Cardiac Rehabilitation Following Percutaneous Revascularization, Heart Transplant, Heart Valve Surgery, and for Chronic Heart Failure*

Kerry J. Stewart, EdD; Dalynn Badenhop, PhD; Peter H. Brubaker, PhD; Steven J. Keteyian, PhD; and Marjorie King, MD

This review discusses the scientific and clinical evidence for cardiac rehabilitation in patients who have undergone percutaneous revascularization, heart transplant, and heart valve surgery, and in patients with chronic heart failure. Across these diagnoses, regardless of age, there is considerable benefit of cardiac rehabilitation and supervised exercise training for increasing functional capacity, favorably modifying disease-related risk factors, decreasing symptoms, detecting signs and symptoms of disease before they become serious complications, and improving quality of life. The available evidence for this component of cardiovascular disease management, albeit not perfect, still warrants its more widespread application. (CHEST 2003; 123:2104–2111)

Key words: cardiac rehabilitation; chronic heart failure; exercise training; heart transplant; heart valve surgery; percutaneous revascularization; secondary prevention

Abbreviations: LV = left ventricular; NYHA = New York Heart Association; $\mathrm{VO}_{2}$ = oxygen uptake

The benefits of cardiac rehabilitation for patients who have experienced a myocardial infarction, who have undergone coronary artery bypass surgery, or who have stable angina have been known for many years. In 1995, the Agency for Health Care Policy and Research Clinical Practice Guidelines also recommended cardiac rehabilitation for patients with chronic heart failure, and for those who have undergone heart transplantation and heart valve surgery. Unfortunately, it is estimated that only 10 to 40% of eligible patients participate in cardiac rehabilitation programs. These rates of participation are even lower for older patients, a group with the highest prevalence of cardiovascular disease. Although Medicare provides payments for cardiac rehabilitation for the diagnoses of myocardial infarction, coronary artery bypass surgery, and stable angina, there is no such payment available for patients who have undergone percutaneous revascularization, heart transplantation, and heart valve surgery, or for patients with heart failure. Insurance coverage by other third-party payers varies considerably throughout the United States.

WHAT IS CARDIAC REHABILITATION?

In 2000, the American Heart Association and the American Association of Cardiovascular and Pulmonary Rehabilitation recommended that cardiac rehabilitation programs provide several important core components consisting of baseline patient assessment, nutritional counseling, risk factor management (ie, lipid levels, hypertension, weight, diabetes, and smoking), psychosocial management, physical activity counseling, and exercise training. The American Heart Association also recommends these cardiac rehabilitation components for the elderly. Although secondary prevention therapies such as pharmacologic management of atherosclerosis risk factors and depression are provided by clinicians in their offices, cardiac rehabilitation is often the most advantageous setting for exercise training, patient education, behavioral counseling, and psychosocial support.
THE SCOPE OF THIS REVIEW: TYPES OF STUDIES

Since the publication of the 1995 clinical practice guidelines, there has been additional evidence demonstrating the efficacy of cardiac rehabilitation. The present review focuses on cardiac rehabilitation for patients with heart failure and for those who have undergone percutaneous revascularization, heart valve surgery, or heart transplantation, and, where data exist, for older patients with these diagnoses. A review of all of the components of secondary prevention (such as smoking cessation and pharmacotherapy) is beyond the scope of this review. To some extent, the scope is limited by the nature of the studies on cardiac rehabilitation for these diagnoses, which used supervised exercise training as the primary treatment modality. Although not fully evaluated, a comprehensive approach to cardiac rehabilitation would be more than just exercise training, and presumably would produce greater improvements in health and functional status for patients than is evident in the literature.

PERCUTANEOUS REVASCULARIZATION

Percutaneous interventions are effective for interrupting the process of acute coronary stenosis. Although it is fortunate that myocardial tissue damage can be avoided or minimized if the patient is treated in a timely manner, the need to treat the underlying disease that precipitated the stenosis is not changed after a revascularization procedure. Despite the restoration of coronary blood flow and the lack of myocardial damage, some patients are anxious about resuming physical activity following percutaneous revascularization and need the supportive environment of supervised cardiac rehabilitation to enhance their confidence to undertake physical activity and other favorable lifestyle changes. Supervised cardiac rehabilitation also promotes the early identification of new signs and symptoms indicating possible restenosis, leading to prompt medical evaluation treatment.

