribute to lower BP after training. Vascular responsiveness to  $\alpha$ -adrenergic receptor stimulation by NE is attenuated after training (38,52,244,273). Chronic exercise has been shown to decrease  $\alpha$ -adrenergic vasoconstriction in spontaneously hypertensive rats (37). Endothelin-1, which is a potent vasoconstrictor, has been demonstrated to elicit greater vasoreactivity in HTN (34,233,249). Training also lowers endothelin-1 levels in humans (171). Animal studies have reported reduced vascular sensitivity to endothelin-1 after training (138,168). Thus, exercise training alters the vascular responsiveness to two potent vasoconstrictors, NE and endothelin-1.

HTN impairs endothelial function. Associated with endothelial dysfunction are greater vascular tone and less vasodilator function. Endothelial-dependent vasodilation is critically dependent upon the production of nitric oxide. Exercise training has been shown to increase nitric oxide production and improve vasodilatory function in healthy subjects (155,156). Higashi et al. (124,125) reported reactive hyperemia and an enhanced forearm blood flow response to acetylcholine infusion were enhanced after 12 wk of walking in subjects with essential HTN that showed reductions in resting BP. However, endothelial-independent vasoconstriction remained unchanged. These findings are consistent with animal studies using normotensive (51,53) and spontaneous hyper-



tensive rats (284). Collectively, these findings indicate improved endothelial-dependent mediated vasodilation is an important training adaptation to reduce peripheral resistance in HTN.

**Structural adaptations.** Considerable evidence suggests changes in vascular structure occur in muscle in response to exercise training (167). These include vascular remodeling (i.e., increased length, cross-sectional area, and/or diameter of already existing arteries and veins) and angiogenesis (i.e., new vessel growth). Currently, few data are available concerning the effects of training on the size or number of small arteries and arterioles. Arteriolar density is greater in spinotrapezius muscle in endurance trained rats (165). However, the most convincing evidence in this regard is the decreased precapillary vascular resistance of hindquarters isolated from trained normotensive (236,237) and spontaneously hypertensive rats (60). Therefore, the training-induced alterations in vascular structure (i.e., increased muscle precapillary vessel number) that elevate the total cross-sectional area of resistance vessel lumina suggest a possible mechanism to lower peripheral resistance and reduce resting BP.

Cross-sectional data indicate endurance trained subjects have larger arterial lumen diameter in conduit arteries than untrained controls (130,239,274). A recent longitudinal training study confirmed these findings (55); moreover, intima-media thickness and the intima-media thickness/lumen ratio were reduced. Similarly, both cross-sectional and longitudinal studies have demonstrated greater arterial compliance after training in normotensive subjects (32,182,259). However, short-term aerobic training does not appear to increase large artery compliance in isolated systolic hypertensive subjects (74). In summary, training-induced vascular remodeling may contribute to the antihypertensive effect of exercise, but additional confirmatory studies are needed.

**Genetic influences.** The antihypertensive effects of exercise training may also be associated with genetic factors. Genetic influences on BP adaptations have been observed both at rest and during exercise. Much of these data have been generated by the HERITAGE Family Study (281). Rice et al. (224) suggested genetic factors account for approximately 17% of the reduction in resting SBP after exercise training. A number of genes were identified that may contribute to this response. Rankinen and colleagues (218) examined the associations between NOS3-Glu298Asp variants (gene responsible for nitric oxide synthase) and changes in BP at rest and during submaximal exercise after an endurance training program in 471 normotensive men and women from the HERITAGE Family Study. Although there were no associations between NOS3-Glu/Asp single nucleotide polymorphisms and resting BP, individuals with the NOS3-Asp allele reduced DBP less during submaximal exercise than those homozygous for the NOS3-Glu allele. These investigators also demonstrated an association between angiotensinogen, angiotensin-converting enzyme, TGF- $\beta_1$

gene polymorphisms, and physical training on resting and exercise BP (216,217,226). Because these studies were conducted in normotensive subjects, differing responses may be observed in hypertensive individuals. Accordingly, Hagberg et al. (105) reported that hypertensive subjects with different alleles for apoE gene had different training-induced changes in resting BP.

In summary, although there appears to be a genetic component to BP adaptations to exercise training, the overall contribution seems small. The reasons for these weak associations may be due to the complexities of BP regulation in normal and diseased states, unidentified genetic interactions between multiple loci and environmental factors, or both (279). Future studies should provide greater insight in this burgeoning area of study.

**Acute exercise.** The exact mechanism for PEH is unclear and is most likely multifactorial. With one exception (i.e., older hypertensive subjects) (102), studies suggest that the acute decrease in BP is related to reductions in peripheral resistance rather than cardiac output. Two prominent mechanisms have been proposed to explain the decrease in peripheral resistance: sympathetic inhibition and altered vascular responsiveness after exercise. Reductions in sympathetic outflow after exercise have been reported in both animals and humans (80,109,164). How sympathetic outflow is reduced by acute exercise remains unclear. It appears the arterial and cardiopulmonary baroreflexes are important for PEH (35,42). Chandler and DiCarlo (35) reported sinoaortic denervation prevented PEH in SHR rats. After acute exercise, PEH is associated with a resetting of the operating point of the arterial baroreflex to a lower BP (109). Recent studies of the central nervous system have shown an augmented GABA<sub>A</sub> signaling at the rostral ventrolateral medulla may contribute to decreased sympathetic outflow during PEH (141). Additionally, substance P receptors in the nucleus tractus solitarius and central vasopressin V<sub>1</sub> receptors have also been shown to contribute to PEH (36,43). Although it was formerly believed activation of the endogenous opioid system contributed significantly to reductions in sympathetic outflow and PEH (253), more recent studies in humans have questioned the importance of this mechanism (114).

Changes in vascular responsiveness related to PEH are associated with decreased transduction of sympathetic outflow to vascular resistance and the release of local vasodilator substances induced by muscle contraction and augmented muscle blood flow (e.g., nitric oxide released). After acute exercise, vascular responsiveness to  $\alpha$ -adrenergic stimulation is blunted (109,203). This response alone would facilitate vasodilation and reductions in peripheral resistance. Local release of nitric oxide, prostaglandins, adenosine, and ATP is augmented during exercise and would also facilitate peripheral vasodilation after acute exercise (110). Rao et al. (219) reported the reduced  $\alpha_1$ -adrenergic responsiveness after exercise in male spontaneously hypertensive rats may be attributed to nitric oxide. This study provides an example of the multitude of interactions that may occur

TABLE 4. American College of Sports Medicine exercise and hypertension Position Stand evidence statements.

Section Heading	Evidence Statement	Evidence Category*
Exercise BP and the prediction of hypertension and CVD morbidity and mortality	<ul style="list-style-type: none"> <li>● Abnormal or exaggerated exercise BP contributes to the prediction of future HTN in persons with normal BP.</li> <li>● The prognostic value of exercise BP regarding CVD complications depends on the underlying clinical status and hemodynamic response and is therefore limited.</li> </ul>	C D
Exercise BP benefits	<ul style="list-style-type: none"> <li>● Higher levels of physical activity and greater fitness at baseline are associated with a reduced incidence of HTN in white men; however, the paucity of data precludes definitive conclusions regarding the role of sex and ethnicity.</li> <li>● Dynamic aerobic training reduces resting BP in individuals with normal BP and in those with HTN.</li> <li>● The decrease in BP with aerobic training appears to be more pronounced in those with HTN.</li> <li>● Aerobic training reduces ambulatory BP and BP measured at a fixed submaximal work load.</li> <li>● BP response differences among individual studies are incompletely explained by the characteristics of the training programs, that is, the weekly exercise frequency, intensity, time and type of exercise.</li> <li>● Dynamic exercise acutely reduces BP among people with HTN for a major portion of the daytime hours.</li> <li>● Resistance training performed according to the ACSM guidelines reduces BP in normotensive and hypertensive adults.</li> <li>● Limited evidence suggests static exercise reduces BP in adults with elevated BP.</li> <li>● Limited evidence suggests resistance exercise has little effect on BP for up to 24 h after the exercise session.</li> <li>● There are currently no studies available to provide a recommendation regarding the acute effects of static exercise on BP in adults.</li> <li>● Regular endurance exercise reduces BP in older adults as it does in younger persons.</li> <li>● Limited evidence suggests PEH occurs in older adults.</li> <li>● The evidence to date does not support endurance and resistance training as a nonpharmacologic intervention for reducing BP in children and adolescents.</li> <li>● Endurance exercise training reduces BP similarly in men and women.</li> <li>● Limited evidence suggests acute endurance exercise reduces BP similarly in white men and women.</li> <li>● Currently no convincing evidence exists to support the notion that ethnic differences exist in the BP response to chronic exercise training.</li> <li>● Currently no convincing evidence exists to support the notion that ethnic differences exist in the BP response to acute exercise.</li> </ul>	C A B B B B C C None B C B B C C C
Exercise recommendations	<ul style="list-style-type: none"> <li>● For persons with high BP, an exercise program that is primarily aerobic-based is recommended.</li> <li>● Resistance training should serve as an adjunct to an aerobic-based program.</li> <li>● The evidence is limited regarding frequency, intensity, time, and type recommendations.</li> <li>● Limited evidence exists regarding special considerations for those with HTN.</li> </ul>	A B C D
Mechanisms	<ul style="list-style-type: none"> <li>● Neural and vascular changes contribute to the decreases in BP that result from acute and chronic endurance exercise.</li> <li>● Emerging data suggest possible genetic links to acute and chronic exercise BP reductions.</li> </ul>	C D

\* See Table 3 for definition of evidence based categories.

BP, blood pressure; CVD, cardiovascular disease; PEH, postexercise hypotension.

between vascular transduction systems to decrease vascular resistance. Although the mechanism for PEH remains elusive, multiple factors probably interact to mediate the response (110).

The extent to which the lowering effects of exercise training on BP reflect the integration or carryover of the acute effects of exercise (i.e., PEH) remains unknown. Studies linking PEH to the long-term antihypertensive adaptations associated with exercise training have yet to be conducted.

