

Exercise and Heart Disease

James C. Puffer, MD

Professor and Chief
Division of Sports Medicine
Department of Family Medicine
UCLA School of Medicine
Los Angeles, California

Atherosclerotic coronary artery disease (CAD) is a leading cause of death in the United States. It accounts for myocardial infarction in ~1.5 million Americans annually and results in ~500,000 deaths yearly. Half of these deaths occur prior to patients being admitted to a hospital. A dramatic reduction in mortality related to atherosclerotic CAD has occurred in the last decade. This reduction has resulted from significant advances in both the medical and surgical management of this disease as well as from increased efforts to modify risk factors known to increase the likelihood of developing CAD. Modifiable risk factors include cigarette smoking, hypercholesterolemia, and hypertension. More recently, physical inactivity has been found to be a significant modifiable risk factor that can influence the development of CAD. This article reviews some of the observational data that support these conclusions, discusses the role of exercise in the prevention of CAD in select groups, and reviews some of the mechanisms by which exercise may modify the development of CAD.

EPIDEMIOLOGY

A large body of literature supports the view that physical activity can significantly alter the risk for coronary artery disease (CAD). A meta-analysis performed by Powell and colleagues, which evaluated 121 articles from 54 studies, reached the following conclusions: (a) physically active subjects had ~ half the incidence of CAD as those who were sedentary; (b) no studies showed a higher rate of CAD among the most active subjects; (c) two thirds of the studies adequately demonstrated that physical inactivity precedes the onset of the disease; and (d) in the 18 studies that presented data evaluating multiple levels of activity, a strong inverse relationship between physical activity and the development of CAD was demonstrated (ie, those who were more physically active had a lower likelihood of disease). Given this information, physical inactivity seems to play an equally important role in contributing to the relative risk of developing CAD as do the long accepted risk

factors (eg, smoking, hypercholesterolemia, and hypertension). The relative contributions of each of these risk factors are provided in the **Table**.

More striking, however, is the large population potentially at risk of CAD because of physical inactivity. It has been shown that only ~20% of our adult population engage in physical activity at levels sufficient to promote health and that ~25% of adults are completely sedentary; the remaining 55% are inadequately active. These data are remarkably consistent across most age groups. Since almost 75% of our population may be at significant risk for CAD given their relative inactivity, encouraging and promoting physical activity could have a profound influence on public health. These data are demonstrated in the **Figure**.

A review of selected studies provides useful information on how age, gender, intensity of exercise, and genetics influence the effect of physical activity on CAD.

TABLE.

RISK FACTORS FOR ATHEROSCLEROTIC CORONARY ARTERY DISEASE

<i>Risk Factor</i>	<i>Relative Risk</i>
• Smoking (>1 pack/d)	2.5
• Hypercholesterolemia (Total cholesterol > 6.93 mmol/L)	2.4
• Hypertension (Systolic blood pressure > 150 mm Hg)	2.1
• Physical inactivity	1.9

AGE

The Harvard Alumni Study examined the physical activity and lifestyle behaviors of almost 17,000 Harvard alumni. Sedentary men were found to be at 64% higher risk for a first myocardial infarction (MI) compared with those men who expended 2000 kcal of energy per week in physical activity. Death rates declined as energy expenditure increased from 500 to 3500 kcal per week. When other known risk factors for disease were controlled for, mortality rates remained lower in the physically active. Most interesting in this study were the age-stratified data that showed quite clearly the continued benefit of exercise for those in their 60s and 70s. The decline in death rates as energy expenditure increased remained consistent with that found in younger age groups. Even more important, the largest reduction in death rate occurred when comparing the most sedentary in the 60- to 70-year age group with those who were only moderately active.