Despite the expanded use of percutaneous revascularization, there are few controlled studies of cardiac rehabilitation after these procedures. In one study, 93 patients who had been treated with percutaneous transluminal coronary angioplasty were randomly assigned to receive a behaviorally oriented intervention or a control group. After 12 months, the intervention patients, compared with the control subjects, improved significantly on self-rated measures of smoking, exercise, and diet habits. Patients also lost weight, improved their exercise capacity, and experienced less chest pain during exertion. Although the mechanisms for decreased mortality with exercise have not been fully explained, exercise training improves the lipid profile, reduces BP, lowers the fasting glucose level, and reduces body fat and increases lean body mass. Many of these risk factors exist in patients who have had percutaneous revascularization procedures. Studies in elderly patients have demonstrated the favorable effects of cardiac rehabilitation on plasma lipids, obesity, peak oxygen uptake ($V_O_2$), depression, and quality of life. The benefits in older patients (mean age, 70 years) are similar to those in younger patients (mean age, 54 years).

Thrombosis and endothelial dysfunction contribute to the occurrence of acute coronary syndromes and restenosis after patients undergo angioplasty or stent placement. Exercise training reduces the levels of fibrinogen and plasminogen activators and modulates platelet activation after short-term exercise, thereby theoretically reducing the risk of restenosis. This mechanism may be of particular value to patients who have undergone a revascularization procedure. Factors contributing to endothelial dysfunction include advanced age, diabetes, smoking, hypertension, and coronary artery disease. Exercise training has been shown to improve impaired endothelium-dependent vasodilation in patients with diabetes and hypertension. Exercise-induced improvements in endothelial function also may enhance myocardial blood flow reserve. Other interventions offered in cardiac rehabilitation that improve endothelial function include BP and cholesterol reduction, and smoking cessation. Medications that are commonly prescribed as part of a secondary prevention treatment to improve endothelial responses are estrogen and converting enzyme inhibitors and statins. Autonomic dysfunction increases the risk of sudden death. Exercise training increases parasympathetic tone in patients with congestive heart failure and after myocardial infarction, and may be cardioprotective. It is unknown whether this benefit of cardiac rehabilitation also extends to patients after percutaneous revascularization.

Cardiac rehabilitation, through some of the mechanisms elucidated, is an important adjunct to a physician’s range of interventions for patients who have undergone percutaneous revascularization. The risk factor management is no less critical for these patients than for those with other manifestations of atherosclerosis, even in the absence of myocardial damage, and may lead to a slowing of coronary disease progression.

HEART TRANSPLANTATION

Heart transplant patients experience persistent heart failure, diminished aerobic capacity, muscle
atrophy, side effects of immunosuppressive medications (ie, muscle and bone loss, and fat gain), infections, rejection, and premature coronary atherosclerosis. Functional capacity during exercise testing is reduced by 40 to 50% in heart transplant patients compared to age-matched healthy control subjects, and heart rate, BP, and cardiac output responses also are impaired.24–28 The ventilatory response to exercise is also excessive and inefficient.29

Exercise training studies have demonstrated the potential for reversing or diminishing physiologic abnormalities in heart transplant patients.24–26,28,30 Most studies are modeled after cardiac rehabilitation, in which exercise is conducted three to four times per week for 8 to 12 weeks at moderate intensity. Improvements in aerobic capacity range between 20% and 50%. The mechanisms underlying these increases are improved peripheral metabolic adaptations, leading to increased oxygen extraction, and hemodynamic changes, including an increase in heart rate and cardiac output.25,26,30 Ventilatory efficiency also improves at any given level of effort.25,26

Resistance training has been utilized in heart transplant patients for increasing lean muscle mass and bone density, thereby improving peripheral muscle capacity and minimizing postural deficiencies.25,31 These changes are particularly important because of the muscle and bone wasting consequences of heart failure and the medications used after heart transplantation. In a 6-month study31 in which heart transplant patients performed resistance training, bone mineral density was restored toward pretransplant levels, whereas that of control subjects decreased by 6%. In another 6-month trial,32 fat-free mass was restored to levels greater than before heart transplantation and dramatically increased muscle strength. Thus, resistance training contributes to the prevention and reversal of glucocorticoid-induced bone and muscle loss.

