Healthy Exercise

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Persons at any age can substantially improve their fitness for work and play through appropriate exercise training. Considerable evidence indicates that physical activity is valuable for weight control, modifying lipids and improving carbohydrate tolerance. Less rigorous scientific data are available for associated long-term blood pressure and psychological changes with habitual exercise. Strenuous physical activity most likely reduces the incidence of coronary heart disease and the detrimental impact of certain chronic diseases on health. Adverse effects may result from a training program, but the major concern is the susceptibility to cardiovascular events during and immediately after exertion. To achieve optimal benefits with minimal risk, exercise must be carefully prescribed within the context of overall health and training objectives. Taken altogether, a distinct rationale exists for regular vigorous exercise as an integral part of a personal health maintenance program.

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More and more people are turning to exercise to insure long-term health. Prestigious health organizations, including the World Health Organization, the American Heart Association, the Public Health Service and the American College of Sports Medicine, have endorsed the concept.[1-4] Working parties of the Royal College of Physicians of London have recommended adoption of a more physically active life-style to reduce the prevalence of coronary heart disease and obesity.[5]

New information has dispelled many of the myths and dogmas associated with training for athletic competition. These data indicate many potentially beneficial physiologic adaptations, but until further clarification of these changes can be obtained, the evidence and control for disease prevention must be mainly inferential.[5-7] Numerous unanswered questions persist on the long-term effects of vigorous exercise, including possible hazards. How much and what kinds of exercise are needed require further investigation to provide appropriate exercise plans for persons at different ages and different levels of health. Nevertheless, it seems likely that regular physical activity is desirable for personal health maintenance and probably reduces the impact of certain chronic diseases.

Generally, the medical community has not kept pace with the growing public awareness of exercise as a health measure. Persons want assistance from their physicians on the therapeutic value of exercise, the kinds of exercise required to obtain health benefits and the necessary evaluations before embarking on an exercise program. Numerous fitness centers and health spas that have sprung up in response to this desire for exercise attempt to fill this void in health counseling. However, exercise must be carefully prescribed within the context of a patient's health. It is important for physicians to familiarize themselves with the physiologic and psychologic consequences of exercise training so that they can best inform patients.

General Health Benefits

Physical activity provides the stimulus required to maintain the structural and functional integrity of the cardiovascular system, the musculoskeletal system and probably the autonomic nervous system and motor neurons.[6-8] With an appropriate exercise schedule, adaptive responses will determine a person's capacity to meet exertional stress. Depending on the exercise routine, an array of other health advantages can accrue over a period of months.

Most Americans get little strenuous exercise at work, or even during leisure hours, at least not enough to induce the metabolic adaptations associated with exercise training. There is no way to achieve physical fitness and the desired adaptations other than through regular physical activity. Healthy persons and patients who have chronic diseases, in-

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ABBREVIATIONS USED IN TEXT

CHD = coronary heart disease
Vo_{2max} = oxygen consumption during maximal exercise

cluding coronary heart disease (CHD), can substantially increase their levels of fitness at all ages to attain maximal functional capabilities. Also, habitual exercise can minimize the secular functional impairment beginning after the second decade of life. Aerobic capacity declines with advancing age twice as rapidly in sedentary men as in physically active men.[9]

Components of Fitness

Because the nature of the training determines the specific adaptations that occur, an exercise program should address the major components of fitness: cardiorespiratory endurance, flexibility and strength.

Cardiorespiratory Endurance

Dynamic or endurance-type exercise, commonly termed aerobic exercise, increases aerobic capacity of exercising skeletal muscle in proportion to the muscle mass used and the intensity of exertion.[9] Oxygen consumption during maximal dynamic exercise (Vo_{2max}) determines aerobic functional capacity and is defined as the product of cardiac output and arteriovenous oxygen differences. Functional limits are determined primarily by the adaptive responses in the cardiovascular system leading to enhanced transport of oxygen, carbon dioxide and heat.

Cardiorespiratory endurance activities involve rhythmic activity of large muscle groups, such as those used in brisk walking, jogging or swimming. An exercise session conducted at 60% to 80% of the Vo_{2max}, corresponding to about 70% to 85% of the maximal heart rate for a 20- to 40-minute period, three to four times a week, will bring about the desired adaptations.[7,10,11] Less strenuous levels of exercise may be sustained for hours, but, though useful for weight reduction and psychological benefits, are usually insufficient to confer optimal levels of fitness on most persons. Whereas more exhaustive "workouts" may be essential for achieving peak athletic performance, such an approach may actually be detrimental for maintaining health and preventing disease because of the associated hazards. By raising the maximal capacity, the submaximal oxygen requirements of daily living should not lead to undue fatigue and dyspnea (Figure 1). Assuming symptoms develop when a person exceeds 75% or more of maximal capacity, a 40-year-old man with low fitness of 24 ml of oxygen per kg of body weight per minute will have symptoms at an 8-km (5-mile) per hour walk-jog requiring 30 ml O_2 per kg per minute until he increases his maximal capacity to 40 ml per kg per minute, at which point he becomes asymptomatic. Alternatively, he could reduce his speed to 5 to 6 km (3 to 4 miles) per hour rather than exercise to improve fitness so that the exertional level would not exceed the 75% threshold. Healthy 50-year-old men who train regularly can achieve a Vo_{2max} 20% to 30% higher than that of younger, sedentary men.[6] Increments in functional capacity may be especially important for older persons who may be starting from a very low level of fitness. Such improvements can result in almost complete self-care and independence.