GENDER

Much of the early data on physical activity and modification of risk for heart disease originated from observational studies in which men were the subjects. Subsequent work has shown that similar benefits may occur in women. The Institute for Aerobics Research Longitudinal Study examined the effects of physical fitness on mortality in both men and women. In this study, fitness was measured by a maximal exercise tolerance test and the cohort was followed for an average of 8 years. Other risk factors for CAD were controlled for in the data analysis. The data demonstrated an age-adjusted decline in mortality across varying fitness

levels for both men and women. A low rate of death from cardiovascular (CV) disease was found among higher fitness groups. As in the Harvard Alumni Study, the greatest reduction in risk occurred in simply moving from the least-fit fitness group to the moderately active fitness group.

The Iowa Women’s Health Study, which involved postmenopausal women recruited from a random sample of 55- to 69-year-old women drivers in Iowa, analyzed 40,417 women for whom

KEY POINT

Much of the early data on physical activity and modification of risk for heart disease originated from observational studies in which men were the subjects. More recent studies have shown that similar benefits may occur in women.

physical activity and cigarette smoking status were available at the time of entry into the study. The most active group in this study had about half the CV mortality as did the least active group, and women who engaged in moderate activity 4 or more times per week had a 47% lower risk than women who were sedentary or rarely exercised.

INTENSITY OF EXERCISE

While the previous data would suggest that exercise is equally beneficial for men and women and that age does not weaken the relationship between

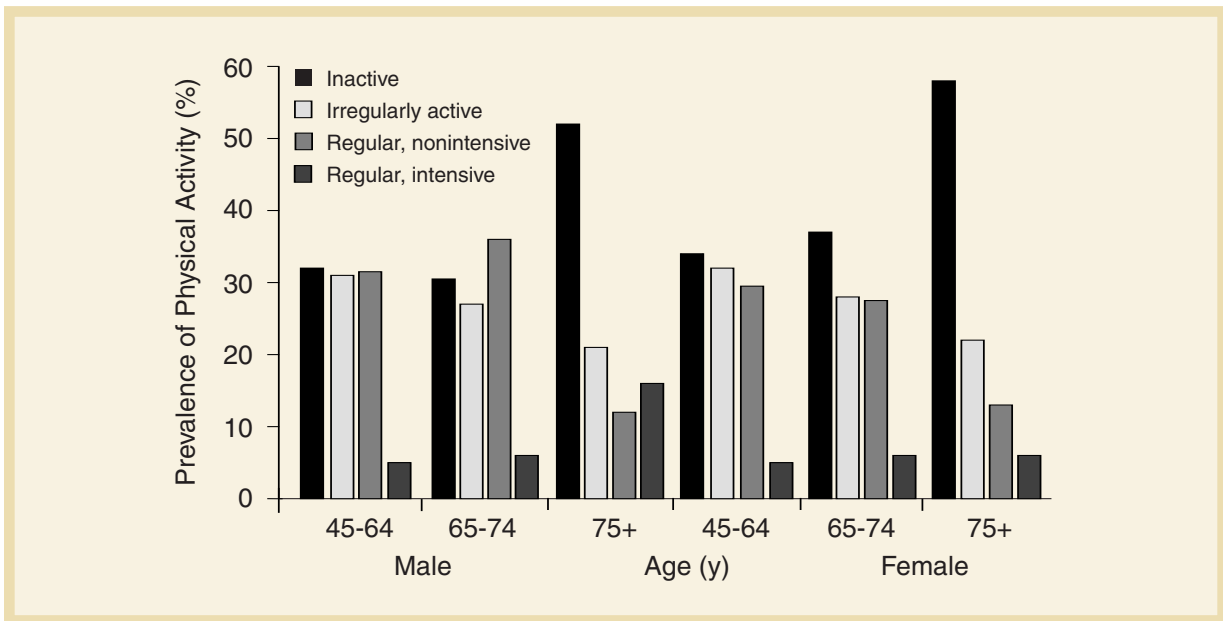


Figure. Prevalence of four patterns of physical activity according to sex and age. Caspersen CJ, DiPietro L. *Med Sci Sports Exerc.* 1991;23:S105.