Although cardiac rehabilitation improves exercise capacity, patients may still experience the consequences of their transplants and treatments for it. One study30 randomly assigned heart transplant patients to a supervised cardiac rehabilitation or home exercise for 6 months. Each program included aerobic and resistance exercises. The supervised group increased peak VO2 by 49%, compared to 18% for the home group, and achieved a greater increase in peak exercise workload and a reduction in ventilatory responses. Nevertheless, the mean dose of prednisone, the number of patients receiving antihypertensive medications, the average number of rejection or infection episodes, and the level of weight gain did not differ between groups.

The published studies of cardiac rehabilitation for cardiac transplantation do not address allograft arteriopathy, the slowly progressive, diffuse, atherosclerosis that is noted after heart transplantation. This process of chronic rejection is the most common cause of late cardiac graft failure and patient death.33 Although cardiac rehabilitation and secondary prevention interventions would theoretically delay or prevent coronary artery disease progression in the transplanted heart, this issue has not been studied.

Because of improvements in surgical procedures, organ preservation, and immunosuppressive drug therapy, most patients survive the short-term recovery phase of the operation and can hope to achieve improved functional status. Although the studies reviewed are small, there is sufficient evidence that cardiac rehabilitation improves physiologic hemodynamic responses and helps to preserve or reverse bone and muscle loss (Table 1). Dealing with the continued medical consequences of cardiac transplantation is challenging, and the multidisciplinary nature of cardiac rehabilitation, including exercise, education, nutrition, and behavioral interventions, is ideally suited to these patients. One study24 reported that heart transplantation in selected patients who were ≥70 years of age could be performed with similar morbidity, mortality, and intermediate-term survival as found in younger persons. Although the efficacy of cardiac rehabilitation for elderly heart transplant patients has not been studied, it would be expected that the same benefits as demonstrated in younger persons would result in these patients.

### CHRONIC HEART FAILURE

Patients with heart failure often experience fatigue and dyspnea with exertion. Although the primary pathology of heart failure results from abnormalities in cardiovascular function, abnormalities in peripheral blood flow, skeletal muscle morphology, metabolism, strength, and endurance all contribute to the

<table>
<thead>
<tr>
<th>Study</th>
<th>Benefits</th>
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<tbody>
<tr>
<td>Niset et al24</td>
<td>Increases oxygen extraction by muscle, which decreases the need for cardiac output at a given level of muscular work</td>
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<tr>
<td>Kavanagh et al25</td>
<td>Improves ventilatory efficiency</td>
</tr>
<tr>
<td>Kavanagh et al25</td>
<td>Restores bone mineral density, which is lost because of immunosuppressive medications</td>
</tr>
<tr>
<td>Kavanagh et al25</td>
<td>Preserves or reverses skeletal muscle atrophy and increases muscle strength</td>
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Table 1—Benefits of Cardiac Rehabilitation Following Heart Transplant
Several trials have shown that cardiac rehabilitation improves disease-related symptoms, quality of life, and clinical outcomes. Overall, prescribed exercise attenuates the fatigue and dyspnea that limit exercise intolerance. The improvements ranged from 15 to 30% in peak VO₂, which is greater than or equal to the gains in exercise capacity observed in many clinical drug trials.

Several mechanisms contribute to improved functional capacity in patients with heart failure who participate in cardiac rehabilitation (Table 2). Central hemodynamic mechanisms include increases in peak cardiac output, heart rate, and stroke volume. Peripheral mechanisms include improved endothelial vasodilator function, increased cellular oxidative enzyme activity, a greater oxygen extraction from the blood, and an improved neurohumoral axis. These peripheral adaptations result in an increased oxygen delivery or utilization in the metabolically more active skeletal muscle, thus delaying reliance on anaerobic metabolism. Exercise training has beneficial effects on skeletal muscle directly by improving function, histologic characteristics, and biochemical characteristics, and indirectly by reducing the activation of the muscle neural afferents that are known as ergoreceptors. A 10-week randomized, controlled trial demonstrated the benefits and safety of resistance training in frail, older women with chronic heart failure. The mean age of the resistance trained group was 77 years, the mean left ventricular (LV) ejection fraction was 36%, mean New York Heart Association (NYHA) class was 2.2, and the mean duration of chronic heart failure was 29 months. The gains in functional performance following resistance training paralleled the improvement in muscle metabolism and function. Resistance training did not alter resting cardiac function, suggesting that peripheral adaptations mediated increased exercise performance. Although maximal exercise tolerance is an important outcome in patients with heart failure, equally important is the ability to perform the activities of daily living with fewer symptoms. Such observations are consistent with trials showing improvements in submaximal capacity as measured by ventilatory threshold or time at a fixed submaximal workload (Table 2).