**Evidence statement.** Both neural and vascular changes contribute to the decreases in BP that result from acute and chronic endurance exercise. *Evidence category C.* Emerging data suggest possible genetic links to acute and chronic exercise BP reductions. *Evidence category D.* Because of multifactorial mechanisms and system redundancies contributing to the BP reductions from acute and chronic endurance exercise, definitive conclusions regarding the mechanisms for the hypotensive influence of endurance exercise cannot be made at this time.

## SUMMARY AND CONCLUSIONS

HTN, one of the most common medical disorders, is associated with an increased incidence of all-cause and

CVD mortality, and is the most prevalent cardiovascular condition found in recreational exercisers and athletes. Because of the importance of the topic, the ACSM presents an evidence based review of the current state of knowledge on exercise and HTN. Table 4 lists and categorizes the evidence statements made within this Position Stand.

Despite conclusive evidence that antihypertensive therapy reduces CVD complications, HTN has a low successful treatment rate. Lifestyle modifications are advocated for the treatment and prevention of HTN, with exercise being an integral component. This recommendation stems from longitudinal studies showing higher levels of physical activity and fitness are associated with decreased risks of developing HTN. Exercise programs that primarily include endurance activity, resistance training, or both, not only have a role in the primary prevention of HTN but also lower BP. The BP-lowering effects of exercise are most pronounced in people with HTN who engage in endurance exercise with BP-decreasing approximately 5–7 mm Hg after an isolated exercise session (acute) or after exercise training (chronic). Moreover, BP is reduced for up to 22 h after an endurance exercise bout (e.g., PEH), with the greatest decreases among those with the highest baseline BP. Small decrements in SBP and DBP of 2 mm Hg reduce the risk of stroke by 14% and 17%, and risk of coronary artery disease by 9% and 6%, respectively, in the general population. Enormous public

health benefit could be realized by people with HTN if they habitually exercised.

There are numerous proposed mechanisms for the salutary effect of exercise on BP, including neurohumoral, vascular, and structural adaptations. Decreases in catecholamines, TPR, and body weight and fat stores, as well as improved insulin sensitivity and alterations in vasodilators and vasoconstrictors are postulated to explain the antihypertensive effects of exercise. Emerging data suggest possible genetic links to BP reductions associated with acute and chronic endurance exercise. Nonetheless, definitive conclusions regarding the mechanisms for the BP reductions after endurance exercise cannot be made at this time.

Individuals with controlled HTN and no CVD or renal complications may participate in an exercise program or competitive athletics, but should be evaluated, treated, and monitored closely. There are limitations in establishing BP cutoff levels for participation because BP exists on a continuum. Preliminary peak or symptom-limited exercise testing may be warranted, especially for men over 45 and women over 55 yr planning a vigorous exercise program (i.e.,  $\geq 60\% \dot{V}O_{2R}$ ). In the interim, while formal evaluation and management are taking place, it is reasonable for the majority of patients to begin moderate-intensity exercise training ( $40\text{--}<60\% \dot{V}O_{2R}$ ) such as walking. When pharmacologic therapy is indicated in physically active people it should, ideally: a) lower arterial BP not only at rest, but during exertion; b) decrease TPR; and c) not adversely affect exercise capacity. For these reasons, angiotensin converting enzyme (ACE) inhibitors (or angiotensin II receptor blockers in case of ACE inhibitor intolerance) and calcium channel blockers are currently the drugs of choice for recreational exercisers and athletes with HTN, except that the

combination of an angiotensin converting enzyme inhibitor and an angiotensin II receptor blocker is currently not warranted. If a third drug is required, a low-dose thiazide-like diuretic, possibly in combination with a potassium sparing agent, can be recommended. There is no evidence that antihypertensive agents would impair performance in static sports.

Exercise remains a cornerstone therapy for the primary prevention, treatment, and control of HTN. The optimal training FITT needs to be more precisely defined in order to optimize the BP-lowering capacities of exercise, particularly in children, women, older adults, and certain ethnic groups. Based upon the current evidence, the following exercise prescription is recommended for those with high BP:

Frequency: on most, preferably all, days of the week

Intensity: moderate intensity ( $40\text{--}<60\%$  of  $\dot{V}O_{2R}$ )

Time:  $\geq 30$  min of continuous or accumulated physical activity per day

Type: primarily endurance physical activity supplemented by resistance exercise.

## ACKNOWLEDGMENT

This pronouncement was reviewed for the American College of Sports Medicine by the members-at-large; the Pronouncements Committee; Carl Foster, Ph.D., FACSM; James Hagberg, Ph.D., FACSM; Gary Jennings, Ph.D.; Mark Williams, Ph.D., FACSM; Jack Wilmore, Ph.D., FACSM, and Edward Zambraski, Ph.D., FACSM.

This Position Stand replaces the 1993 ACSM position paper, "Physical Activity, Physical Fitness, and Hypertension," *Med. Sci. Sports Exerc.* 25(10):i–x, 1993.

## REFERENCES

1. ABBOUD, F. M. The sympathetic system in hypertension: State-of-the-art review. *Hypertens.* 4:208–225, 1982.
2. AKINPELU, A. O. Responses of the African hypertensive to exercise training: preliminary observations. *J. Hum. Hypertens.* 4:74–76, 1990.
3. ALBRIGHT, A., M. FRANZ, G. HORNSBY, et al. American College of Sports Medicine position stand: exercise and Type 2 diabetes. *Med. Sci. Sports Exerc.* 32:1345–1360, 2000.
4. ALLEN, D. H., I. B. PUDDY, A. R. MORTON, and L. J. BEILIN. A controlled study of the effects of aerobic exercise on antihypertensive drug requirements of essential hypertensive patients in the general practice setting. *Clin. Exp. Pharmacol. Physiol.* 18: 279–282, 1991.
5. ALPERT, B. S., J. H. WILMORE. Physical activity and blood pressure in adolescents. *Pediatr. Exerc. Sci.* 8:361–380, 1994.
6. AMERICAN COLLEGE OF SPORTS MEDICINE. Position stand: physical activity, physical fitness, and hypertension. *Med. Sci. Sports Exerc.* 25:i–x, 1993.
7. AMERICAN COLLEGE OF SPORTS MEDICINE. Position stand: exercise for patients with coronary artery disease. *Med. Sci. Sports Exerc.* 26:i–v, 1994.
8. AMERICAN COLLEGE OF SPORTS MEDICINE AND AMERICAN DIABETES Association joint position statement: diabetes mellitus and exercise. *Med. Sci. Sports Exerc.* 29:i–vi, 1997.
9. 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.* 30:975–991, 1998.
10. AMERICAN COLLEGE OF SPORTS MEDICINE POSITION STAND. Exercise and physical activity for older adults. *Med. Sci. Sports Exerc.* 30:992–1008, 1998.
11. AMERICAN HEART ASSOCIATION. 2003 Heart and Stroke Statistical Update. Dallas, TX: American Heart Association, 2002.
12. ANDERSON, E. A., R. P. HOFFMAN, T. W. BALON, C. A. SINKEY, and A. L. MARK. Hyperinsulinemia produces both sympathetic neural activation and vasodilation in normal humans. *J. Clin. Invest.* 87:2246–2252, 1991.
13. BARON, A. D., G. BRECHTEL-HOOK, A. JOHNSON, and D. HARDIN. Skeletal muscle blood flow. A possible link between insulin resistance and blood pressure. *Hypertens.* 21:129–135, 1993.
14. BELLES, D. R. *The Benefits of Circuit Weight Training in Law Enforcement Personnel*. Gainesville, FL: University of Florida, 1989.
15. BENETOS, A., A. RUDNICKI, M. SAFAR, and L. GUIZE. Pulse pressure and cardiovascular mortality in normotensive and hypertensive subjects. *Hypertens.* 32:560–564, 1998.
16. BENNETT, T., R. G. WILCOX, and I. A. MACDONALD. Post-exercise reduction of blood pressure in hypertensive men is not due to acute impairment of baroreflex function. *Clin. Sci. (Lond.)* 67: 97–103, 1984.
17. BERTAGNOLI, K., P. HANSON, and A. WARD. Attenuation of exercise-induced ST depression during combined isometric and dynamic exercise in coronary artery disease. *Am. J. Cardiol.* 65: 314–317, 1990.