physical activity and reduction of risk, the findings further suggest that encouraging people who are sedentary to become moderately active can have a profound influence on health. Much interest has been generated in trying to identify a threshold at which this occurs. In the Lipid Research Clinics Mortality Follow-up Study, an attempt was made to quantitate this threshold. This study examined the relationship between physical fitness and mortality from CAD in men 30 to 69 years of age. A baseline risk-factor assessment and exercise tolerance test were performed, and heart rate during stage 2 of the exercise protocol and duration of exercise were used as markers of fitness. A total of >3000 asymptomatic men were followed for an average of 8.5 years. Forty-five CV deaths occurred, and low levels of fitness were associated with a high risk of death due to CV disease. Those with an increment of 35 beats per minute during stage 2 of the exercise protocol or with a decrement of 4.4 minutes in total exercise duration were at ~3 times greater risk of dying from CAD.

While these data may provide quantifiable information about the fitness levels required to achieve significant benefit, they provide little practical information with regard to the amount or level

of activity that is required to reduce risk. A study performed by Lakka and associates provides more useful clinical information on the quantity and type of exercise necessary to modify risk. In this study, men engaging in ≥ 2 hours of conditioning activity

KEY POINT

The Finnish Twin Cohort Study provided sound evidence that physical activity reduces CV mortality independent of genetic influence.

per week had a 60% lower risk for MI after controlling for multiple confounding factors. A similar reduction in risk was found in men with moderate fitness levels, as measured by maximum oxygen consumption.

GENETIC FACTORS

While the studies that have been cited demonstrate quite convincingly the powerful influence that physical activity has on modification of risk for CAD, they provide little information with regard to

the role that genetic factors may play in modifying or influencing this effect. The Finnish Twin Cohort Study provides data to address this issue. In this study, almost 8000 healthy men and 8000 healthy women (aged 25 to 64 years) responded to a questionnaire on physical activity habits and known predictors of mortality at baseline. Those who exercised ≥ 6 times per month at an intensity corresponding to at least vigorous walking for a mean duration of 30 minutes were classified as conditioning exercisers, while those who reported no leisure-time physical activity were classified as sedentary. Subjects who fell between these 2 groups were classified as occasional exercisers. All causes of mortality and discordant deaths among same-sex twin pairs were collected from 1977 through 1994. Among the 444 twin pairs who were healthy at baseline and discordant for death, mortality was reduced by 34% in occasional exercisers and by 56% in the conditioning exercisers compared with those who were sedentary. This effect remained significant after controlling for other potential causes of mortality. These data provide sound evidence that physical activity reduces CV mortality independent of genetic influence.

SUDDEN DEATH

Much has been made of the risk of sudden death during exercise. Several studies provide useful insight into this phenomenon. One such study, performed by Thompson and associates, examined jogging deaths in Rhode Island from 1975 to 1980. Twelve deaths occurred, 11 of which were attributable to CAD in men >30 years of age. The prevalence of jogging in Rhode Island was determined by using random telephone surveys. Using these data, the researchers estimated that the incidence of death during jogging was 1 death per year for every 7620 joggers or every 396,000 man-hours of jogging. This rate was 7 times higher than that found during sedentary activity. When these data were recalculated to look only at those individuals who were not known to have pre-existing CAD, the death rate was ~ 1 death for every 15,000 joggers or every 800,000 man-hours of jogging. Thus, while the relative risk of dying during physical activity is increased, the absolute risk is incredibly small for asymptomatic men.

MYOCARDIAL INFARCTION

Additional studies have looked at the risk of acute MI during physical exertion. Mittleman and colleagues demonstrated that the relative risk for MI increased about sixfold during the first hour after heavy exertion. Interestingly, the relative risk during the first hour after heavy exertion was only 2.4 for those exercising at least 5 times weekly, but was 107 for those exercising less than once weekly.

Similar results were found in a study by Willich and coworkers, in which the overall relative risk for infarction related to heavy exertion was increased \sim twofold but was significantly higher among those who were less physically fit. While both of these studies demonstrated, after controlling for confounding factors, that physical exertion independently predicted MI, it is important to note that only 4% to 7% of all infarctions in both studies were associated with exertion. Furthermore, when these data are used to calculate the absolute risk for MI in a 50-year-old, nonsmoking, nondiabetic man who exercises, he has about a 1 in a million chance of suffering an infarction during physical activity.

