Practice Guidelines

AHA Releases Statement on Exercise and Heart Failure

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Although discomfort during exercise is a principal reason that patients with heart failure seek medical care, exercise training is safe and can benefit these patients, according to the American Heart Association, whose report "Exercise and Heart Failure" is available in the March 4 issue of Circulation. Circulation is online at www.circ.ahajournals.org.

Exercise intolerance, the reduced ability to perform activities that involve dynamic movement of large skeletal muscles because of dyspnea or fatigue, may be one of the first symptoms experienced by patients with heart failure. A correlation between resting ventricular function and exercise capacity is expected, but data indicate that the relationship is weak.

Factors Affecting Exercise Tolerance

CIRCUITOUS

In healthy persons, the increase in cardiac output during maximal upright exercise typically is four- to sixfold, which is accomplished by a two- to fourfold increase in heart rate and a 20 to 50 percent increase in stroke volume. Although cardiovascular disease limits the increase of cardiac output during exercise, this response also is affected by age, gender, and conditioning status.

The reduced aerobic capacity that is common in patients who have heart failure is attributable to inadequate blood flow to skeletal muscles secondary to impaired cardiac output. Patients may have less than 50 percent of the maximal cardiac output of a healthy person at peak exercise. Stroke volume rises only modestly, to peak of 50 to 65 mL compared with at least 100 mL in healthy persons. The inability to increase cardiac output is related mainly to the minimal increase in stroke volume and a lower maximal heart rate achieved at a lower workload. The primary means to augment cardiac output in patients with heart failure is by cardioacceleration.

PERIPHERAL

Blood Flow Abnormalities. In patients with heart failure, blood flow to muscles does not increase normally during exercise because of reduced cardiac output and impaired peripheral vasodilatory capacity. This abnormality in vasodilatory capacity has been attributed to excessive sympathetic stimulation, which causes vasoconstriction, activation of the plasma renin-angiotensin system, and higher-than-normal levels of endothelin. Another mechanism may be vascular stiffness secondary to increased vascular sodium content.

Endothelial Function. Vasodilating and vasoconstricting factors are released from the vascular endothelium in response to various chemical, pharmacologic, mechanical, and exercise stimuli. The endothelium is now recognized as having a pivotal role in coordinating tissue perfusion in patients with heart failure. The release of nitric oxide, an important mediator of flow-dependent vasodilation, is stimulated by exercise in healthy persons but seems to be attenuated in patients with heart failure. The impairment in endothelial-dependent vasodilation correlates with the degree of exercise intolerance and severity of New York Heart Association class.

Muscle. Anaerobic metabolism that occurs early during exercise in patients with heart failure is likely an important cause of exercise intolerance. Abnormalities in skeletal muscle metabolism occur in patients with heart failure, and changes in muscle may contribute to abnormal oxygen extraction or substrate delivery and use.

Distribution of Cardiac Output. Exercise tolerance depends on the capacity of the pulmonary system to deliver oxygen to the working muscles and the capacity of the vasculature to redistribute cardiac output to the muscle during exercise. In healthy persons, as much as 85 percent of the cardiac output is redistributed to the muscle at high levels of exercise. Some evidence suggests that in patients with heart failure, muscle blood flow is reduced at the same rate as the reduction in cardiac output. However, several studies have found that the reduction in blood flow to the muscle during exercise occurs to a degree out of proportion to the reduction in cardiac output. Vascular resistance in the muscle fails to decrease normally during exercise in patients with heart failure, and flow to the nonexercising tissues may be maintained preferentially at the expense of hypoperfusion in the exercising muscle.

Ergoreflex Activation. The presence of a specific signal from the exercising muscle may be abnormally enhanced in patients with heart failure. These signals contribute to the abnormal hemodynamic, autonomic, and ventilatory responses to exercise that characterize heart failure. Afferent fibers in the muscle (i.e., ergoreceptors) are sensitive to metabolic changes related to muscular work. Ergoreceptors, which mediate circulatory adaptations in the early...
stages of exercise, are stimulated by metabolic acidosis and are partially responsible for sympathetic vasoconstriction and an increase in heart rate. The result of this enhanced ergoreflex response is hyperventilation and heightened sympathetic outflow, causing an increase in peripheral resistance and a decrease in muscle perfusion. Activation of these reflexes seems to be attenuated by exercise training.