18. BLAIR, S. N., N. N. GOODYEAR, L. W. GIBBONS, and K. H. COOPER. Physical fitness and incidence of hypertension in healthy normotensive men and women. *JAMA* 252:487–490, 1984.
19. BLAIR, S. N., H. W. KOHL, N. F. GORDON, and R. S. Paffenbarger, Jr. How much physical activity is good for health? *Annu. Rev. Public Health* 13:99–126, 1992.
20. BLESSING, D. L., R. E. KEITH, H. N. WILLIFORD, M. E. BLESSING, and J. A. BARKSDALE. Blood lipid and physiological responses to endurance training in adolescents. *Pediatr. Exerc. Sci.* 7:192–202, 1995.
21. BLUMENTHAL, J. A., C. F. EMERY, D. J. MADDEN, et al. Cardiovascular and behavioral effects of aerobic exercise training in healthy older men and women. *J. Gerontol.* 44:147–157, 1989.
22. BLUMENTHAL, J. A., A. SHERWOOD, E. C. GULLETTE, et al. Exercise and weight loss reduce blood pressure in men and women with mild hypertension: effects on cardiovascular, metabolic and hemodynamic functioning. *Arch. Intern. Med.* 160:1947–1958, 2000.
23. BLUMENTHAL, J. A., W. C. SIEGEL, and M. APPELBAUM. Failure of exercise to reduce blood pressure in patients with mild hypertension. *JAMA* 266:2098–2104, 1991.
24. BRAITH, R. W., M. L. POLLOCK, D. T. LOWENTHAL, J. E. GRAVES, and M. C. LIMACHER. Moderate- and high-intensity exercise lowers blood pressure in normotensive subjects 60 to 79 years of age. *Am. J. Cardiol.* 73:1124–1128, 1994.
25. BROWN, M. K. *The Effects of Diet and Exercise on Selected Coronary Risk Factors in Children*. Provo, UT: Brigham Young University; 1981.
26. BROWN, M. D., G. E. MOORE, M. T. KORYTHOWSKI, S. D. MCCOLE, and J. M. HAGBERG. Improvement of insulin sensitivity by short-term exercise training in hypertensive African American women. *Hypertens.* 30:1549–1553, 1997.
27. BROWN, M. D., D. R. DENGEL, R. V. HOGIKYAN, and M. A. SUPIANO. Sympathetic activity and the heterogeneous blood pressure response to exercise training in hypertensives. *J. Appl. Physiol.* 92:1434–1442, 2002.
28. BROWNLEY, K. A., S. G. WEST, A. L. HINDERLITER, and K. C. LIGHT. Acute aerobic exercise reduces ambulatory blood pressure in borderline hypertensive men and women. *Am. J. Hypertens.* 9:200–206, 1996.
29. BRUM, P. C., G. J. DA SILVA, E. D. MOREIRA, F. IDA, C. E. NEGRAO, and E. M. KRIEGER. Exercise training increases baroreceptor gain sensitivity in normal and hypertensive rats. *Hypertens.* 36:1018–1022, 2000.
30. BURT, V. L., P. WHELTON, E. J. ROCELLA, et al. Prevalence of hypertension in the US Adult Population. Results from the Third National Health and Nutrition Examination Survey, 1988–1991. *Hypertension* 25:305–313, 1995.
31. BYRNE, H. K. *The Effects of Exercise Training on Resting Metabolic Rate and Resting Blood Pressure in Women*. Austin, TX: University of Texas at Austin, 1997.
32. CAMERON, J. D., and A. M. DART. Exercise training increases total systemic arterial compliance in humans. *Am. J. Physiol.* 266:H693–H701, 1994.
33. CANTWELL, J. D., and G. F. FLETCHER. Cardiovascular complications while jogging. *JAMA* 210:130–131, 1969.
34. CARDILLO, C., C. M. KILCOYNE, M. WACLAWI, R. O. CANNON, III, and J. A. PANZA. Role of endothelin in the increased vascular tone of patients with essential hypertension. *Hypertens.* 33:753–758, 1999.
35. CHANDLER, M. P., and S. E. DICARLO. Sinoaortic denervation prevents postexercise reductions in arterial pressure and cardiac sympathetic tonus. *Am. J. Physiol.* 273:H2738–H2745, 1997.
36. CHEN, C. Y., P. A. MUNCH, A. W. QUAIL, and A. C. BONHAM. Postexercise hypotension in conscious SHR is attenuated by blockade of substance P receptors in NTS. *Am. J. Physiol. Heart Circ. Physiol.* 283:H1856–H1862, 2002.
37. CHEN, H. I., and I. P. CHIANG. Chronic exercise decreases adrenergic agonist-induced vasoconstriction in spontaneously hypertensive rats. *Am. J. Physiol.* 271:H977–H983, 1996.
38. CHEN, H. I., H. T. LI, and C. C. CHEN. Physical conditioning decreases norepinephrine-induced vasoconstriction in rabbits: possible roles of norepinephrine-evoked endothelium-derived relaxing factor. *Circulation* 90:970–975, 1994.
39. CHOBANIAN, A. V., G. L. BAKRIS, H. R. BLACK, et al., and the National High Blood Pressure Education Program Coordinating Committee: the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. The JNC 7 Report. *Hypertens.* 42:1206–1252, 2003.
40. CLÉROUX, J. M. KOUAMÉ, A. NADEAU, D. COULOMBE, and Y. LACOURCIÈRE. After effects of exercise on regional and systemic hemodynamics in hypertension. *Hypertens.* 19:183–191, 1992.
41. COATS, A. J. S., J. CONWAY, J. D. ISEA, G. PANNARALE, P. SLEIGHT, and V. K. SOMERS. Systemic and forearm vascular resistance changes after upright bicycle exercise in man. *J. Physiol.* 413:289–298, 1989.
42. COLLINS, H. L., and S. E. DICARLO. Attenuation of postexercise hypotension by cardiac afferent blockade. *Am. J. Physiol.* 265:H1179–H1183, 1993.
43. COLLINS, H. L., D. W. RODENBAUGH, and S. E. DICARLO. Central blockade of vasopressin V(1) receptors attenuates postexercise hypotension. *Am. J. Physiol. Regul. Integr. Comp. Physiol.* 281:R375–R380, 2001.
44. COLLINS, R., R. PETO, S. MCMAHON, et al. Blood pressure, stroke and coronary heart disease: part 2, short-term reductions in blood pressure: overview of randomized drug trials in their epidemiological context. *Lancet* 335:827–838, 1990.
45. CONONIE, C. C., J. E. GRAVES, M. L. POLLOCK, M. I. PHILLIPS, C. SUMNERS, and J. M. HAGBERG. Effect of exercise training on blood pressure in 70- to 79-yr-old men and women. *Med. Sci. Sports Exerc.* 23:505–511, 1991.
46. COOPER, A. R., L. A. MOORE, J. MCKENNA, and C. J. RIDDOCH. What is the magnitude of blood pressure response to a programme of moderate intensity exercise? Randomised controlled trial among sedentary adults with unmedicated hypertension. *Br. J. Gen. Pract.* 50:958–962, 2000.
47. COX, J. L., I. B. PUDDY, A. R. MORTON, V. BURKE, L. J. BEILIN, and M. MCALEER. Exercise and weight control in sedentary overweight men: effects on clinic and ambulatory blood pressure. *J. Hypertens.* 14:779–790, 1996.
48. CUNNINGHAM, D. A., P. A. RECHNITZER, J. H. HOWARD, and A. P. DONNER. Exercise training of men at retirement: a clinical trial. *J. Gerontol.* 42:17–23, 1987.
49. DAVIDOFF, R., C. L. SCHAMROTH, A. P. GOLDMAN, T. H. DIAMOND, A. J. CILLIERS, and D. P. MYBURGH. Postexercise blood pressure as a predictor of hypertension. *Aviat. Space Environ. Med.* 53:591–594, 1982.
50. DE BUSK, R., W. PITTS, W. HASKELL, and N. HOUSTON. Comparison of cardiovascular responses to static-dynamic effort and dynamic effort alone in patients with chronic ischemic heart disease. *Circulation* 59:977–984, 1979.
51. DELP, M. D., R. M. MCALLISTER, and M. H. LAUGHLIN. Exercise training alters endothelium-dependent vasoreactivity of rat abdominal aorta. *J. Appl. Physiol.* 75:1354–1363, 1993.
52. DELP, M. D., R. M. MCALLISTER, and M. H. LAUGHLIN. Exercise training alters aortic vascular reactivity in hypothyroid rats. *Am. J. Physiol.* 268:H1428–H1435, 1995.
53. DELP, M. D., and M. H. LAUGHLIN. Time course of enhanced endothelium-mediated dilation in aorta of trained rats. *Med. Sci. Sports Exerc.* 29:1454–1461, 1997.
54. DINENNO, F. A., P. P. JONES, D. R. SEALS, and H. TANAKA. Age-associated arterial wall thickening is related to elevations in sympathetic activity in healthy humans. *Am. J. Physiol. Heart Circ. Physiol.* 278:H1205–H1210, 2000.
55. DINENNO, F. A., H. TANAKA, K. D. MONAHAN, et al. Regular endurance exercise induces expansive arterial remodeling in the trained limbs of healthy men. *J. Physiol.* 534:287–295, 2001.
56. DON, B. W. M. *The Effects of Strength Training on Cardiovascular Reactivity to Stress and Psychological Well-Being in College Women*. Boston: Boston University, 1996.
57. DUEY, W. J., W. L. O'BRIEN, A. B. CRUTCHFIELD, L. A. BROWN, H. N. WILLIFORD, and M. SHARFF-OLSON. Effects of exercise training on aerobic fitness in African-American females. *Ethn. Dis.* 8:308–311, 1998.