KEY POINT

It has been shown that people who exercise have lower triglyceride levels and higher HDL concentrations than those who are physically inactive.

SERUM LIPID MODIFICATION

Given the data that have demonstrated a beneficial effect of exercise on modifying the risk for CAD, what mechanisms might explain this change in risk? Early animal studies by Kramsch and associates have suggested that exercise has a profound effect on lipoprotein subfractions. Kramsch studied the effect of moderate conditioning on the development of CAD in monkeys. After randomization, exercise and sedentary groups of monkeys were fed highly atherogenic diets, and electrocardiograms and serum lipids were measured serially. Angiography was performed postmortem. The mean total serum cholesterol level

in both the sedentary and exercise groups measured ~600 mg/dL. However, the exercising group had higher high-density lipoprotein (HDL) levels as well as lower low-density lipoprotein cholesterol levels and lower triglyceride levels. Only the sedentary monkeys demonstrated ischemic electrocardiogram changes, suffered sudden death, and had evidence of coronary artery narrowing on angiography.

It is known that an HDL receptor exists for unesterified cholesterol and for breakdown products of chylomicrons and very low-density lipoprotein particles. HDL serves as the only site of cholesterol esterification in plasma, and these HDL cholesterol esters are then provided to other lipoprotein particles via core lipid transfer. This mechanism serves as a means of reverse cholesterol transport from the endothelium. It has been shown that people who exercise have lower triglyceride levels and higher HDL concentrations than do those who are physi-

KEY POINT

Recent research has shown that physical activity can actually cause regression of preexisting disease in persons with CAD.

cally inactive. This is mediated acutely by increased lipoprotein lipase activity and enhanced fat clearance in people who exercise. Furthermore, HDL protein survival is ~25% higher in athletes compared with sedentary controls. These modifications can result in reduced risk for atherogenesis and stabilization of existing plaque in the coronary arteries, thereby modifying the risk for plaque rupture and infarction.

REGRESSION OF EXISTING DISEASE

Recent research has shown that physical activity can actually cause regression of preexisting disease in persons with CAD. Hambrecht and colleagues studied the effect of physical activity on CAD lesions in a prospective, randomized, controlled trial. Eight of 29 subjects who exercised demonstrated regression of disease compared with only 2 of 33 controls.

KEY POINT

Studies show that exercise can reduce the vasoconstrictor response of damaged endothelium in patients with both CAD and hypertension.

Expenditure of ~2200 kcal per week was necessary to demonstrate regression, and expenditure of 1530 kcal per week was necessary to halt the progression of disease. Additional work has shown that significant reduction in stress-induced ischemia, as measured by exercise testing, is not limited to only those individuals with demonstrated regression of disease on quantitative angiography.

ENDOTHELIAL FUNCTION

Alteration in endothelial function may explain some of the study findings. It is known that the endothelium produces vasoactive substances that regulate both smooth muscle function and structure. Damage to the endothelium leads to vasoconstrictor responses to vasoactive substances that normally cause vasodilation. In healthy endothelium, vasoactive agents stimulate the production of nitric oxide, which inhibits vascular smooth muscle contraction. When the endothelium is damaged, however, it does not produce sufficient amounts of nitric oxide; therefore, these same vasoactive substances will directly activate smooth muscle contraction. This action accounts for the paradoxical vasoconstriction that occurs in diseased and stenotic coronary artery segments in response to endothelium-dependent stimuli such as sympathetic stimulation. It has been shown that exercise can reduce the vasoconstrictor response of damaged endothelium in patients with both CAD and hypertension. Similarly, enhanced endothelium-dependent vasodilation has been shown in older master runners as well as in normotensive subjects, which is probably mediated by an increase in the release of endothelium-derived nitric oxide.