Flexibility

Flexibility can best be obtained by stretching exercises that result in improved suppleness and neuromuscular coordination. These activities are valuable for overall fitness and

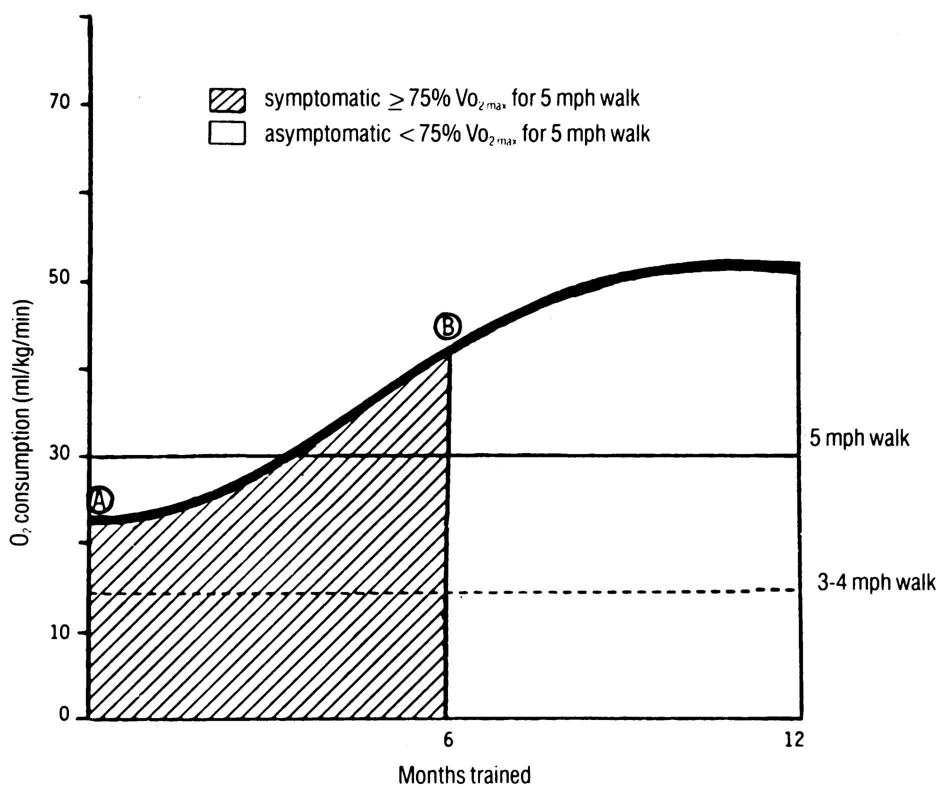

Figure 1.—Assuming symptoms such as undue fatigue and dyspnea develop when a man exceeds 75% or more of his maximal capacity, this figure shows that a 40-year-old man with low fitness of 24 ml of oxygen per kg of body weight per minute (point A) will have symptoms at a five-mile-per-hour (mph) walk-jog requiring 30 ml O_2 per kg a minute until he increases his maximal capacity to 40 ml per kg per minute (point B), at which point he becomes asymptomatic. Alternatively, he could reduce his speed to three to four miles per hour, rather than exercise so that the exertional level would not exceed the 75% threshold. Vo_{2max} = oxygen consumption during maximal dynamic exercise

muscle tone, thus indirectly contributing to feeling better and facilitating specific physical activities encountered in jobs or hobbies.

Strength

Strength training involves working muscle groups with progressive resistance overloading, which results in increased muscular strength. Strength is maintained by completing several sets of repetitive muscular contractions with work loads of 1 to 4.5 kg (2 to 10 lbs). Most of these contractions are a mixture of isotonic (a constant resistance moved through a range of motion of the involved joint, such as lifting a weight) and isometric (no change in angle of involved joint, with little or no change in length of contracting muscle, such as pushing palms of hands together) maneuvers. Isometric contractions may be less irritating to a painful joint or soft tissue injury, but tend to produce pronounced elevations of blood pressure, thus inordinately increasing the work load on the heart.[12]

Specific Metabolic Benefits

Maintenance of optimal body weight and composition and favorable changes in carbohydrate and lipid metabolism through regular exercise have been well documented.

Body Composition and Weight Control

The value of exercise for reducing weight is grossly underestimated, as most people associate weight loss with dieting rather than with exercise. Exercise conditioning has been shown to reduce body weight and adiposity by increasing energy expenditure, raising resting metabolic rates, accelerating mobilization of fat stores and increasing the likelihood of changes in eating patterns.[7,8,13]

Physically active persons, such as those running several miles per day, consume up to 15% more calories a day than their sedentary counterparts, yet remain significantly leaner. Weight gain or loss reflects small changes in food intake or exercise over a long period of time. Most recent data show little change in food intake with moderate exercise, and short-term suppression of appetite with more vigorous exercise. Furthermore, exercise counters the autoregulatory decrease of basal metabolic rate with reduced caloric intake by increasing the metabolic rate for several hours, accelerating caloric expenditure and facilitating weight loss.[13] This elevated metabolic rate becomes particularly important if the exercise occurs regularly and frequently over a long period of time.

It has become increasingly apparent that effective long-term weight control requires a balance of energy intake (food) and energy output (exercise). The calories required each day can be estimated by multiplying the weight of a person by the number of calories per kilogram (or pound) for usual activity levels (Table 1). A 68-kg (150-lb) woman engaging in moderate activity requires 2,100 calories per day (150 lbs × 14 calories per lb = 2,100 calories). To lose 1 lb per week, she must reduce 500 calories a day (3,500 calories per week), either by cutting back on intake, by increasing activity or by some combination of the two.