58. DUNSTAN, D. W., I. B. PUDDEY, L. J. BEILIN, V. BURKE, A. R. MORTON, and K. G. STANTON. Effects of a short-term circuit weight training program on glycaemic control in NIDDM. *Diabetes Res. Clin. Pract.* 40:53–61, 1998.
59. DWYER, T., W. E. COONAN, D. R. LEITCH, B. S. HETZEL, and R. A. BAGHURST. An investigation of the effects of daily physical activity on the health of primary school students in South Australia. *Int. J. Epidemiol.* 12:308–313, 1983.
60. EDWARDS, M. T., and J. N. DIANA. Effect of exercise on pre- and postcapillary resistance in the spontaneously hypertensive rat. *Am. J. Physiol.* 234:H439–446, 1978.
61. EUROPEAN SOCIETY OF HYPERTENSION. European Society of Cardiology guidelines for the management of arterial hypertension. *J. Hypertens.* 21:1011–1053, 2003.
62. EWART, C. K., D. ROHM YOUNG, and J. M. HAGBERG. Effects of school-based aerobic exercise on blood pressure in adolescent girls at risk for hypertension. *Am. J. Public Health* 88:949–951, 1998.
63. FAGARD, R., R. GRAUWELS, D. GROESENEN, P. LUNEN, J. STAESSEN, L. VANHEES, and A. AMERY. Plasma levels of renin, angiotensin II, and 6-ketoprostaglandin F1 alpha in endurance athletes. *J. Appl. Physiol.* 59:947–952, 1985.
64. FAGARD, R. H. Physical fitness and blood pressure. *J. Hypertens.* 11(Suppl. 5):S47–S52, 1993.
65. FAGARD, R. H., K. PARDAENS, J. A. STAESSEN, and L. THijs. Prognostic value of invasive hemodynamic measurements at rest and during exercise in hypertensive men. *Hypertens.* 28:31–36, 1996.
66. FAGARD, R. H., K. PARDAENS, J. A. STAESSEN, and L. THijs. Should exercise blood pressure be measured in clinical practice? *J. Hypertens.* 16:1215–1217, 1998.
67. FAGARD, R. Exercise and hypertension. *J. Hum. Hypertens.* 13: 359–360, 1999.
68. FAGARD, R., K. PARDAENS, and J. VANHAECHE. Prognostic significance of exercise versus resting blood pressure in patients with chronic heart failure. *J. Hypertens.* 17:1977–1981, 1999.
69. FAGARD, R. H. Physical activity in the prevention and treatment of hypertension in the obese. *Med. Sci. Sports Exerc.* 31(Suppl.): S624–S630, 1999.
70. FAGARD, R. H. Physical activity, fitness and blood pressure. In: *Handbook of Hypertension: Epidemiology of Hypertension*, W. H. Birkenhäger, J. L. Reid, and C. J. Bulpitt (Eds.). Amsterdam: Elsevier, 2000, pp. 191–211.
71. FAGARD, R. H. Exercise characteristics and the blood pressure response to dynamic physical training. *Med. Sci. Sports Exerc.* 33(Suppl):S484–492; discussion S493–494, 2001.
72. FAGARD, R. H., M. VAN DEN ENDEN, M. LEEMAN, and X. WARLING. Survey on treatment of hypertension and implementation of WHO/ISH risk stratification in primary care in Belgium. *J. Hypertens.* 20:1297–1302, 2002.
73. FAIGENBAUM, A. D., L. D. ZAICHKOWSKY, W. L. WESTCOTT, L. J. MICHELI, and A. F. FEHLANDT. The effects of twice-a-week strength training program on children. *Pediatr. Exerc. Sci.* 5:339–346, 1993.
74. FERRIER, K. E., T. K. WADDELL, C. D. GATZKA, J. D. CAMERON, A. M. DART, and B. A. KINGWELL. Aerobic exercise training does not modify large-artery compliance in isolated systolic hypertension. *Hypertens.* 38:222–226, 2001.
75. FILIPOVSKY, J., P. DUCIMETIÈRE, and M. E. SAFAR. Prognostic significance of exercise blood pressure and heart rate in middle-aged men. *Hypertens.* 20:333–339, 1992.
76. FITZGERALD, W. Labile hypertension and jogging: new diagnostic tool or spurious discovery? *Br. Med. J.* 282:542–544, 1981.
77. FLEG, J. L., and E. G. LAKATTA. Prevalence and significance of postexercise hypotension in apparently healthy subjects. *Am. J. Cardiol.* 57:1380–1384, 1986.
78. FLETCHER, G. F., G. BALADY, S. N. BLAIR, 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* 94:857–862, 1996.
79. FLETCHER, G. F., G. J. BALADY, E. A. AMSTERDAM, et al. Exercise standards for testing and training: a statement for healthcare professionals from the American Heart Association. *Circulation* 104:1694–1740, 2001.
80. FLORAS, J. S., C. A. SINKEY, P. E. AYLWARD, D. R. SEALS, P. N. THOREN, and A. L. MARK. Postexercise hypotension and sympathoinhibition in borderline hypertensive men. *Hypertens.* 14:28–35, 1989.
81. FLORAS, J. S., and J. WESCHE. Haemodynamic contributions to post-exercise hypotension in young adults with hypertension and rapid resting heart rates. *J. Hum. Hypertens.* 6:265–269, 1992.
82. FLORAS, J. S., and K. HARA. Sympathoneural and haemodynamic characteristics of young subjects with mild essential hypertension. *J. Hypertens.* 11:647–655, 1993.
83. FOLSOM, A. R., R. J. PRINEAS, S. A. KAYE, and R. G. MUNGER. Incidence of hypertension and stroke in relation to body fat distribution and other risk factors in older women. *Stroke* 21: 701–706, 1990.
84. FORJAZ, C. L. M., D. F. SANTAELLA, L. O. REZENDE, A. C. P. BARRETTO, and C. E. NEGRÃO. Exercise duration determines the magnitude and duration of post-exercise hypotension. *Arq. Bras. Cardiol.* 70:99–104, 1998.
85. FORJAZ, C. L., Y. MATSUDAIRA, F. B. RODRIGUES, N. NUNES, and C. E. NEGRÃO. Post-exercise changes in blood pressure, heart rate and rate pressure product at different exercise intensities in normotensive humans. *Braz. J. Med. Biol. Res.* 31:1247–1255, 1998.
86. FORJAZ, C. L., D. F. SANTAELLA, L. O. REZENDE, A. C. BARRETTO, and C. E. NEGRÃO. Effect of exercise duration on the magnitude and duration of post-exercise hypotension. *Arq. Bras. Cardiol.* 70:99–104, 1998.
87. FORTMANN, S. P., W. L. HASKELL, P. D. WOOD, and THE STANFORD WEIGHT CONTROL PROJECT TEAM. Effects of weight loss on clinic and ambulatory blood pressure in normotensive men. *Am. J. Cardiol.* 62:89–93, 1988.
88. FRANKLIN, B. A., M. H. WHALEY, and E. T. HOWLEY (Eds.). *ACSM's Guidelines for Exercise Testing and Prescription*, 6th Ed. Baltimore: Lippincott Williams & Wilkins, 2000.
89. FRANKLIN, P. J., D. J. GREEN, and N. T. CABLE. The influence of thermoregulatory mechanisms on post-exercise hypotension in humans. *J. Physiol.* 470:231–241, 1993.
90. FRANKLIN, S. S., S. A. KHAN, N. D. WONG, M. G. LARSON, and D. LEVY. Is pulse pressure useful in predicting coronary heart disease? The Framingham Heart Study. *Circulation* 100:354–360, 1999.
91. FRIDAY, W. W. *Physiological, Psychological, and Behavioral Effects of Aerobic Exercise, and Cognitive Experiential Therapy on Juvenile Delinquent Males*. Columbus, OH: The Ohio State University, 1987.
92. GALLAGHER, J. S. *The Chronic Effects of a Weight Training Program on Blood Pressure in Adolescent Males*. Philadelphia: Temple University, 1987.
93. GASOWSKI, J., R. H. FAGARD, J. A. STAESSEN, et al. Pulsatile blood pressure component as predictor of mortality in hypertension: a meta-analysis of clinical trial control groups. *J. Hypertens.* 20: 145–151, 2002.
94. GEYSSANT, A., G. GEELEN, C. DENIS, et al. Plasma vasopressin, renin activity, and aldosterone: effect of exercise and training. *Eur. J. Appl. Physiol. Occup. Physiol.* 46:21–30, 1981.
95. GIBBONS, R. J., G. J. BALADY, J. W. BEASELY, et al. ACC/AHA guidelines for exercise testing: executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Exercise Testing). *J. Am. Coll. Cardiol.* 30:260–315, 1997.
96. GILLET, P. A., A. T. WHITE, M. S. CAASERTA. Effect of exercise and/or fitness education on fitness in older, sedentary, obese women. *J. Aging Phys. Activity* 4:42–55, 1996.
97. GIRI, S., P. D. THOMPSON, F. J. KIERNAN, et al. Clinical and angiographic characteristics of exertion-related acute myocardial infarction. *JAMA* 282:1731–1736, 1999.
98. GORDON, N. F. Hypertension. In: *ACSM's Exercise Management for Persons with Chronic Diseases and Disabilities*, J. L. Durs-tine (Ed.). Champaign, IL: Human Kinetics, 1997, pp. 59–63.