EXERCISE PRESCRIPTION

Recommendations for physical activity have

KEY POINT

Recent guidelines established by the Centers for Disease Control and Prevention, the National Institutes of Health, and the American College of Sports Medicine now recommend that children and adults alike should set a minimum goal of doing ≥ 30 minutes of moderately intense physical activity on most, and preferably all, days of the week.

changed dramatically in the last decade. In 1990, the American College of Sports Medicine recommended that exercise should occur at 60% to 90% of the maximum heart rate for 20 to 60 minutes in duration and this activity should be performed 3 to 5 days weekly. As a result of the substantial body of literature that has shown significant improvement in health and wellness with moderate levels of physical activity, recent guidelines established by the Centers for Disease Control and Prevention, the National Institutes of Health, and the American College of Sports Medicine now recommend that children and adults alike should set a goal of doing ≥ 30 minutes of moderately intense physical activity on most, and preferably all, days of the week. While these should be the minimum requirements, review of data cited previously suggests that those who meet these requirements would gain additional benefit from more vigorous activity.

It is known that most adults would like to be more physically active. However, when they are asked if their physician has ever recommended physical activity, almost 70% indicate that they have never been counseled about the beneficial aspects of exercise. Given the demonstrated benefits of regular physical activity, it is essential that discussions about physical activity between physicians and their patients occur frequently.

One of the major factors affecting exercise is the issue of compliance. A lack of time and family responsibilities are often cited as major barriers to exercise. Since studies show that women are moti-

vated by social factors to exercise while for men convenience is the primary motivator, these issues should be specifically addressed by the clinician. Although the optimum strategy for prescribing exercise has not been found, recent data suggest that a written prescription together with educational materials will produce modest short-term improvement in self-reported physical activity levels among sedentary patients.

SUMMARY

Considerable evidence exists to demonstrate that regular physical activity can significantly modify and alter the risk for CAD. This effect is independent of age, gender, or genetic influence and occurs at relatively modest levels of physical activity. Modification of oxidative stress on the endothelium of coronary arteries by both alteration of the lipid profile and enhanced production of nitric oxide may explain the mechanisms responsible for this change in risk. Physicians should regularly and frequently counsel their patients on the benefits of regular physical activity. Compliance with the exercise prescription can be enhanced by carefully exploring the factors that serve as barriers to exercise and by providing written educational material to reinforce important points. Regular follow-up and assessment of a patient's exercise program can reinforce compliance with the regimen.

SUGGESTED READING

American College of Sports Medicine position stand. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness in healthy adults. *Med Sci Sports Exerc.* 1990;22:265–274.

Blair SN, Kampert JB, Kohl HW III, et al. Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. *JAMA.* 1996;276:205–210.

Blair SN, Kohl HW III, Paffenbarger RS Jr, et al. Physical fitness and all-cause mortality: a prospective study of healthy men and women. *JAMA.* 1989;262:2395–2401.

Ekelund LG, Haskell WL, Johnson JL, et al. Physical fitness as a predictor of cardiovascular mortality in asymptomatic North American men: The Lipid Research Clinics Mortality Follow-up Study. *N Engl J Med.* 1988;319:1379–1384.

Hambrecht R, Niebauer J, Marburger C, et al. Various

intensities of leisure time physical activity in patients with coronary artery disease: effects on cardiorespiratory fitness and progression of coronary atherosclerotic lesions. *J Am Coll Cardiol*. 1993;22:468–477.

Hambrecht R, Wolf A, Gielen S, et al. Effect of exercise on coronary endothelial function in patients with coronary artery disease. *N Engl J Med*. 2000;342:454–460.

Harris SS, Caspersen CJ, DeFries GH, Estes EH. Physical activity counseling for healthy adults as a primary preventive intervention in the clinical setting. Report for the US Preventive Services Task Force. *JAMA*. 1989;261:3588–3598.

Higashi Y, Sasaki S, Kurisu S, et al. Regular aerobic exercise augments endothelium-dependent vascular relaxation in normotensive as well as hypertensive subjects: role of endothelium-derived nitric oxide. *Circulation*. 1999;100:1194–1201.