Other advantages obtained through exercise include the relatively greater loss of body fat compared with muscle and bone tissue. Fat is preferentially used for energy needs. After about 20 minutes of exercise, the body relies increasingly on fat for energy, mobilizing undesired fat deposits.[13] Thus, exercise results in more muscle and less fat even if overall weight is not altered.

TABLE 1.—*Calories Needed Daily for Each Pound of Weight*

Activity Level	*Calories per Pound* *Women*	*Men*
Very light (sitting mostly)	12	14
Light	13	15
Moderate (housework, gardening)	14	16
Strenuous	15	17
Very strenuous	16	18

Carbohydrates

Less insulin is secreted in the trained state. However, glucose tolerance changes little, or even improves. Physical training mediates this favorable adaptation through at least three separate mechanisms: increased sensitivity of insulin receptors; depletion of muscle and liver glycogen, creating available glucose storage space, and enhanced muscle cell permeability to glucose.[14] Although a single bout of exercise can greatly influence the insulin response to a carbohydrate load, the effects are not as great as the cumulative response to habitual vigorous exercise. Short-term muscular activities that produce these metabolic changes in carbohydrate metabolism do not persist with prolonged periods of inactivity.[14] This reduction in insulin may have an effect on the development of coronary heart disease. Apart from its role in carbohydrate metabolism, insulin has been postulated to be independently atherogenic.[15]

Lipids

Exercise training consistently raises levels of high-density-lipoprotein cholesterol. This lipid subfraction is inversely related to CHD and is important for predicting the development of CHD.[16] Other advantageous changes are a fall in low-density-lipoprotein cholesterol level, a rise in apolipoprotein A1 level and an increase in lipoprotein lipase content.[17] The effect of exercise on total serum cholesterol remains controversial. Exercise reduces triglyceride levels, presumably through the lipoprotein lipase changes, and, as with carbohydrate metabolism, the effects depend on both acute exercise activities and long-term training.[18] Seasonal variations in lipids, concomitant weight and dietary changes and lack of true control groups complicate interpretation of the studies of lipid modification with exercise. Another unresolved consideration is the question of the exercise threshold for lipid changes. In recent studies favorable lipoprotein changes were confined to those who had either been running for at least four years, or had been running 56 km (35 miles) or more per week,[19] those who averaged at least 20 km (12.4 miles) per week of running[20] or those who ran at least 13 km (8 miles) per week for one year.[21] The concept of an exercise threshold for lipid changes is consistent with observations that more vigorous forms of exercise are required for reducing the risk of coronary heart disease.

Psychological Benefits

From available research, mainly correlational studies, the conclusion is generally made that exercise is associated with an improved affect and self-concept, especially in persons who are moderately depressed or anxious.[22,23] Claims have also been made that exercise bolsters cognitive performance during or after stress.[24] Patients with mild to moderate depression often respond poorly to drugs and tend to do well with the mood-enhancing effects of an exercise program.[8,23] "Feeling better" has been almost universally claimed as an accepted benefit of exercise, while others report that exercise enables persons to handle stress better.[8,24] Also, some investigators have claimed that exercise can modify personality type and thus indirectly reduce susceptibility to stress-induced disorders.[25] However, the general belief that exercise results in improved health habits has never been subjected to rigorous scientific scrutiny.

Current emphasis on neurohumoral control of stress concerns the influence of submaximal exercise on circulating catecholamines and endorphins, and the anecdotal evidence of a "high" with exercise. Training tends to enhance parasympathetic activity and reduce the response of catecholamines to exercise. The perception of effort during exercise has also been directly related to circulating catecholamines.[26] Current work indicates that endogenous opiates stimulated by exercise dampen the release of catecholamines and consequently appear to be important modulators of neuroendocrine responses to stress.

Specific Disorders

Coronary Heart Disease

Despite the decline in cardiovascular mortality over the past decade, CHD remains the leading cause of death and disability in this country. For those reasons alone, any personal health maintenance program should be strongly oriented toward preventing CHD and its complications. Whether or not physical activity protects against CHD has been widely debated.[6,7,27] Population studies consistently showed significantly lower mortality rates in physically active workers compared with sedentary workers.[28] However, the design of such studies did not permit a clear distinction between protection by exercise, or selection of healthier persons by virtue of their continuing to be physically active at work. Taking possible selection factors into account, a study of longshoremen showed 49% less coronary events for those who expended 5 to 8 kcal per minute of energy at work, as compared with their less active co-workers.[29] Data from more recent incidence studies with less potential for bias have now accumulated (Table 2). Analysis of CHD morbidity and mortality among Harvard alumni observed for ten years or more, expending 2,000 or more kcal per week in exercise, showed a threshold above which there was a significant 50% reduction of myocardial infarctions and sudden death in men, as compared with those expending less per week.[30,31] Contemporary physical activities of the alumni outweighed any influence from earlier participation in sports and athletics. Others have found, however, that exercise responses in youth, presumably an index of fitness, significantly relates to risk factors for CHD at middle age.[32] In a prospective study of nearly 3,000 healthy men, but with at least two of the major risk factors, the adjusted relative risk for those below the median in physical work capacity was 6.6, suggesting that poor physical fitness, when accompanied by other major risk factors, enhanced susceptibility to CHD.[33] Several prospective studies have reported a definite relationship between low physical activity and cardiovascular disease and death, while controlling for other prognostic factors. The Framingham study showed an inverse correlation between physical activity and cardiovascular morbidity and mortality, but only one of modest magnitude and only in men.[34] In a study of British civil servants designed to determine the role of exercise in preventing CHD, physical activity significantly reduced both the incidence of fatal coronary disease and combined fatal and nonfatal incidence rates at all ages, especially at older ages.[35] In Finland low physical activity at work increased the likelihood of cardiovascular problems and at leisure time decreased the risk of death.[35a] Data from the Puerto Rican Heart Study convincingly showed physical inactivity being second only to hypertension as the major risk factor for CHD in that population.[36]