Exercise training in patients with heart failure is associated with improvements in shortness of breath, the ability to perform activities of daily living, anxiety, depression, and general well-being. The magnitude of improvement in quality-of-life parameters

<table>
<thead>
<tr>
<th>Study</th>
<th>Patients, No.</th>
<th>Key Outcomes</th>
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</thead>
<tbody>
<tr>
<td>Jette et al&lt;sup&gt;33&lt;/sup&gt;</td>
<td>15</td>
<td>25% increase in peak VO₂ in T, 5% decrease in C; no change in LV dimensions</td>
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<tr>
<td>Giannuzzi et al&lt;sup&gt;34&lt;/sup&gt;</td>
<td>31</td>
<td>Increase in work capacity in T, slight improvements in LV ejection fraction; no change in LV ejection fraction, and slight increase in LV dimensions in C</td>
</tr>
<tr>
<td>Hambrecht et al&lt;sup&gt;35&lt;/sup&gt;</td>
<td>22</td>
<td>33% increase in peak VO₂ in T, no change in C; 30% increase in skeletal muscle mitochondria and oxidative enzymes; and decrease rest and exercise norepinephrine</td>
</tr>
<tr>
<td>Kiilavuori et al&lt;sup&gt;36&lt;/sup&gt;</td>
<td>20</td>
<td>15% increase in peak VO₂ in T, 1% increase in C; improved parasympathetic activity (22–25%) in T</td>
</tr>
<tr>
<td>Belardinelli et al&lt;sup&gt;37&lt;/sup&gt;</td>
<td>43</td>
<td>15% increase in peak VO₂ in T, 6% decrease in C</td>
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<tr>
<td>Keteyian et al&lt;sup&gt;38&lt;/sup&gt;</td>
<td>29</td>
<td>14% increase in peak VO₂ in T, 5% increase in C; increase in peak heart rate of 10 beats/min in T; decrease in resting and exercise plasma norepinephrine level</td>
</tr>
<tr>
<td>Kiilavouri et al&lt;sup&gt;39&lt;/sup&gt;</td>
<td>27</td>
<td>12% increase in peak VO₂ in T, no change in C; 16% reduction in minute ventilation during submaximal exercise after training</td>
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<tr>
<td>Dubach et al&lt;sup&gt;40&lt;/sup&gt;</td>
<td>25</td>
<td>29% increase in peak VO₂ in T, 5% increase in C</td>
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<tr>
<td>Dubach et al&lt;sup&gt;41&lt;/sup&gt;</td>
<td>33</td>
<td>No change in LV function in T</td>
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<tr>
<td>Giannuzzi et al&lt;sup&gt;34&lt;/sup&gt;</td>
<td>72</td>
<td>Improved LV ejection fraction and no change in LV size in T</td>
</tr>
<tr>
<td>Tyuni-Lenne et al&lt;sup&gt;42&lt;/sup&gt;</td>
<td>16</td>
<td>Increase in skeletal muscle oxidative enzymes</td>
</tr>
<tr>
<td>Belardinelli et al&lt;sup&gt;33&lt;/sup&gt;</td>
<td>46</td>
<td>24% increase in peak VO₂ in T, 6% decrease in C</td>
</tr>
<tr>
<td>Hambrecht et al&lt;sup&gt;43&lt;/sup&gt;</td>
<td>20</td>
<td>Endothelial dependent blood flow increased in skeletal muscle in T; this improvement strongly related to improvement in peak VO₂</td>
</tr>
<tr>
<td>Belardinelli et al&lt;sup&gt;38&lt;/sup&gt;</td>
<td>99</td>
<td>27% increase in peak VO₂ in T, 5% increase in C; hospitalizations and cardiac mortality reduced 19% and 22%, respectively, in T compared to C</td>
</tr>
<tr>
<td>Braith et al&lt;sup&gt;45&lt;/sup&gt;</td>
<td>19</td>
<td>Peak angiotensin (26%), aldosterone (32%), vasopressin (30%), and atrial natriuretic peptide (27%) all decreased in T vs C</td>
</tr>
<tr>
<td>Hambrecht et al&lt;sup&gt;46&lt;/sup&gt;</td>
<td>77</td>
<td>Peak cardiac output and stroke volume increased 19% and 15%, respectively; peripheral resistance decreased 21%</td>
</tr>
<tr>
<td>Pu et al&lt;sup&gt;46&lt;/sup&gt;</td>
<td>16</td>
<td>Randomized controlled trial; resistance training increased strength by 43%, muscle endurance by 29%, 6-min walk distance by 13%, increases in muscle morphology, histology, and oxidative capacity but not cardiac adaptations explained gains in exercise performance</td>
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</table>