99. GREENWOOD, J. P., J. B. STOKER, and D. A. MARY. Single-unit sympathetic discharge: quantitative assessment in human hypertensive disease. *Circulation* 100:1305–1310, 1999.
100. GUTIN, and S. OWENS. Role of exercise intervention in improving body fat distribution and risk profile in children. *Am. J. Hum. Biol.* 11:237–247, 1999.
101. HAAKANEN, N., S. MIILUNPALO, I. VUORI, P. OJA, and M. PASANEN. Association of leisure time physical activity with the risk of coronary heart disease, hypertension and diabetes in middle-aged men and women. *Int. J. Epidemiol.* 26:739–747, 1997.
102. HAGBERG, J. M., S. J. MONTAIN, and W. H. MARTIN, III. Blood pressure and hemodynamic responses after exercise in older hypertensives. *J. Appl. Physiol.* 63:270–276, 1987.
103. HAGBERG, J. M., S. J. MONTAIN, W. H. MARTIN, III, and A. A. Ehsani. Effect of exercise training in 60-to 69-year-old persons with essential hypertension. *Am. J. Cardiol.* 64:348–353, 1989.
104. HAGBERG, J. M., J. E. GRAVES, M. LIMACHER, et al. Cardiovascular responses of 70- to 79-yr-old men and women to exercise training. *J. Appl. Physiol.* 66:2589–2594, 1989.
105. HAGBERG, J. M., R. E. FERRELL, D. R. DENGEL, and K. R. WILUND. Exercise training-induced blood pressure and plasma lipid improvements in hypertensives may be genotype dependent. *Hypertens.* 34:18–23, 1999.
106. HAGBERG, J. M., J. J. PARK, and M. D. BROWN. The role of exercise training in the treatment of hypertension: an update. *Sports Med.* 30:193–206, 2000.
107. HAJJAR, I, and T. A. KOTCHEN. Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1998–2000. *JAMA* 290:199–206, 2003.
108. HALBERT, J. A., C. A. SILAGY, P. FINUCANE, R. T. WITHERS, P. A. HAMDORF, and G. R. ANDREWS. The effectiveness of exercise training in lowering blood pressure: a meta-analysis of randomized controlled trials of 4 weeks or longer. *J. Hum. Hypertens.* 11:641–649, 1997.
109. HALLIWILL, J. R., J. A. TAYLOR, and D. L. ECKBERG. Impaired sympathetic vascular regulation in humans after acute dynamic exercise. *J. Physiol.* 495(Pt. 1):279–288, 1996.
110. HALLIWILL, J. R. Mechanisms and clinical implications of post-exercise hypotension in humans. *Exerc. Sport Sci. Rev.* 29:65–70, 2001.
111. HAMDORF, P. A., R. T. WITHERS, R. K. PENHALL, and M. V. HASLAM. Physical training effects on the fitness and habitual activity patterns of elderly women. *Arch. Phys. Med. Rehabil.* 73:603–608, 1992.
112. HANNUM, S. M., and F. W. KASCH. Acute postexercise blood pressure response of hypertensive and normotensive men. *Scand. J. Sports Sci.* 3:11–15, 1981.
113. HANSEN, H. S., K. FROBERG, N. HYLDEBRANDT, and J. R. NIELSEN. A controlled study of eight months of physical training and reduction of blood pressure in children: the Odense schoolchild study. *Br. Med. J.* 303:682–685, 1991.
114. HARA, K., and J. S. FLORAS. Effects of naloxone on hemodynamics and sympathetic activity after exercise. *J. Appl. Physiol.* 73:2028–2035, 1992.
115. HARA, K., and J. S. FLORAS. Influence of naloxone on muscle sympathetic nerve activity, systemic and calf haemodynamics and ambulatory blood pressure after exercise in mild essential hypertension. *J. Hypertens.* 13:447–461, 1994.
116. HARRIS, K. A., and R. G. HOLLY. Physiological response to circuit weight training in borderline hypertensive subjects. *Med. Sci. Sports Exerc.* 19:246–252, 1987.
117. HARSHFIELD, G. A., L. M. DUPAUL, B. S. ALPERT, et al. Aerobic fitness and the diurnal rhythm of blood pressure in adolescents. *Hypertens.* 15:810–814, 1990.
118. HASKELL, W. L. Health consequences of physical activity: understanding and challenges regarding dose-response. *Med. Sci. Sports Exerc.* 26:649–660, 1994.
119. HAYASHI, T., K. TSUMURA, C. SUEMATSU, K. OKADA, S. FUJII, and G. ENDO. Walking to work and the risk for hypertension in men: the Osaka Health Survey. *Ann. Intern. Med.* 130:21–26, 1999.
120. HEADLEY, S. A., J. M. CLAIBORNE, C. R. LOTTES, and C. G. KORBA. Hemodynamic responses associated with post-exercise hypotension in normotensive black males. *Ethn. Dis.* 6:190–201, 1996.
121. HEADLEY, S. A., T. G. KEENAN, and T. M. MANOS. Renin and hemodynamic responses to exercise in borderline hypertensives. *Ethn. Dis.* 8:312–318, 1998.
122. HENRIKSEN, E. J. Effects of acute exercise and exercise training on insulin resistance. *J. Appl. Physiol.* 93:788–796, 2002.
123. HESPEL, P., P. LUNEN, R. VAN HOOFF, et al. Effects of physical endurance training on the plasma renin-angiotensin-aldosterone system in normal man. *J. Endocrinol.* 116:443–449, 1988.
124. HIGASHI, Y., S. SASAKI, S. KURISU, et al. Regular aerobic exercise augments endothelium dependent vascular relaxation in normotensive as well as hypertensive subjects: role of endothelium-derived nitric oxide. *Circulation* 100:1194–1202, 1999.
125. HIGASHI, Y., S. SASAKI, N. SASAKI, et al. Daily aerobic exercise improves reactive hyperemia in patients with essential hypertension. *Hypertens.* 33:591–597, 1999.
126. HILL, D. W., M. A. COLLINS, K. J. CURETON, and J. J. DEMELLO. Blood pressure response after weight training exercise. *J. Appl. Sport Sci. Res.* 3:44–47, 1989.
127. HIROYUKI, D., T. G. ALLISON, R. W. SQUIRES, et al. Peak exercise blood pressure stratified by age and gender in apparently healthy subjects. *Mayo Clin. Proc.* 71:445–452, 1996.
128. HOBERG, E., G. SCHULER, B. KUNZE, et al. Silent myocardial ischemia as apotential link between lack of premonitoring symptoms and increased risk of cardiac arrest during physical stress. *Am. J. Cardiol.* 65:583–589, 1990.
129. HOWLEY, E. T. Type of activity: resistance, aerobic and leisure versus occupational physical activity. *Med. Sci. Sports Exerc.* 33(Suppl.):S364–S369, 2001.
130. HUONKER, M., M. HALLE, and J. KEUL. Structural and functional adaptations of the cardiovascular system by training. *Int. J. Sports Med.* 17(Suppl. 3):S164–172, 1996.
131. IRVING, J. B., R. A. BRUCE, and T. A. DEROUEN. Variations in and significance of systolic pressure during maximal exercise (treadmill) testing: relation to severity of coronary artery disease and cardiac mortality. *Am. J. Cardiol.* 39:841–848, 1977.
132. ISHIKAWA-TAKATA, K., T. OHTA, and H. TANAKA. How much exercise is required to reduce blood pressure in essential hypertensives: a dose-response study. *Am. J. Hypertens.* 16:629–633, 2003.
133. JAKICIC, J. M., K. CLARK, E. COLEMAN, et al. American College of Sports Medicine position stand: appropriate intervention strategies for weight loss and prevention of weight regain for adults. *Med. Sci. Sports Exerc.* 33:2145–2156, 2001.
134. JENNINGS, G., L. NELSON, P. NESTEL, M. ESLER, P. KORNER, D. BURTON, and J. BAZELMANS. The effects of changes in physical activity on major cardiovascular risk factors, hemodynamics, sympathetic function, and glucose utilization in man: a controlled study of four levels of activity. *Circulation* 73:30–40, 1986.
135. JENNINGS, G. L., G. DEAKIN, P. KORNER, I. MEREDITH, B. KINGWELL, and L. NELSON. What is the dose-response relationship between exercise training and blood pressure? *Ann. Med.* 23: 313–318, 1991.
136. JESSUP, J. V., D. T. LOWENTHAL, M. L. POLLOCK, and T. TURNER. The effects of endurance exercise training on ambulatory blood pressure in normotensive older adults. *Geriatr. Nephrol. Urol.* 8:103–109, 1998.
137. JOINT NATIONAL COMMITTEE ON DETECTION, EVALUATION, AND TREATMENT OF HIGH BLOOD PRESSURE. The 6th Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI). *Arch. Intern. Med.* 157:2413–2446, 1997.
138. JONES, A. W., L. J. RUBIN, and L. MAGLIOLA. Endothelin-1 sensitivity of porcine coronary arteries is reduced by exercise training and is gender dependent. *J. Appl. Physiol.* 87:1172–1177, 1999.
139. JONES, T. M. *Regular Aerobic Exercise and Blood Pressure in Elementary School Children: A Randomized Clinical Trial*. Chapel Hill, North Carolina: University of North Carolina @ Chapel Hill; 1988.
140. JULIUS, S., T. GUDBRANDSSON, K. JAMERSON, S. TARIQ SHAHAB, and O. ANDERSSON. The hemodynamic link between insulin resistance and hypertension. *J. Hypertens.* 9:983–986, 1991.