TABLE 2.—*Recent Findings From Major Studies on Exercise and Coronary Heart Disease*

Population and Observation Years	*Number*	*Comparison*	*Conclusion*
Harvard alumni 1962-1972, Paffenbarger et al[30,31]	16,934	Reported energy expenditure	Risk of first heart attack was inversely related to energy expenditure in adult life, independent of other influences on heart attack risk.
The Framingham Study, 1957-1971, Kannel & Sorlie[34]	4,220	Physical activity index	Cardiovascular mortality and morbidity were inversely related to physical activity only in men; this relationship was significant and independent of other major risk factors, but the effect was modest in comparison.
British civil servants, 1969-1978, Morris et al[35]	17,944	Leisure activity; questionnaire	The coronary heart disease rates of men who took vigorous exercise were lower for both fatal and nonfatal events; these findings were most striking in older men.
North Karelia and Kuopio (Finland), 1972-1978, Salonen et al[35a]	6,665	Reported physical activity at work and leisure	Low physical activity at work was associated with increased risk of acute myocardial infarction, cerebral stroke and death from any cause in men and women, controlling for age and other risk factors; leisure-time physical activity related only to a decreased risk of death.
Puerto Rico Heart Health Program, 1965-1973, Garcia-Palmieri et al[36]	8,793	Physical activity index	The physical activity index was inversely correlated to the known coronary risk factors; multivariate analyses indicated a significant, independent inverse relationship between exercise and the incidence of coronary heart disease; physical activity appeared to be a separate protective factor against heart attack.

TABLE 3.—*Secondary Prevention With Physical Training in Previous Randomized Controlled Trials*

Trials*	Number	Intervention	Follow-up (months)	Mortality (%)		Nonfatal MI (%)	
				Control	Exercise	Control	Exercise
Sweden (1968-1972)	315	Supervised exercise 3 ×/week	48	22.3	17.7	17.9	15.7
Finland (1969-1972)	298	Supervised exercise 2-3 ×/week	12	21.9	17.1	5.4	8.1
Finland (1969-1972)	380	Daily home exercise	29	14.0	10.0	11.2	18.1
Canada (1972-1978)	733	Partially supervised exercise 2-4 ×/week	48	7.3	9.5	9.3	10.3
United States (1974-1979)	651	Supervised exercise 3 ×/week	36	7.3	4.6	3.3	4.6

MI = myocardial infarction

*From Oberman[11] and May et al.[44]

In reports from these large-scale studies, the energy expenditure necessary for protection is estimated to be about 7.5 kcal per minute for exercise sessions. Such levels correspond to brisk walking, jogging at 8 km (5 miles) per hour, heavy gardening or bicycling at 16 km (10 miles) per hour. This effect of sustained physical activity may emanate in part from its influence on some of the risk factors that play major roles in the development of atherosclerosis. However, the impact of vigorous exercise appears to be independent of the major cardiovascular risk factors and somewhat less powerful.[37]

Habitual strenuous exercise may augment myocardial blood supply. In longitudinal studies, coronary flow patterns changed early after the onset of a training program, which indicates that regulatory adaptations are taking place.[12] Experimental evidence also exists for a training-induced increase in the size of the coronary vascular bed, even though multiple studies have failed to show an increase in collateralization in the absence of coronary lesions in humans.[11,12] Patients may better tolerate thrombotic stimuli after training. In hypercoagulable states, the fibrinolytic response to various stimuli may be enhanced in trained persons[8,11]; further work is required to clarify changes in platelet activity.[8] Also, a decreased propensity toward arrhythmias may be a product of long-term training.[38] In this experimental study daily exercise modified cardiac electrostability and autonomic control of heart rate in such a way as to decrease vulnerability to life-threatening dysrhythmias. Reduced sympathetic tone after conditioning may reduce serious dysrhythmias; however, hygienic intervention trials failed to suppress stress-induced ventricular ectopic rhythms in healthy men having frequent premature ventricular beats.[17]

Cardiac rehabilitation programs have demonstrated their influence on morbidity in the broad sense as evidenced by reducing symptoms through suitable training for angina patients, post-myocardial infarction patients or patients who have had coronary artery bypass grafting. Such patients can increase their maximal oxygen uptake by 20% or more, alleviating symptoms associated with exertion.[11] The improved functional capacity is due mainly to the adaptations in the peripheral muscles rather than directly in the myocardium or myocardial oxygen delivery.[12,37] The issue of whether myocardial oxygen consumption is enhanced remains controversial. Some studies show an increase with the angina threshold as measured by the rate-pressure product, presumably representing enhanced myocardial oxygen delivery. Others clearly show an increase in the rate-pressure product and infer increased oxygen delivery to the myocardium.[39] The fact that trained cardiac patients can maintain the same pace as many normal persons, however, may be due to physiologic changes other than in maximal capacity. Coyle and co-workers[40] showed that patients with coronary heart disease can maintain a high lactate threshold relative to maximal oxygen consumption, enabling them to exercise at near-maximal levels.