Kramsch DM, Aspen AJ, Abramowitz BM, et al. Reduction of coronary atherosclerosis by moderate conditioning exercise in monkeys on an atherogenic diet. *N Engl J Med*. 1981;305:1483–1489.

Kujala UM, Kaprio J, Sarna S, Koskenvuo M. Relationship of leisure-time physical activity and mortality: the Finnish Twin Cohort. *JAMA*. 1998;279:440–444.

Kushi LH, Fee RM, Folsom AR, et al. Physical activity and mortality in postmenopausal women. *JAMA*. 1997;277:1287–1292.

Lakka TA, Venalainen JM, Rauramaa R, et al. Relation of leisure-time physical activity and cardiorespiratory fitness to the risk of acute myocardial infarction in men. *N Engl J Med*. 1994;330:1549–1554.

Lee I-M, Paffenbarger RS. Preventing coronary heart disease: the role of physical activity. *Phys Sportsmed*. 2001;29:37–52.

McBride P, Einerson J, Hanson P, et al. Exercise and the primary prevention of coronary heart disease. *Med Exerc Nutr Health*. 1992;1:5–15.

Mittleman MA, Maclure M, Tofler GH, et al. Triggering of acute myocardial infarction by heavy physical exer-

tion: protection against triggering by regular exertion. *N Engl J Med*. 1993;329:1677–1683.

NIH Consensus Development Panel on Physical Activity and Cardiovascular Health. Physical activity and cardiovascular health. *JAMA*. 1996;276:241–246.

Paffenbarger RS, Hyde RT, Wing AL, Hsien CC. Physical activity, all-cause mortality, and longevity of college alumni. *N Engl J Med*. 1986;314:605–613.

Paffenbarger RS, Hyde RT, Wing AL, et al. The association of changes in physical activity level and other lifestyle characteristics with mortality among men. *N Engl J Med*. 1993;328:538–545.

Powell KE, Thompson PD, Caspersen CJ, Kendrick JS. Physical activity and the incidence of coronary heart disease. *Annu Rev Public Health*. 1987;8:253–287.

Rinder MR, Spina RJ, Ehsani AA. Enhanced endothelium-dependent vasodilation in older endurance-trained men. *J Appl Physiol*. 2000;88:761–766.

Shoenhair CL, Wells CL. Women, physical activity and coronary heart disease: a review. *Med Exerc Nutr Health*. 1995;4:200–216.

Smith BJ, Bauman AE, Bull FC, et al. Promoting physical activity in general practice: a controlled trial of written advice and information materials. *Br J Sports Med*. 2000;34:262–267.

Thompson PD, Cullinane EM, Sady SP, et al. Modest changes in high-density lipoprotein concentration and metabolism with prolonged exercise training. *Circulation*. 1988;78:25–34.

Thompson PD, Funk EJ, Carleton RA, Sturner WQ. Incidence of death during jogging in Rhode Island from 1975 through 1980. *JAMA*. 1982;247:2535–2538.

Wee CC, McCarthy EP, Davis RB, Phillips RS. Physician counseling about exercise. *JAMA*. 1999;282:1583–1588.

Willich SN, Lewis M, Lowel H, et al. Physical exertion as a trigger of acute myocardial infarction. *N Engl J Med*. 1993;329:1684–1690.



Dialogue Box

ADVISORY BOARD

How long do the benefits of aerobic fitness last in someone who was physically fit in the past but is now sedentary?

PUFFER

Although the precise period of time is not known, we do know that if such a person were to become sedentary, the previous level of fitness has absolutely no protective effect whatsoever. The best data in this regard come from a secondary data analysis of the Harvard Alumni Study conducted by Paffenbarger and colleagues. This study looked at 4 discreet subsets in this cohort—former Harvard athletes who were now sedentary, former athletes who continued to be physically active, nonathletes who were sedentary, and nonathletes who were physically active. When CV morbidity and mortality were compared among these 4 groups, the group with the highest morbidity and mortality were the former athletes who were sedentary. The group best off were the nonathletes who were currently physically active. The next best were the athletes who were still active followed by the nonathletes who were sedentary. On the basis of these data, it is quite clear that previous high levels of fitness confer no long-lasting protection against the development of CV disease and that regular ongoing physical activity, even at relatively low thresholds, can have a significant impact on influencing the risk for both CV morbidity and mortality.