141. KAJEKAR, R., C. Y. CHEN, T. MUTOH, and A. C. BONHAM. Gaba(a) receptor activation at medullary sympathetic neurons contributes to postexercise hypotension. *Am. J. Physiol. Heart Circ. Physiol.* 282:H1615–H1624, 2002.
142. KANNEL, W. B., P. A. WOLF, D. L. MCGEE, T. R. DAWBER, P. MCNAMARA, and W. P. CASTELLI. Systolic blood pressure, arterial rigidity, and risk of stroke. *JAMA* 245:1225–1229, 1981.
143. KATZ, J., and B. R. A. WILSON. The effects of a six-week, low intensity nautilus circuit training program on resting blood pressure in females. *J. Sports Med. Phys. Fitness* 32:299–302, 1992.
144. KAUFMAN, F. L., R. L. HUGHSON, and P. SCHAMAN. Effect of exercise on recovery blood pressure in normotensive and hypertensive subjects. *Med. Sci. Sports Exerc.* 19:17–20, 1987.
145. KELLEY, G., and P. MCCLELLAN. Antihypertensive effects of aerobic exercise: a brief meta-analytic review of randomized controlled trials. *Am. J. Hypertens.* 7:115–119, 1994.
146. KELLEY, G., and Z. V. TRAN. Aerobic exercise and normotensive adults: a meta-analysis. *Med. Sci. Sports Exerc.* 27:1371–1377, 1995.
147. KELLEY, G. A. Aerobic exercise and resting blood pressure among women: a meta-analysis. *Prev. Med.* 28:264–275, 1999.
148. KELLEY, G. A., and K. S. KELLEY. Progressive resistance exercise and resting blood pressure: a meta-analysis of randomized controlled trials. *Hypertens.* 35:838–843, 2000.
149. KELLEY, G. A., K. S. KELLEY, and Z. V. TRAN. Aerobic exercise and resting blood pressure: a meta-analytic review of randomized, controlled trials. *Prev. Cardiol.* 4:73–80, 2001.
150. KELLEY, G. A., K. S. KELLEY, and Z. V. TRAN. Walking and resting blood pressure in adults: a meta-analysis. *Prev. Med.* 33:120–127, 2001.
151. KELLEY, G. A., and K. K. SHARPE. Aerobic exercise and resting blood pressure in older adults: a meta-analytic review of randomized controlled trials. *J. Gerontol. A Biol. Sci. Med. Sci.* 56:M298–M303, 2001.
152. KELLEY, G. A., K. S. KELLEY, Z. V. TRAN. The effects of exercise on resting blood pressure in children and adolescents: a meta-analysis of randomized controlled trials. *Prev. Cardiol.* 6:8–16, 2003.
153. KENNEY, M. J., and D. R. SEALS. Postexercise hypotension: key features, mechanisms, and clinical significance. *Hypertens.* 22: 653–664, 1993.
154. KING, A. C., W. L. HASKELL, C. B. TAYLOR, H. C. KRAEMER, and R. F. DEBUSK. Group- vs home-based exercise training in healthy older men and women: a community- based clinical trial. *JAMA* 266:1535–1542, 1991.
155. KINGWELL, B. A. Nitric oxide-mediated metabolic regulation during exercise: effects of training in health and cardiovascular disease. *FASEB J.* 14:1685–1696, 2000.
156. KINGWELL, B. A., B. TRAN, J. D. CAMERON, G. L. JENNINGS, and A. M. DART. Enhanced vasodilation to acetylcholine in athletes is associated with lower plasma cholesterol. *Am. J. Physiol.* 270: H2008–H2013, 1996.
157. KITAMURA, K., C. R. JORGENSEN, F. L. GOBEL, H. L. TAYLOR, and Y. WANG. Hemodynamic correlates of myocardial oxygen consumption during upright exercise. *J. Appl. Physiol.* 32:516–522, 1972.
158. KIVELOFF B., and O. HUBER. Brief maximal isometric exercise in hypertension. *J. Am. Geriatr. Soc.* 19:1006–1012, 1971.
159. KOHL, H. W., M. Z. NICHAMAN, R. F. FRANKOWSKI, and S. N. BLAIR. Maximal exercise hemodynamics and risk of mortality in apparently healthy men and women. *Med. Sci. Sports Exerc.* 28:601–609, 1996.
160. KOHNO, K., H. MATSUOKA, K. TAKENAKA, et al. Depressor effect by exercise training is associated with amelioration of hyperinsulinemia and sympathetic overactivity. *Intern. Med.* 39:1013–1019, 2000.
161. KOHRT, W. M., M. T. MALLEY, A. R. COGGAN, et al. Effects of gender, age, and fitness level on response of  $\dot{V}O_{2\max}$  to training in 60–71 yr olds. *J. Appl. Physiol.* 71:2004–2011, 1991.
162. KOKKINOS, P. F., P. NARAYAN, J. A. COLLERAN, et al. Effects of regular exercise on blood pressure and left ventricular hypertrophy in African-American men with severe hypertension. *N. Engl. J. Med.* 333:1462–1467, 1995.
163. KRÁUL, J., J. CHRASTEK, and J. ADAMIROVA. The hypotensive effect of physical activity. In: *Prevention of Ischemic Heart Disease: Principles and Practice*, W. Rabb (Ed.). Springfield, IL: Charles C Thomas, 1966, pp. 359–371.
164. KULICS, J. M., H. L. COLLINS, and S. E. DICARLO. Postexercise hypotension is mediated by reductions in sympathetic nerve activity. *Am. J. Physiol.* 276:H27–H32, 1999.
165. LASH, J. M., and H. G. BOHLEN. Functional adaptations of rat skeletal muscle arterioles to aerobic exercise training. *J. Appl. Physiol.* 72:2052–2062, 1992.
166. LAUER, R. M., T. L. BURNS, W. R. CLARKE, and L. T. MAHONEY. Childhood predictors of future blood pressure. *Hypertens.* 18(Suppl. 1):I-74–I-81, 1991.
167. LAUGHLIN, M. H., R. J. KORTHUIS, D. J. DUNCKER, and R. J. BACHE. Control of blood flow to cardiac and skeletal muscle during exercise. In: *Handbook of Physiology, Exercise: Regulation and Integration of Multiple Systems*, L. B. Rowell and J. T. Sheperd (Eds.). Bethesda, MD: American Physiological Society, 1996, pp. 705–769.
168. LAUGHLIN, M. H., W. G. SCHRAGE, R. M. MCALLISTER, H. A. GARVERICK, and A. W. JONES. Interaction of gender and exercise training: vasomotor reactivity of porcine skeletal muscle arteries. *J. Appl. Physiol.* 90:216–227, 2001.
169. LINDER, C. W., R. H. DURANT, and O. M. MAHONEY. The effect of physical conditioning on serum lipids and lipoproteins in white male adolescents. *Med. Sci. Sports Exerc.* 15:232–236, 1983.
170. MACDONALD, J. R., J. M. ROSENFELD, M. A. TARNOPOLSKY, C. D. HOGGEN, C. S. BALLANTYNE, and J. D. MACDOUGALL. Post exercise hypotension is sustained during subsequent bouts of mild exercise and simulated activities of daily living. *J. Hum. Hypertens.* 15:567–571, 2001.
171. MAEDA, S., T. MIYAUCHI, T. KAKIYAMA, et al. Effects of exercise training of 8 weeks and detraining on plasma levels of endothelium-derived factors, endothelin-1 a, endothelin-1 and nitric oxide, in healthy young humans. *Life Sci.* 69:1005–1016, 2001.
172. MANOLIO, T. A., G. L. BURKE, P. J. SAVAGE, S. SIDNEY, J. M. GARDIN, and A. OBERMAN. Exercise blood pressure response and 5-year risk of elevated blood pressure in a cohort of young adults: the CARDIA study. *Am. J. Hypertens.* 7:234–241, 1994.
173. MARCEAU, M., N. KOUAMÉ, Y. LACOURCI, and J. CLÉROUX. Effects of different training intensities on 24-hour blood pressure in hypertensive subjects. *Circulation* 88:2803–2811, 1993.
174. MATSUKAWA, T., E. GOTOH, S. UNEDA, et al. Augmented sympathetic nerve activity in response to stressors in young borderline hypertensive men. *Acta Physiol. Scand.* 141:157–165, 1991.
175. MATSUKAWA, T., T. MANO, E. GOTOH, and M. ISHII. Elevated sympathetic nerve activity in patients with accelerated essential hypertension. *J. Clin. Invest.* 92:25–28, 1993.
176. MATTHEWS, C. E., R. R. PATE, K. L. JACKSON, et al. Exaggerated blood pressure response to dynamic exercise and risk of future hypertension. *J. Clin. Epidemiol.* 51:29–35, 1998.
177. MEREDITH, I. T., G. L. JENNINGS, M. D. ESLER, et al. Time-course of the antihypertensive and autonomic effects of regular endurance exercise in human subjects. *J. Hypertens.* 8:859–866, 1990.
178. MEREDITH, I. T., P. FRIBERG, G. L. JENNINGS, et al. Exercise training lowers resting renal but not cardiac sympathetic activity in humans. *Hypertens.* 18:575–582, 1991.
179. MINSON, C. T., J. R. HALLIWILL, T. M. YOUNG, and M. J. JOYNER. Influence of the menstrual cycle on sympathetic activity, baroreflex sensitivity, and vascular transduction in young women. *Circulation* 101:862–868, 2000.
180. MITTLEMAN, M. A., M. MACLURE, G. H. TOFLER, J. B. SHERWOOD, R. J. GOLDBERG, and J. E. MULLER. Triggering of acute myocardial infarction by heavy physical exertion. *N. Engl. J. Med.* 329:1677–1683, 1993.
181. MIYAI, N., M. ARITA, K. MIYASHITA, I. MORIYAKA, T. SHIRAIISHI, and I. NISHIO. Blood pressure response to heart rate during exercise test and risk of future hypertension. *Hypertens.* 39:761–766, 2002.
182. MOHIADDIN, R. H., S. R. UNDERWOOD, H. G. BOGREN, et al. Regional aortic compliance studied by magnetic resonance imaging: the effects of age, training, and coronary artery disease. *Br. Heart J.* 62:90–96, 1989.