One explanation for the lack of myocardial adaptations has been the limited intensity and duration of most exercise programs. Ehsani and colleagues[41] have found an increased rate-pressure product in patients with coronary artery disease who are on an exercise program of four times per week to levels of 90% maximum over a period of six months to a year. They and others have postulated that the skeletal and autonomic nervous system adaptations occur within weeks but the myocardial changes, only with more intense exertional levels, occur over a period of months.[11]

Exercise training in patients with coronary heart disease appears to be more effective for secondary prevention in those with reasonable left ventricular function.[11] The left ventricular ejection fraction during exercise may be improved after exercise conditioning, probably by the lower heart rate at comparable work loads after training, though the favorable changes in loading conditions of the heart and an increase in myocardial contractility are alternative explanations.[42] Exercise intolerance in patients with congestive heart failure may be due to impaired nutritive flow to skeletal muscles with resultant muscular fatigue.[43]

Early studies of the effect of exercise on recurrent myocardial infarction and death in those with overt CHD were largely uncontrolled, but they clearly established the safety of a supervised training program. Over the past ten years at least five clinical trials on the role of exercise in reducing the reinfarction rate and mortality have been carried out.[11,44] This second generation of randomized, controlled trials disclosed no significant differences in the recurrence of nonfatal myocardial infarctions (Table 3). Yet, with the exception of the study from Canada, all of these investigations with follow-up extending from 12 to 48 months found reductions in mortality of from 21% to 37% in those exercising at an effective level.[11] May and associates[44] justified pooling these data on the basis of the similarity of the studies and because no single study had enough subjects to truly test the exercise hypothesis. Combined analyses from these studies indicate a 19% reduction in mortality ($P < .05$) and suggest that exercise influences the course of patients who have CHD.

Hypertension

Population studies show an inverse correlation between physical activity and blood pressure levels.[8,45] A consistent finding is the lowering of blood pressure at submaximal exercise as might be expected from raising the maximal capacity, so that any submaximal exertional level represents a smaller proportion of maximal capacity and a lessened physiologic stimulus for heart rate and blood pressure changes (Figure 1). However, the increase in blood pressure from rest to exercise is similar before and after training, suggesting no change in the regulation of blood pressure during submaximal exercise in patients with hypertension.[45]

In several studies endurance training has been more effective in lowering blood pressure at rest in patients with borderline or essential hypertension.[8,39] Generally, these studies have not included control groups, have not assessed simultaneous nutritional changes, have involved small numbers of middle-aged men for only short durations and have varied widely in methods of measuring blood pressure and types of training programs. Evidence at present appears inadequate to recommend exercise training as a form of nonpharmacologic treatment of hypertension.[45] In their 1984 report, the Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure suggested that exercise is useful for weight control and may be helpful in reducing blood pressure for those with uncomplicated hypertension.[46]

Diabetes Mellitus

Because its glucose-lowering effect is largely independent of insulin secretion, physical training can be useful in the management of cases of diabetes mellitus. However, moderate exercise alone appears to be ineffective as therapy for non-insulin-dependent diabetes mellitus and cannot subserve the other actions of insulin in juvenile-onset diabetes.[47] In addition, precautions are advised for exercising middle-aged persons who have diabetes mellitus because of the possibility of exercise-induced hypoglycemia and their vulnerability for coronary events.

Musculoskeletal Disorders

Localized osteoporosis develops several months after a limb is immobilized. With vigorous exercise, the mineral content of exercising limbs substantially increases, and it is postulated that exercise can be used to increase the strength of the skeleton and possibly retard the changes associated with inactivity and age. Calcium loss from the os calcis was minimized during weightlessness experienced during space flights by repeated applications of force to the bone through muscle contractions.[48] Regular exercise may also reduce the loss of calcium by postmenopausal women.[7] A cross-sectional survey of postmenopausal women showed that the level of habitual physical activity correlated with bone mass as measured by computed tomographic scanning. Yet, women with exercise-induced amenorrhea have decreased vertebral mineral content.[49] Bone mineral content has been increased in several studies of older adults in exercise programs. This supports the concept that older adults can reduce the loss of bone mineral content that occurs through unnecessary bed rest or immobilization.[7,8]

Studies on the effectiveness of both dynamic and isometric exercise toward improving the function of patients with rheumatoid arthritis have indicated some improvement without adverse effects.[50-52] Isometric exercises have been preferred because of the expected aggravation of joint pain and inflammation with dynamic exercise. Yet, patients with ankylosing spondylitis have reported diminished pain and joint stiffness after dynamic physical training.[48] Well-controlled studies are noticeably lacking, but available data indicate that under careful supervision, patients with arthritis may be encouraged to participate more fully in exertional activities. Muscle weakness, a common clinical problem in the presence of joint disease, might be alleviated without damaging joints further through the judicious use of exercise.

No body of experimental evidence exists showing the efficacy of exercise in preventing or treating low back pain. The premise is based on the knowledge that abdominal and costal muscles relieve the stress on the lumbar spine when lifting and carrying heavy objects.[48] When lifting correctly, the quadriceps effectively distribute weight. Thus, it reasonably follows that strengthening of the abdominal, costal and quadriceps muscles will relieve the load on the lumbar spine and contribute to the relief of low back pain.