**Exercise Training in Patients with Heart Failure**

**Benefits**

**Exercise Capacity.** In patients with heart failure, exercise training improves tolerance as assessed by peak \( \text{Vo}_2 \) and exercise duration. Most improvement occurs in the first three weeks but can continue for up to six months if the patient complies with the training program. Indexes of submaximal exercise, such as the six-minute walk or the ventilatory threshold, also improve. Changes in peak \( \text{Vo}_2 \) have been greater in patients with nonischemic cardiomyopathy than in patients with ischemic cardiomyopathy.

**Catecholamines.** Increased plasma catecholamines have been associated with a poor prognosis in patients with heart failure, and investigators have measured changes in catecholamines in response to exercise in the hopes that they would decrease. However, the results of these studies have been variable, possibly in relation to the severity of the disease, etiology and duration of the heart failure syndrome, intensity and duration of exercise, and the presence of sympathetic activity.

**Ventilatory Responses.** Symptoms in patients with heart failure are related to an excessive increase in blood lactate levels during low exercise levels, reduction in \( \text{Vo}_2 \) at peak exercise, and disproportionate increases in ventilation at submaximal and peak workloads. The increased ventilatory requirement from the hyperventilatory response to exercise and increase in pulmonary dead space leads to rapid and shallow breathing during exercise. Because skeletal muscles become deconditioned, the same state of deconditioning in respiratory muscles can be expected. Exercise training potentially can improve these abnormalities.

**Endothelial Function.** Invasive and noninvasive studies have shown a significant correlation between the endothelial function of the coronary and forearm arteries in patients with coronary artery disease. The response of brachial artery diameter to reactive hyperemia or acetylcholine may reflect the endothelium-dependent relaxation of peripheral arteries of similar size in other locations. Therefore, exercise training may improve flow-dependent relaxation of peripheral arteries, and this effect may translate to increased blood flow to skeletal muscles.

**Myocardial Adaptations.** Although improvement in exercise capacity after exercise training seems to be related mainly to peripheral adaptations, a favorable effect on myocardial adaptations and the outcome of exercise-induced coronary vessel adaptations has been suggested.

**Hypoxic and Ischemic Injury.** Endurance-type exercise training has improved indexes of diastolic function. Exercise training may benefit clinically significant, symptomatic diastolic dysfunction, but this benefit is unproved. Until definitive clinical trials are performed, it is reasonable to recommend endurance-type exercise training with careful initial supervision.

**Valvular Disease.** Exercise training has no therapeutic role in patients with heart failure in the setting of severe stenotic or regurgitant valvular heart disease. No data support the safety and efficacy of exercise training in patients with heart failure and mild to moderate stenotic or regurgitant valvular disease.

**Risks**

Although many factors affect the risk of exercise, the most important are age, presence of heart disease, and intensity of the exercise. Sudden cardiac death during exercise is rare in persons who are apparently healthy. Persons who have heart disease are at greater risk of sudden cardiac arrest during vigorous exercise than are healthy persons. The incidence of major cardiovascular complications during outpatient cardiac exercise programs has been estimated to be one in 60,000 participant-hours. The type and intensity of exercise and the use of monitoring affect incidence of sudden cardiac arrest.

Myocardial infarction (MI) is more likely to occur than sudden cardiac death. Exercise is a potent trigger; approximately 4 to 20 percent of MIs occur during or soon after exertion. The adjusted relative risk is greater in persons who do not exercise regularly. The least active patients are at greatest risk of MI during exercise, and leisure activity and cardiorespiratory fitness have a strong inverse relationship with the risk for MI during exercise.

**Recommendations**

The Committee on Exercise, Rehabilitation, and Prevention of the American Heart Association Council on Clinical Cardiology concludes that exercise training in patients with heart failure seems to be safe and beneficial in improving exercise capacity, as measured by peak \( \text{Vo}_2 \), peak workload, exercise duration, and parameters of submaximal exercise performance. Benefits have been reported in muscle structure and physiologic responses to exercise, such as improvements in endothelial function, catecholamine spillover, and oxygen extraction in the periphery.