183. MORLIN, C., B. G. WALLIN, and B. M. ERIKSSON. Muscle sympathetic activity and plasma noradrenaline in normotensive and hypertensive man. *Acta Physiol. Scand.* 119:117–121, 1983.
184. MOTOYAMA, M., Y. SUNAMI, F. KINOSHITA, et al. Blood pressure lowering effect of low intensity aerobic training in elderly hypertensive patients. *Med. Sci. Sports Exerc.* 30:818–823, 1998.
185. MOUL, J. *The Effects of 16-Week Walking and 16-Week Weight-Training Programs on the Performance of Men and Women Ages 65–77 on the Ross Information Processing Assessment*. Greensboro, North Carolina: University of North Carolina at Greensboro, 1993.
186. MUNDAL, R., S. E. KJELDSSEN, L. SANDVIK, G. ERIKSSON, E. THAULOW, and J. ERIKSSON. Exercise blood pressure predicts cardiovascular mortality in middle-aged men. *Hypertens.* 24:56–62, 1994.
187. MUNDAL, R., S. E. KJELDSSEN, L. SANDVIK, G. ERIKSSON, E. THAULOW, and J. ERIKSSON. Exercise blood pressure predicts mortality from myocardial infarction. *Hypertens.* 27(Pt. 1):324–329, 1996.
188. MURPHY, M., A. NEVILL, C. NEVILLE, S. BIDDLE, and A. HARDMAN. Accumulating brisk walking for fitness, cardiovascular risk, and psychological health. *Med. Sci. Sports Exerc.* 34:1468–1474, 2002.
189. MYRTEK, M., and V. VILLINGER. Psychologische und physiologische Wirkungen eines fünföchigen Ergometertrainings bei Gesunden. *Med. Klin.* 71:1623–1630, 1976.
190. MYSLIVCEK, P. R., C. A. BROWN, and L. A. WOLFE. Effects of physical conditioning on cardiac autonomic function in healthy middle-aged women. *Can. J. Appl. Physiol.* 27:1–18, 2002.
191. NATIONAL INSTITUTES OF HEALTH AND NATIONAL HEART, LUNG, AND BLOOD INSTITUTE. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the Evidence Report. *Obes. Res.* 6(Suppl. 2):51S–209S, 1998.
192. NAUGHTON, J., and R. HAIDER. Methods of exercise testing. In: *Exercise Testing and Exercise Training in Coronary Heart Disease*, J. P. Naughton, H. K. Hellerstein, and I. C. Mohler (Eds.). New York: Academic Press, 1973, p. 79.
193. NELSON, L., G. L. JENNINGS, M. D. ESLER, and P. I. KORNER. Effect of changing levels of physical activity on blood-pressure and haemodynamics in essential hypertension. *Lancet* 2:473–476, 1986.
194. NELSON, R. R., F. L. GOBEL, C. R. JORGENSEN, et al. Hemodynamic predictors of myocardial oxygen consumption during static and dynamic exercise. *Circulation* 50:1179–1189, 1974.
195. NOBLE, B. J., G. A. BORG, I. JACOBS, R. CECI, and P. KAISER. A category-ratio perceived exertion scale: relationship to blood and muscle lactates and heart rate. *Med. Sci. Sports Exerc.* 15:523–528, 1983.
196. O'CONNOR, P. J., C. X. BRYANT, J. P. VELTRI, and S. M. GEBHARDT. State anxiety and ambulatory blood pressure following resistance exercise in females. *Med. Sci. Sports Exerc.* 25:516–521, 1993.
197. OKUMIYA, K., K. MATSUBAYASHI, T. WADA, S. KIMURA, Y. DOI, and T. OZAWA. Effects of exercise on neurobehavioral function in community dwelling older people more than 75 years of age. *J. Am. Geriatr. Soc.* 44:569–572, 1996.
198. OLUSEYE, K. A. Cardiovascular responses to exercise in Nigerian women. *J. Hum. Hypertens.* 4:77–79, 1990.
199. OSADA, N., B. R. CHAITMAN, L. W. MILLER, et al. Cardiopulmonary exercise testing identifies low risk patients with heart failure and severely impaired exercise capacity considered for heart transplantation. *J. Am. Coll. Cardiol.* 31:577–582, 1998.
200. PAFFENBARGER, R. S., Jr., A. L. WING, R. T. HYDE, and D. L. JUNG. Physical activity and incidence of hypertension in college alumni. *Am. J. Epidemiol.* 117:245–257, 1983.
201. PAFFENBARGER, R. S., Jr., D. L. JUNG, R. W. LEUNG, and R. T. HYDE. Physical activity and hypertension: an epidemiological view. *Ann. Med.* 23:319–327, 1991.
202. PATE, R. R., M. PRATT, S. N. BLAIR, 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* 273:402–407, 1995.
203. PATIL, R. D., S. E. DICARLO, and H. L. COLLINS. Acute exercise enhances nitric oxide modulation of vascular response to phenylephrine. *Am. J. Physiol.* 265:H1184–H1188, 1993.
204. PAULEV, P. E., R. JORDAL, O. KRISTENSEN, and J. LADEFOGED. Therapeutic effect of exercise on hypertension. *Eur. J. Appl. Physiol. Occup. Physiol.* 53:180–185, 1984.
205. PEREIRA, M. A., A. R. FOLSOM, P. G. MCGOVERN, et al. Physical activity and incident hypertension in black and white adults: the Atherosclerosis Risk in Communities Study. *Prev. Med.* 28:304–312, 1999.
206. PESCATELLO, L. S., G. W. MACK, C. N. LEACH, Jr., and E. R. NADEL. Thermoregulation in mildly hypertensive men during beta-adrenergic blockade. *Med. Sci. Sports Exerc.* 22:222–228, 1990.
207. PESCATELLO, L. S., A. E. FARGO, C. N. LEACH, Jr., and H. H. SCHERZER. Short-term effect of dynamic exercise on arterial blood pressure. *Circulation* 83:1557–1561, 1991.
208. PESCATELLO, L. S., B. MILLER, P. G. DANIAS, et al. Dynamic exercise normalizes resting blood pressure in mildly hypertensive premenopausal women. *Am. Heart J.* 138:916–921, 1999.
209. PESCATELLO, L. S., and J. M. KULIKOWICH. The after effects of dynamic exercise on ambulatory blood pressure. *Med. Sci. Sports Exerc.* 33:1855–1861, 2001.
210. PESCATELLO, L. S., L. BAIROS, J. L. VANHEEST, et al. Postexercise hypotension differs between white and black women. *Am. Heart J.* 145:364–370, 2003.
211. PIEPOLI, M., A. J. S. COATS, S. ADAMOPOULOS, et al. Persistent peripheral vasodilation and sympathetic activity in hypotension after maximal exercise. *J. Appl. Physiol.* 75:1807–1814, 1993.
212. POLLOCK, M. L., B. A. FRANKLIN, G. J. BALADY, et al. AHA Science Advisory. 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; position paper endorsed by the American College of Sports Medicine. *Circulation* 101:828–833, 2000.
213. POSNER, J. D., K. M. GORMAN, L. W. LANDSBERG, et al. Low to moderate intensity endurance training in healthy older adults: physiological responses after four months. *J. Am. Geriatr. Soc.* 40:1–7, 1992.
214. QUINN, T. J. Twenty-four hour, ambulatory blood pressure responses following exercise: impact of exercise intensity. *J. Hum. Hypertens.* 14:547–553, 2000.
215. RADAELLI, A., M. PIEPOLI, S. ADAMOPOULOS, et al. Effects of mild physical activity, atenolol and the combination on ambulatory blood pressure in hypertensive subjects. *J. Hypertens.* 10:1279–1282, 1992.
216. RANKINEN, T., J. GAGNON, L. PERUSSE, et al. AGT M235T and ACE ID polymorphisms and exercise blood pressure in the HERITAGE Family Study. *Am. J. Physiol. Heart Circ. Physiol.* 279:H368–374, 2000.
217. RANKINEN, T., L. PERUSSE, J. GAGNON, et al. Angiotensin-converting enzyme ID polymorphism and fitness phenotype in the HERITAGE Family Study. *J. Appl. Physiol.* 88:1029–1035, 2000.
218. RANKINEN, T., T. RICE, L. PERUSSE, et al. NOS3 Glu298Asp genotype and blood pressure response to endurance training: the HERITAGE family study. *Hypertens.* 36:885–889, 2000.
219. RAO, S. P., H. L. COLLINS, and S. E. DICARLO. Postexercise alpha-adrenergic receptor hyporesponsiveness in hypertensive rats is due to nitric oxide. *Am. J. Physiol. Regul. Integr. Comp. Physiol.* 282:R960–R968, 2002.
220. RAY, C. A., and K. M. HUME. Sympathetic neural adaptations to exercise training in humans: insights from microneurography. *Med. Sci. Sports Exerc.* 30:387–391, 1998.
221. RAY, C. A. Sympathetic adaptations to one-legged training. *J. Appl. Physiol.* 86:1583–1587, 1999.
222. RAY, C. A., and D. I. CARRASCO. Isometric handgrip training reduces arterial pressure at rest without changes in sympathetic nerve activity. *Am. J. Physiol. Heart Circ. Physiol.* 279:H245–H249, 2000.