Other

Several reports have now shown that progressive exercise is surprisingly effective in managing chronic obstructive lung disease.[7,8,53] Benefits ascribed to physical activity relate to reducing the fatiguability of the respiratory muscles, reversing the effects of inactivity and increasing psychological tolerance of breathlessness. Whereas patients feel better, it is uncertain whether this has any effect on the ultimate course of the disease process. Physical activity has surprisingly little effect on pulmonary function in normal persons.[12] Participation by such patients in training programs is reputedly poor, possibly because of the difficulty in carrying out exercise without supplemental oxygen or the lack of a specific event, as with patients who have had a coronary event, to provoke their interest.[53]

A regular walking exercise may prove of great benefit to patients who have intermittent claudication. Exercising to the point of claudication over a period of time can presumably lead to an increase in calf muscle blood flow during submaximal work.[8]

Physical activity may play a previously unrecognized role in preventing colon cancer.[54] Men with sedentary jobs had a colon cancer risk of 1.6 times that of men whose jobs required a high level of activity, with a stepwise increase in risk as activity level decreased. A proposed physiologic explanation for these observations is that physical activity stimulates colon peristalsis and decreases random, nonpropulsive segmentation activity, thus decreasing the contact time for possible carcinogens. On the other hand, it has been theoretically postulated that exercise training predisposes to the development of cancer.[6] Conceivably, large increases in Vo_{2max} associated with strenuous exercise training accelerate the aging process by increasing the rate of tissue damage by free radicals formed from the oxygen.

Hazards of Exercise

Not to be ignored are the possible risks from exercising. Foremost among the problems of physical activity are those related to the musculoskeletal system; strains to vulnerable areas have been well documented.[8,48] In endurance activities involving the dynamic use of large muscle groups, the muscles and structures to which they attach may become seriously stressed and injured.

Other possible complications of a training program include a variety of reported disorders. Extremes of temperature may predispose to heat exhaustion or heat stroke in unusually warm weather and nonenvironmentally controlled facilities. In cold weather frostbite of extremities and exposed parts of the body is possible. More unusual problems associated with vigorous exertion are hypersensitivity reactions.[55] In some people who have a strong family history of allergies, wheezing, asthmatic attacks or urticaria may develop during or after exercising. Although relatively rare, such episodes can be life-threatening. Little information is available on hormonal changes, especially among men, but lowered testosterone and prolactin levels parallel the changes noted in women runners.[56] Other reported complications include gross hematuria[57] and gastrointestinal bleeding.[58] Experimental evidence suggests that exercise during a viral infection may precipitate subclinical myocarditis.[48] Rhabdomyolysis has been reported as a complication of extremely strenuous exercise and may lead to renal failure.[8]

Much more serious complications arise from an increased susceptibility to cardiovascular events during exertion and immediately afterwards. There are no data to suggest that even strenuous training exercise can harm a "normal" heart, even though training adaptations mimic pathologic findings and may have long-term implications.[8] But in the presence of coronary atherosclerosis, a stressed myocardium may become ischemic and vulnerable to infarction or lethal arrhythmias. Persons dying suddenly of CHD during exertion usually have advanced atherosclerosis regardless of whether or not they were asymptomatic.[59] In a Finnish study, 4.5% of sudden cardiac deaths were related to strenuous or unusual physical activity, usually cardiac arrhythmias due to CHD.[17]

Overall, the acute cardiac risk of vigorous activity has been estimated in several studies and is generally thought to be low.[60] It has been estimated that the hourly CHD mortality increased seven times during jogging, although the absolute death rate of one death per 396,000 jogging hours mitigates against any undue danger.[61] The investigators felt that, due to the rarity of exercise deaths, routine exercise testing in healthy persons at low risk is not justified. Nevertheless, it is not unreasonable to assume that strenuous activity, especially in intemperate weather, can precipitate coronary events in vulnerable persons. Unfortunately, this propensity towards sudden death or infarction with physical activity cannot be predicted with an adequate degree of certainty.[48,60] Consequently, the recommended screening procedure before exercise training for asymptomatic middle-aged men is unresolved. The National Institutes of Health[10] recommend that a graduated exercise program can be initiated without seeing a physician if there is (1) no history of heart trouble, heart murmur, heart attack, hypertension, arthritis or diabetes, (2) no family history of premature coronary artery disease and (3) no exercise-related breathlessness, faintness, dizziness or pain or pressure of the chest, neck, shoulder or arm. The American College of Sports Medicine recommends an exercise test as part of medical screening before a strenuous exercise program for sedentary people 35 years of age or older and younger people at high risk.[4] Guidelines for implementing exercise programs for persons with or without cardiac history wishing to improve their fitness abound in the literature.[7,8,62]

Conclusion

The human body requires exercise to function satisfactorily. Persons at any age can substantially improve their fitness for work and play through appropriate exercise training. Considerable evidence exists for favorable changes in body composition, lipids and carbohydrate tolerance. Blood pressure levels, especially during submaximal efforts, probably can be reduced, but adequate clinical trials are needed. Extensive but nonconclusive data have documented psychological benefits accruing from physical activity. The often-stated belief for associated changes in health habits with exercise has little basis. A variety of other physiologic claims have been reported, not all of which may be corroborated, and some of which may actually be detrimental to one's health. Yet, exercise most likely reduces the incidence of CHD and appears to be a useful adjunct in managing patients who have coronary artery disease, diabetes mellitus, pulmonary disorders and musculoskeletal problems. The risks from long-term training must be taken into account but are not appreciable and are almost negligible in healthy persons.