ADVISORY BOARD

Is it believed that the increased CV risk seen in sedentary former athletes stems from a rebound phenomenon?

PUFFER

Not really. For example, if you look at Saltin's work, which longitudinally followed former ath-

letes and nonathlete controls, the fitness levels, in terms of measured maximum oxygen consumption, of former athletes who were no longer physically active were still higher than those of controls who were sedentary. Thus, at least on the basis of fitness as measured by maximum oxygen consumption, a rebound phenomenon resulting in a greater loss of fitness does not appear to occur.

ADVISORY BOARD

The bottom line would seem to be to stay fit for life.

PUFFER

The bottom line is that physical activity at modest levels confers significant protection as long as it is fairly consistent and regular.

ADVISORY BOARD

How effective is exercise in raising HDL cholesterol (HDL-C)?

PUFFER

The beneficial impact of regular exercise on HDL-C is modest at best—certainly much less than the impact of lipid-lowering drugs such as a statin agent (3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitor) or niacin. Whereas the statins can be expected to raise HDL-C 5% to 15% and niacin can raise HDL-C by 10% to 30%, the impact of exercise pales in comparison to these agents.

ADVISORY BOARD

Can you precisely define what is meant by “moderately intense physical activity,” which is currently recommended for all Americans to reduce CV risk?

PUFFER

As opposed to the fairly rigid exercise prescription



Dialogue Box

recommended in the past, which called for patients to measure their heart rates and exercise within target heart-rate zones, the current recommendation for “moderately intense physical activity” simply calls for the patient to expend 250 to 300 kilocalories per exercise session most if not all days of the week. Generally, this level of exercise can be achieved by 30 minutes or so of brisk walking.

ADVISORY BOARD

For the “goal-oriented” patient, what does this translate into minutes per mile? Walking a mile in 20 minutes or so?

PUFFER

Yes, that’s probably close. But it is important to underscore that we know even if your patients do less than that, they will still derive benefit. Although optimization of CV health occurs with an expenditure between 250 to 300 kilocalories per day, doing less than that still imparts significant benefit.

ADVISORY BOARD

What is the current thinking regarding the ordering of a screening exercise electrocardiogram treadmill study prior to telling a patient to go out and engage at this level of exercise?

PUFFER

Since we’re now recommending exercise at relatively modest levels for persons with no significant risk factors for CV disease, the current recommendation is that it is not necessary to screen patients deemed to be at low risk prior to recommending exercise. This is only logical since this low-risk population will be exercising at levels that pose relatively low risk for an untoward event. Screening such patients would be a waste of money and would result in unnecessary cardiac evaluations for false-positive tests. On the other hand, it makes sense for persons

with known preexisting disease or persons who have significant risk factors for atherosclerosis to be screened with a graded exercise test before recommending exercise because we can use the data from the test to prescribe safe exercise for them—below thresholds at which they might become ischemic.

ADVISORY BOARD

Would you recommend such a study in a 70-year-old man with a negative medical history except for well-controlled hypertension?

PUFFER

No.

ADVISORY BOARD

What about a 70-year-old man with obesity and hypertension?

PUFFER

With those 2 risk factors alone, no. For a 70-year-old male smoker with hypertension, yes.

ADVISORY BOARD

Can you elaborate further on the social factors you think should be dealt with to optimize compliance among male and female patients?

PUFFER

The data we have with regard to compliance demonstrate that for women social factors guarantee continued compliance with an exercise program. As a result, encouraging women to exercise with a partner is more likely to result in long-term compliance than encouraging women to exercise alone. On the other hand, the most important factor that influences compliance in men is convenience—the exercise regimen has to fit into their schedule. If it’s not made convenient for them, the likelihood is that men will not be compliant with the exercise regimen.