223. READY, A. E., B. NAIMARK, J. DUCAS, et al. Influence of walking volume on health benefits in women post-menopause. *Med. Sci. Sports Exerc.* 28:1097–1105, 1996.
224. RICE, T., P. AN, J. GAGNON, et al. Heritability of heart rate and blood pressure response to exercise training in the HERITAGE Family Study. *Med. Sci. Sports Exerc.* 34:972–979, 2002.
225. RICHARDSON, P. D., M. J. DAVIES, and G. V. BORN. Influence of plaque configuration and stress distribution on fissuring of coronary atherosclerotic plaques. *Lancet* 2:941–944, 1989.
226. RIVERA, M. A., M. ECHEGARAY, T. RANKINEN, et al. TGF-beta(1) gene-race interactions for resting and exercise blood pressure in the HERITAGE Family Study. *J. Appl. Physiol.* 91:1808–1813, 2001.
227. ROLTSCH, M. H., T. MENDEZ, K. R. WILUND, and J. M. HAGBERG. Acute resistive exercise does not affect ambulatory blood pressure in young men and women. *Med. Sci. Sports Exerc.* 33:881–886, 2001.
228. RONDA, M. U. P. B., M. J. N. N. ALVES, A. M. F. W. BRAGA, et al. Postexercise blood pressure reduction in elderly hypertensive patients. *J. Am. Coll. Cardiol.* 39:676–682, 2002.
229. RUECKERT, P. A., P. R. SLANE, D. L. LILLIS, and P. HANSON. Hemodynamic patterns and duration of post-dynamic exercise hypotension in hypertensive humans. *Med. Sci. Sports Exerc.* 28:24–32, 1996.
230. SAKAI, T., M. IDEISHI, S. MIURA, et al. Mild exercise activates renal dopamine system in mild hypertensives. *J. Hum. Hypertens.* 12:355–362, 1998.
231. SAWADA, S., H. TANAKA, M. FUNAKOSHI, M. SHINDO, S. KONO, and T. ISHIKO. Five-year prospective study on blood pressure and maximal oxygen uptake. *Clin. Exp. Pharmacol. Physiol.* 20:483–487, 1993.
232. SCHETTINI, C., M. BIANCHI, N. FERNANDO, E. SANDOYA, and H. SENRA. Ambulatory blood pressure normality and comparison with other measurements. *Hypertens.* 34(Pt 2):818–825, 1999.
233. SCHIFFRIN, E. L. Endothelin and endothelin antagonists in hypertension. *J. Hypertens.* 16:1891–1895, 1998.
234. SEALS, D. R., J. M. HAGBERG, B. F. HURLEY, A. A. EHSANI, and J. O. HOLLOSZY. Endurance training in older men and women. I. Cardiovascular responses to exercise. *J. Appl. Physiol.* 57:1024–1029, 1984.
235. SEALS, D. R., and M. J. REILING. Effect of regular exercise on 24-hour arterial pressure in older hypertensive humans. *Hypertens.* 18:583–592, 1991.
236. SEXTON, W. L., R. J. KORTHUIS, and M. H. LAUGHLIN. High-intensity exercise training increases vascular transport capacity of rat hindquarters. *Am. J. Physiol.* 254:H274–H278, 1988.
237. SEXTON, W. L., and M. H. LAUGHLIN. Influence of endurance exercise training on distribution of vascular adaptations in rat skeletal muscle. *Am. J. Physiol.* 266:H483–H490, 1994.
238. SHAPER, A. G., G. WANNAMETHEE, and M. WALKER. Physical activity, hypertension and risk of heart attack in men without evidence of ischaemic heart disease. *J. Hum. Hypertens.* 8:3–10, 1994.
239. SHENBERGER, J. S., G. J. LEAMAN, M. M. NEUMYER, T. I. MUSCH, and L. I. SINOWAY. Physiologic and structural indices of vascular function in paraplegics. *Med. Sci. Sports Exerc.* 22:96–101, 1990.
240. SILVA, G. J., P. C. BRUM, C. E. NEGRAO, and E. M. KRIEGER. Acute and chronic effects of exercise on baroreflexes in spontaneously hypertensive rats. *Hypertens.* 30:714–719, 1997.
241. SINGH, J. P., M. G. LARSON, T. A. MANOLIO, et al. Blood pressure response during treadmill testing as a risk factor for new-onset hypertension: the Framingham Heart Study. *Circulation* 99:1831–1836, 1999.
242. SOMERS, V. K., J. CONWAY, J. JOHNSTON, and P. SLEIGHT. Effects of endurance training on baroreflex sensitivity and blood pressure in borderline hypertension. *Lancet* 337:1363–1368, 1991.
243. SOMERS, V. K., J. CONWAY, and A. COATS. Postexercise hypotension is not sustained in normal and hypertensive humans. *Hypertens.* 18:211–215, 1991.
244. SPIER, S. A., M. H. LAUGHLIN, and M. D. DELP. Effects of acute and chronic exercise on vasoconstrictor responsiveness of rat abdominal aorta. *J. Appl. Physiol.* 87:1752–1757, 1999.
245. STAESSEN, J., A. AMERY, and R. FAGARD. Isolated systolic hypertension in the elderly. *J. Hypertens.* 9:393–405, 1990.
246. STAESSEN, J. A., J. GASOWSKI, J. G. WANG, et al. Risks of untreated and treated isolated systolic hypertension in the elderly: meta-analysis of outcome trials. *Lancet* 355:865–872, 2000.
247. STAMLER, J., G. ROSE, R. STAMLER, P. ELLIOTT, A. DYER, and M. MARMOT. Intersalt study findings: public health and medical care implications. *Hypertens.* 14:570–577, 1989.
248. STARR, M. Brave heart, broken heart. *Newsweek* 126(34):70–71, 1995.
249. TADDEI, S., A. VIRDIS, L. GHIADONI, I. SUDANO, M. NOTARI, and A. SALVETTI. Vasoconstriction to endogenous endothelin-1 is increased in the peripheral circulation of patients with essential hypertension. *Circulation* 100:1680–1683, 1999.
250. TAYLOR-TOLBERT, N. S., D. R. DENGEL, M. D. BROWN, et al. Ambulatory blood pressure after acute exercise in older men with essential hypertension. *Am. J. Hypertens.* 13:44–51, 2000.
251. THOMPSON, P. D. The cardiovascular complications of vigorous physical activity. *Arch. Intern. Med.* 156:2297–2302, 1996.
252. THOMPSON, P. D., S. F. CROUSE, B. GOODPASTER, D. KELLEY, N. MOYNA, and L. PESCATELLO. The acute versus the chronic response to exercise. *Med. Sci. Sports Exerc.* 33:S438–S445; discussion S452–S453, 2001.
253. THOREN, P., J. S. FLORAS, P. HOFFMANN, and D. R. SEALS. Endorphins and exercise: physiological mechanisms and clinical implications. *Med. Sci. Sports Exerc.* 22:417–428, 1990.
254. TIPTON, C. M., R. D. MATTHES, K. D. MARCUS, K. A. ROWLETT, and J. R. LEININGER. Influences of exercise intensity, age, and medication on resting systolic blood pressure of SHR populations. *J. Appl. Physiol.* 55:1305–1310, 1983.
255. TSAI, J. C., J. C. LIU, C. C. KAO, et al. Beneficial effects on blood pressure and lipid profile of programmed exercise training in subjects with white coat hypertension. *Am. J. Hypertens.* 15:571–576, 2002.
256. TSUTSUMI, T. *The Effects of Strength Training on Mood, Self-Efficacy, Cardiovascular Reactivity and Quality of Life in Older Adults*. Boston: Boston University, 1997.
257. U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES. Physical activity and health: a report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, and National Center for Chronic Disease Prevention and Health Promotion, 1996.
258. URATA, H., Y. TANABE, A. KIYONAGA, et al. Antihypertensive and volume-depleting effects of mild exercise on essential hypertension. *Hypertens.* 9:245–252, 1987.
259. VAITKEVICIUS, P. V., J. L. FLEG, J. H. ENGEL, et al. Effects of age and aerobic capacity on arterial stiffness in healthy adults. *Circulation* 88:1456–1462, 1993.
260. VAN HOOF, R., P. HESPEL, R. FAGARD, P. LIJNEN, J. STAESSEN, and A. AMERY. Effect of endurance training on blood pressure at rest, during exercise and during 24 h, during exercise and during 24 h in sedentary men. *Am. J. Cardiol.* 63:945–949, 1989.
261. VAN HOOF, R., F. MACOR, P. LIJNEN, et al. Effect of strength training on blood pressure measured at various conditions in sedentary men. *Int. J. Sports Med.* 17:415–422, 1996.
262. VANDONGEN, R., D. A. JENNER, C. THOMPSON, et al. A controlled evaluation of a fitness and nutrition intervention program on cardiovascular health in 10- to 12-year old children. *Prev. Med.* 24:9–22, 1995.
263. VASAN, R. S., M. G. LARSON, E. P. LEIP, et al. Impact of high-normal blood pressure on the risk of cardiovascular disease. *N. Engl. J. Med.* 345:1291–1297, 2001.
264. VASAN, R. S., A. BEISER, S. SESHADRI, et al. Residual lifetime risk for developing hypertension in middle-aged women and men: the Framingham Heart Study. *JAMA* 287:1003–1010, 2002.
265. VERDECCHIA, P. Prognostic value of ambulatory blood pressure: current evidence and clinical implications. *Hypertens.* 35:844–851, 2000.
266. WALLACE, J. P., P. G. BOGLE, B. A. KING, J. B. KRASNOFF, and C. A. JASTREMSKI. A comparison of 24-h average blood pressures and blood pressure load following exercise. *Am. J. Hypertens.* 10:728–734, 1997.



267. WALLACE, J. P., P. G. BOGLE, B. A. KING, J. B. KRASNOFF, and C. A. JASTREMSKI. The magnitude and duration of ambulatory blood pressure reduction following acute exercise. *J. Hum. Hypertens.* 13:361–366, 1999.
268. WALLACE, J. P. Exercise in hypertension: a clinical review. *Sports Med.* 33:585–598, 2003.
269. WALLIN, B. G., and G. SUNDLOF. A quantitative study of muscle nerve sympathetic activity in resting normotensive and hypertensive subjects. *Hypertens.* 1:67–77, 1979.
270. WANG, J. S., C. J. JEN, and H. I. CHEN. Effects of chronic exercise and deconditioning on platelet function in women. *J. Appl. Physiol.* 83:2080–2085, 1997.
271. WHELTON, P. K., J. HE, L. J. APPEL, et al. Primary prevention of hypertension: clinical and public health advisory from The National High Blood Pressure Education Program. *JAMA* 288:1882–1888, 2002.
272. WHELTON, S. P., A. CHIN, X. XIN, and J. HE. Effect of aerobic exercise on blood pressure: a meta-analysis of randomized, controlled trials. *Ann. Intern. Med.* 136:493–503, 2002.
273. WIEGMAN, D. L., P. D. HARRIS, I. G. JOSHUA, and F. N. MILLER. Decreased vascular sensitivity to norepinephrine following exercise training. *J. Appl. Physiol.* 51:282–287, 1981.
274. WIJNEN, J. A., H. KUIPERS, M. J. KOOL, et al. Vessel wall properties of large arteries in trained and sedentary subjects. *Basic Res. Cardiol.* 86(Suppl. 1):25–29, 1991.
275. WIJNEN, J. A. G., M. J. F. KOOL, M. A. VAN BAAK, et al. Effect of exercise training on ambulatory blood pressure. *Int. J. Sports Med.* 15:10–15, 1994.
276. WILCOX, R. G., T. BENNETT, A. M. BROWN, and I. A. MACDONALD. Is exercise good for high blood pressure? *Br. Med. J.* 285:767–769, 1982.
277. WILEY, R. L., C. L. DUNN, R. H. COX, N. A. HUEPPCHEN, and M. S. SCOTT. Isometric exercise training lowers resting blood pressure. *Med. Sci. Sports Exerc.* 24:749–754, 1992.
278. WILLIAMS, M. A., J. L. FLEG, P. A. ADES, et al. Secondary prevention of coronary heart disease in the elderly (with emphasis on patients  $\geq 75$  years of age). An American Heart Association Scientific Statement from the Council on Clinical Cardiology Subcommittee on Exercise, Cardiac Rehabilitation, and Prevention. *Circulation* 105:1735–1743, 2002.
279. WILLIAMS, S. M., J. H. ADDY, J. A. PHILLIPS, III, et al. Combinations of variations in multiple genes are associated with hypertension. *Hypertens.* 36:2–6, 2000.
280. WILICH, S. N., M. LEWIS, H. LÖWEL, H. R. ARNTZ, F. S. CHUBERT, and R. SCHRÖDER. Physical exertion as a trigger of acute myocardial infarction. *N. Engl. J. Med.* 329:1684–1690, 1993.
281. WILMORE, J. H., P. R. STANFORTH, J. GAGNON, et al. Heart rate and blood pressure changes with endurance training: the Heritage Family Study. *Med. Sci. Sports Exerc.* 33:107–116, 2001.
282. WOOD, R. H., R. REYES, M. A. WELSCH, et al. Concurrent cardiovascular and resistance training in healthy older adults. *Med. Sci. Sports Exerc.* 33:1751–1758, 2001.
283. WORLD HEALTH ORGANIZATION-INTERNATIONAL SOCIETY OF HYPERTENSION. 2003 World Health Organization (WHO)/International Society of Hypertension (ISH) statement on the management of hypertension. *J. Hypertens.* 21:1983–1992, 2003.
284. YEN, M. H., J. H. YANG, J. R. SHEU, Y. M. LEE, and Y. A. DING. Chronic exercise enhances endothelium-mediated dilation in spontaneously hypertensive rats. *Life Sci.* 57:2205–2213, 1995.