<sup>*</sup>T = treatment group; C = control group.
Habel-Verge et al\textsuperscript{46} 8 weeks of exercise training decreased heart rate at fixed workload; 19% increase in peak oxygen uptake in T (p < 0.05) vs 8.5% increase in C (p = NS); 25% increase in physical working capacity in T (p < 0.05) vs 3% decrease (p = NS) in C.

Sire\textsuperscript{45} 44 Rate pressure product and rating of perceived exertion decreased by 13% at peak load; increase in exercise capacity in T was 38% higher than that in C at 6 months, and 37% higher after 12 months.

Jairath et al\textsuperscript{49} 29 Peak VO\textsubscript{2} increased by 25% after 3 mo with no differences between T and C; more than half of C patients exercised despite their group assignment.

*NS = nonsignificant. See Table 2 for abbreviations not used in the text.

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Table 3—Studies of Cardiac Rehabilitation Following Heart Valve Surgery

<table>
<thead>
<tr>
<th>Study</th>
<th>Patients, No.</th>
<th>Key Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Habel-Verge et al\textsuperscript{46}</td>
<td>10</td>
<td>8 weeks of exercise training decreased heart rate at fixed workload; 19% increase in peak oxygen uptake in T (p &lt; 0.05) vs 8.5% increase in C (p = NS); 25% increase in physical working capacity in T (p &lt; 0.05) vs 3% decrease (p = NS) in C.</td>
</tr>
<tr>
<td>Sire\textsuperscript{45}</td>
<td>44</td>
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</table>

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ranged between 15% and 50%. A randomized trial\textsuperscript{48} found improvements in the Minnesota Living with Heart Failure Questionnaire, after 2 months of exercise training, and the benefits persisted at the 14-month of follow-up. The improvements in quality of life paralleled the increases in peak VO\textsubscript{2}. Although no large-scale trials have focused specifically on the safety of exercise training and clinical outcomes, there is little evidence that exercise worsens LV function or increases heart chamber size.\textsuperscript{37,39} The European Heart Failure Training Group\textsuperscript{40} reported results from seven centers involving 134 patients and confirmed the beneficial effects of exercise rehabilitation on functional capacity. Clinical findings such as nonsustained ventricular tachycardia did not preclude a training effect or predict outcomes.

Although adverse events directly related to exercise training are infrequent, patients still can experience complications because of their overall increased risk due to LV dysfunction and an overactive sympathetic nervous system. To enhance safety, patients should undergo a period of supervised exercise to evaluate for potential heart failure-related complications. Supervision and monitoring in cardiac rehabilitation helps to reveal subtle signs and symptoms, which commonly precede clinical decompensation such as weight gain, decreased exercise capacity, and arrhythmias. Prompt intervention often can avert hospitalization for decompensated heart failure.

Recurrent symptoms and hemodynamic decompensation are leading causes of hospital admission. One study\textsuperscript{48} showed that heart failure patients in cardiac rehabilitation reduced readmissions by 19% and mortality by 22%. In stable patients with chronic class II and III heart failure who participated in exercise training for 14 months, survival was prolonged by an additional 1.82 years at a cost of $1,773 per life-year saved in the exercise group compared with nonexercising control subjects.\textsuperscript{41} Based on a recognized categorization scheme in which a value of < $20,000 indicates a highly cost-effective intervention,\textsuperscript{42} the cost-effectiveness for cardiac rehabilitation is substantial. Notwithstanding, more work is needed to help clinicians to identify which heart failure patients are most likely to gain maximum benefit from exercise training and to determine whether severity of illness, etiology of illness, age, gender, or other factors influence physiologic or clinical outcomes due to exercise training.