Taken altogether, a distinct rationale exists for exercise training to maintain health. As with any therapeutic or preventive measure, the benefits and risks for a given objective must be carefully assessed. It is not surprising that exercise is viewed as being so important because of the anticipated changes in physical capacity, appearance and general health. Patients rely on physicians' recommendations, but many practitioners seem reluctant to incorporate exercise into their therapeutic armamentarium. Practicing physicians must take a more active role in prescribing conditioning regimens for those wishing to incorporate exercise into their personal health maintenance program.

REFERENCES

1. Prevention of Coronary Heart Disease—Report of a WHO Expert Committee, Tech Rep Series 678. Geneva, World Health Organization, 1982
2. American Heart Association Heartbook: A Guide to Prevention and Treatment of Cardiovascular Diseases. New York, EP Dutton, 1980
3. Health and Prevention Profile: United States, 1983, US Dept of Health & Human Services publication No. (PHS) 84-1232, Public Health Service, National Center for Health Statistics. Government Printing Office, 1984
4. American College of Sports Medicine: Guidelines for Graded Exercise Testing and Exercise Prescription, 2nd Ed. Philadelphia, Lea & Febiger, 1980
5. Symposium: Exercise, health, and medicine. Lancet 1983; 1:1171
6. Holloszy JO: Exercise, health, and aging: A need for more information. Med Sci Sports Exerc 1983; 15:1-5
7. Haskell WL, Superko R: Designing an exercise plan for optimal health. Fam Community Health 1984; 7:72-88
8. Fletcher GF: Exercise in the Practice of Medicine. Mt Kisco, NY, Futura Publ, 1982
9. Bruce RA: Exercise, functional aerobic capacity, and aging—Another viewpoint. Med Sci Sports Exerc 1984; 16:8-13
10. Exercise and Your Heart, US DHHS publication No. (NIH) 83-1677. Government Printing Office, May 1981
11. Oberman A: Rehabilitation of patients with coronary artery disease, *In* Braunwald E (Ed): Heart Disease: A Textbook of Cardiovascular Medicine, 2nd Ed. Philadelphia, WB Saunders, 1983, pp 1384-1398
12. Blomquist CG, Saltin B: Cardiovascular adaptations to physical training. Annu Rev Physiol 1983; 45:169-189
13. American College of Sports Medicine: Position Statement On: Proper and Improper Weight Loss Programs. Indianapolis, American College of Sports Medicine, 1983
14. Heath GW, Gavin JR III, Hinderliter JM, et al: Effects of exercise and lack of exercise on glucose tolerance and insulin sensitivity. J Appl Physiol 1983; 55:512-517
15. Stout RW: Blood glucose and atherosclerosis. Arteriosclerosis 1981; 1:227-234
16. Castelli WP, Abbott RD, McMamara PM: Summary estimates of cholesterol used to predict coronary heart disease. Circulation 1983; 67:730-734
17. Leon AS, Blackburn H: Physical inactivity, *In* Kaplan NM, Stamler J (Eds): Prevention of Coronary Heart Disease. Philadelphia, WB Saunders, 1983
18. Consensus Development Panel: Treatment of hypertriglyceridemia—Special report of the NIH Consensus Development Conference, Bethesda, Maryland. JAMA 1984; 251:1196-1200

19. Christie RJ, Bloore HG, Logan RL: High-density lipoprotein (HDL) cholesterol in middle-aged joggers. NZ Med J 1980; 91:39-40

20. Kavanagh T, Shephard RJ, Lindley LJ, et al: Influence of exercise and life-style variables upon high density lipoprotein cholesterol after myocardial infarction. Arteriosclerosis 1983; 3:249-259

21. Wood PD, Haskell WL, Blair SN, et al: Increased exercise levels and plasma lipoprotein concentrations—A one-year, randomized controlled study in sedentary middle-aged men. Metabolism 1983; 32:31-39

22. Morgan WP: Psychological effects of exercise. Behav Med Update 1982; 4:25-30

23. Sachs ML, Buffone Gary W (Eds): Running as Therapy: An Integrated Approach. Lincoln, University of Nebraska Press, 1984

24. Folkins CH, Sime WE: Physical fitness training and mental health. Am Psychol 1981; 36:373-389

25. Blumenthal JA, Williams RS, Williams RB, et al: Effects of exercise on the type A (coronary prone) behavior pattern. Psychosom Med 1980; 42:289-296

26. Grossman A: Endorphins and exercise. Clin Cardiol 1984; 7:255-260

27. Oberman A: The role of exercise in preventing coronary heart disease, *In* Rapaport E (Ed): Current Controversies in Cardiovascular Disease. Philadelphia, WB Saunders, 1980

28. Froelicher VF, Oberman A: Analysis of epidemiologic studies of physical inactivity as risk factor for coronary artery disease. Prog Cardiovasc Dis 1972; 15:41-65

29. Paffenbarger RS Jr, Hale WE, Brand RJ, et al: Work-energy level, personal characteristics, and fatal heart attack: A birth-cohort effect. Am J Epidemiol 1977; 105:200-213

30. Paffenbarger RS Jr, Wing AL, Hyde RT: Physical activity as an index of heart attack risk in college alumni. Am J Epidemiol 1978; 108:161-175

31. Paffenbarger RS, Hyde RT, Wing AL, et al: A natural history of athleticism and cardiovascular health. JAMA 1984; 252:491-495

32. Gillum RF, Taylor HL, Anderson J, et al: Longitudinal study (32 years) of exercise tolerance, breathing response, blood pressure and blood lipids in young men. Arteriosclerosis 1981; 1:455-462

33. Peters RK, Cady LD, Bischoff DP, et al: Physical fitness and subsequent myocardial infarction in healthy workers. JAMA 1983: 249:3052-3056

34. Kannel WB, Sorlie P: Some health benefits of physical activity—The Framingham study. Arch Intern Med 1979; 139:857-861

35. Morris JN, Everitt MG, Pollard R, et al: Vigorous exercise in leisure-time: Protection against coronary heart disease. Lancet 1980; 2:1207-1210

35a. Salonen JT, Puska P, Tuomilehto J: Physical activity and risk of myocardial infarction, cerebral stroke and death—A longitudinal study in Eastern Finland. Am J Epidemiol 1982; 115:526-537

36. Garcia-Palmieri MR, Costas R Jr, Cruz-Vidal M, et al: Increased physical activity: A protective factor against heart attacks in Puerto Rico. Am J Cardiol 1982; 50:749-755

37. Kannel WB, Doyle JT, Ostfeld AM, et al: Optimal resources for primary prevention of atherosclerotic diseases—Special Report to the Inter-Society Commission for Heart Disease Resources. Circulation 1984; 70:157A-205A

38. Billman GE, Schwartz PJ, Stone HL: The effects of daily exercise on susceptibility to sudden cardiac death. Circulation 1984; 69:1181-1189

39. Rigotti NA, Thomas GS, Leaf A: Exercise and coronary disease. Annu Rev Med 1983; 34:391-412

40. Coyle EF, Martin WH, Ehsani AA, et al: Blood lactate threshold in some well trained ischemic heart disease patients. J Appl Physiol 1983; 54:18-23

41. Ehsani AA, Martin WH III, Heath GW, et al: Cardiac effects of prolonged and intense exercise training in patients with coronary artery disease. Am J Cardiol 1982; 50:246-254

42. Williams RS, McKinnis RA, Cobb FR, et al: Effects of physical conditioning on left ventricular ejection fraction in patients with coronary artery disease. Circulation 1984; 70:69-75

43. Wilson JR, Martin JL, Schwartz D, et al: Exercise intolerance in patients with chronic heart failure: Role of impaired nutritive flow to skeletal muscle. Circulation 1984; 69:1079-1087

44. May GS, Eberlein KA, Furberg CD, et al: Secondary prevention after myocardial infarction: A review of long-term trials. Prog Cardiovasc Dis 1982; 24:331-352

45. Seals DR, Hagberg JM: The effect of exercise training on human hypertension: A review. Med Sci Sports Exerc 1984; 16:207-215

46. The 1984 Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. Arch Intern Med 1984; 144:1045-1057

47. Koivisto VA, Felig P: Exercise in diabetes: Clinical implications. Cardiovasc Rev Rep 1984; 5:399-404

48. Young A: Exercise in the prevention of disability, *In* Gray M, Fowler G (Eds): Preventive Medicine in General Practice. Oxford, England, Oxford University Press, 1983

49. Drinkwater BL, Nilson K, Chesnut CH III, et al: Bone mineral content of amenorrheic and eumenorrheic athletes. N Engl J Med 1984; 311:277-281

50. Nordemar R, Ekblom B, Zachrisson L, et al: Physical training in rheumatoid arthritis: A controlled long-term study—I. Scand J Rheumatol 1981; 10:17-23

51. Nordemar R: Physical training in rheumatoid arthritis: A controlled long-term study—II. Functional capacity and general attitudes. Scand J Rheumatol 1981; 10:25-30

52. Ekblom B, Lovgren O, Alderin M, et al: Effect of short-term physical training on patients with rheumatoid arthritis, a six-month follow-up study. Scand J Rheumatol 1975; 4:87-91

53. Shephard RJ: Endurance Fitness, 2nd Ed. Toronto, University of Toronto Press, 1977

54. Garrabrant DH, Peters JM, Mack TM, et al: Job activity and colon cancer risk. Am J Epidemiol 1984; 119:1005-1014

55. Sheffer AL, Austen KF: Exercise-induced anaphylaxis. J Allergy Clin Immunol 1980; 66:106-111

56. Wheeler GD, Wall SR, Belcastro AN, et al: Reduced serum testosterone and prolactin levels in male distance runners. JAMA 1984; 252:514-516

57. Daniels J, Fitts R, Sheehan G: Conditions for Distance Running. New York, John Wiley & Sons, 1978

58. Stewart JG, Ahlquist DA, McGill DB, et al: Gastrointestinal blood loss and anemia in runners. Ann Intern Med 1984; 100:843-845

59. Perper JA, Kuller LH, Cooper M: Arteriosclerosis of coronary arteries in sudden unexpected deaths. Circulation 1975; (Suppl III)51,52:27-33

60. Eichner ER: Exercise and heart disease: Epidemiology of the exercise hypothesis. Am J Med 1983; 74:1008-1023

61. Thompson PD, Funk EJ, Carleton RA, et al: Incidence of death during jogging in Rhode Island from 1975 through 1980. JAMA 1982; 247:2535-2538

62. Oberman A: Cardiac rehabilitation, key references. Circulation 1980; 62:909