### Heart Valve Surgery

Heart valve surgery patients have no unique characteristics that differentiate them from patients with myocardial infarction, patients who have undergone coronary artery bypass surgery, or patients who have angina in terms of the need for cardiac rehabilitation.\textsuperscript{43} Before aortic or mitral valve replacement or repair, many patients are classified as being in NYHA class III to IV,\textsuperscript{44,45} with a functional capacity of three to four metabolic equivalents or less.\textsuperscript{46,47} Cardiac hemodynamics and symptomatology with valve disease are similar to those of heart failure. Pulmonary capillary wedge pressures are elevated and cardiac indexes are depressed, and there is extreme dyspnea with minimal exertion.\textsuperscript{47} Six months after surgery, the average improvement without cardiac rehabilitation is one NYHA class.\textsuperscript{44,45,47,48} Abnormal rest and exercise cardiac hemodynamics often persist for 6 to 12 months after surgery.\textsuperscript{48} Many patients continue to exhibit abnormal rest-to-exercise changes in LV ejection fraction.\textsuperscript{49}

The exercise component of cardiac rehabilitation is useful for reversing the symptoms associated with deconditioning. Women with mitral valve prostheses improved their peak metabolic equivalent capacity by 19% and their physical working capacity by 25% after undergoing an 8-week program, whereas control subjects did not improve.\textsuperscript{46} After aortic valve replacement, exercise training increased peak aerobic capacity and decreased rate pressure product and the rating of perceived exertion at a fixed workload.\textsuperscript{50} The increase in aerobic capacity in the exercise group was 35% higher than that in the control group at 6 months and was 37% higher after 12 months.
a randomized controlled study,49 patients who had aortic/mitral valve surgery were assigned to supervised exercise or a control group. Nevertheless, more than half of the control subjects routinely exercised on their own or joined community exercise programs. After the 3-month intervention period, peak \( \dot{V}O_2 \) was improved by 25% with no group differences.

For most patients, enhanced functional capacity leads to a greater ability to perform the activities of daily living and to tolerate activity for a longer duration with less perceived exertion.51 Older patients who undergo heart valve surgery have longer hospital stays and more complications, and they require more follow-up care after hospital discharge. Cardiac rehabilitation is also an opportunity to evaluate medical management and to educate patients about the safety of increasing physical activity and monitoring symptoms. Atrial fibrillation is a common arrhythmia that is seen in patients after heart valve surgery. Although uncontrolled atrial fibrillation is a contraindication to exercise rehabilitation, patients with controlled atrial fibrillation can participate safely in exercise. Because patients with chronic atrial fibrillation are usually receiving anticoagulation therapy, an important role for cardiac rehabilitation is providing patient education about avoiding the complications of anticoagulation therapy.

**CONCLUSION**

Although cardiac rehabilitation is currently underused in patients who have undergone percutaneous revascularization, heart transplantation, and heart valve surgery, and in patients with chronic heart failure, the efficacy of this important therapeutic modality warrants its more widespread application. Cardiac rehabilitation and supervised exercise training are effective for increasing functional capacity, favorably modifying disease-related risk factors, decreasing symptoms, detecting the signs and symptoms of disease before they become serious complications, and improving quality of life for affected patients. Among the limitations in the literature for the diagnoses discussed are studies with small sample sizes and the lack of adequate control groups. Furthermore, the intervention used has mostly been exercise training without the broader range of secondary prevention components that are currently recommended for cardiac rehabilitation, such as patient education, risk factor modification, and nutrition counseling, all of which are likely to augment the proven benefits of exercise. Clinicians should recognize that there are no supportive data for the efficacy of casual advice such as "go home and walk," "lose some weight," and "cut the fat in your diet." A formal referral to cardiac rehabilitation would increase the likelihood of participation and long-term compliance. A supervised hospital-based or clinic-based program, which assures that patients are receiving an appropriate exercise prescription in a safe environment, with related comprehensive lifestyle modification programs, represents the best opportunity for patient success. The challenge for cardiac rehabilitation programs is to provide services that are appropriate for older patients so that they can take full advantage of this effective therapy, thereby optimizing independence, health, and quality of life.

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