

Cardiopulmonary Rehabilitation and Cancer Rehabilitation.

1. Cardiac Rehabilitation Review

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ABSTRACT. Bartels MN, Whiteson JH, Alba AS, Kim H. Cardiopulmonary rehabilitation and cancer rehabilitation. 1. Cardiac rehabilitation review. *Arch Phys Med Rehabil* 2006; 87(3 Suppl 1):S46-56.

Cardiac rehabilitation includes not only the rehabilitation of people with ischemic heart disease but also those with congestive heart failure, heart transplantation, congenital heart disease, and other conditions. New advances in medical treatment have arisen, and there are new approaches in treatment, including alternative medicine and complementary care. New surgical approaches that help restore cardiac function have also been introduced, and rehabilitation professionals must be aware of these advances and be able to incorporate this knowledge into the practice of rehabilitation medicine.

Overall Article Objectives: (a) To identify major categories of cardiac disease, (b) to elucidate appropriate interventions and support for patients with coronary artery disease, (c) to describe the new interventions available for the treatment of cardiac disease, and (d) to describe the appropriate role of cardiac rehabilitation for people with various forms of cardiac disease.

Key Words: Arrhythmia; Cardiomyopathies; Cardiovascular diseases; Complementary therapies; Coronary artery bypass; Heart defects, congenital; Heart failure, congestive; Heart transplantation; Hypertension, pulmonary; Ischemic heart disease; Myocardial infarction; Nutrition; Rehabilitation.

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1.1 Clinical Activity: To evaluate a 47-year-old female executive who presents for cardiac rehabilitation 2 weeks after coronary stent placement for an ischemic event.

CARDIAC REHABILITATION AFTER a myocardial infarction (MI) has 4 phases. Phase 1, which takes place while the patient is still in the hospital, focuses on early mobilization, with activity progressing up to 5 to 7 metabolic equivalents (METs). This activity includes activities of daily living (ADLs) and community ambulation. In phase 2, patients recuperate at home and increase duration of activity and ambulation within the 5 to 7 MET limit. Phase 3, the outpatient program, starts 2 to 4 weeks after the MI. Evaluation includes

history, physical examination, a required exercise stress test, and review of previous cardiovascular test results. Patients exercise 3 times a week for 12 weeks with physician and telemetry monitoring. Training intensity is determined by peak heart rate that defines maximal intensity as achieved on the exercise stress test. Goals include improved function, greater aerobic capacity, and improved quality of life (QOL), patient education, and secondary prevention. On completion of phase 3, a repeat exercise stress test is performed to develop the exercise prescription for phase 4, the lifelong maintenance phase.

Coronary revascularization has reduced the morbidity and mortality associated with MI and unstable angina. After angioplasty alone, coronary restenosis that is sufficiently severe to require repeated intervention occurs within 12 months in 25% to 40% of patients. Coronary stenting reduces restenosis to 10% to 15% in most cases. Early restenosis within weeks after stent placement is related to acute thrombosis and occurs more commonly in emergent stents. Reduction in thrombosis rates to 0.5% to 2.5% has been achieved with the use of aspirin and clopidogrel (Plavix). Later restenosis associated with neointimal hyperplasia has been limited by drug-eluting (sirolimus or paclitaxel) stents.

Concern exists about the timing of stress testing and exercise participation after coronary angiography with stenting. Recent trials confirm that exercise stress tests and cardiac rehabilitation are safe in the early poststent period.¹⁻³

With shortening lengths of acute hospital stay, cardiac rehabilitation programs play a significant role in secondary prevention. Despite a greater percentage of women in the aging population, most research has focused on men. Women benefit as much as men from cardiac rehabilitation in improving aerobic capacity and more in improving function after coronary artery bypass grafting (CABG).⁴ However, rates of referral to cardiac rehabilitation and attendance by women are less than those of men. Women have a higher dropout rate than men because of transportation, medical comorbidities, and psychosocial impairment. To improve participation of women in cardiac rehabilitation, these nonmedical factors affecting compliance must be addressed.⁵

Survival is improved by cardiac rehabilitation. Patients involved in cardiac rehabilitation after an MI had a 3-year survival rate of 95% compared with 64% in nonparticipants. There is also a significant (28%) reduction in risk of recurrent MI.⁶

Heart disease is the foremost cause of mortality in older women and is 34% higher in African-American women than in white American women. Modifiable risk factors for the development of heart disease are the same in men and women. Compared with their male counterparts, women with diabetes have twice the risk for developing heart disease. A low test result for high-density lipoprotein (HDL) is also a stronger predictor of heart disease in women than men.

Primary and secondary prevention of heart disease is cost effective. Unfortunately, smoking rates are declining less for women than men, the prevalence of obesity is increasing, and 25% of women report no regular physical activity. About 52% of women over the age of 45 years have hypertension, and 40%

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Supported in part by the Vidda Foundation.

No commercial party having a direct financial interest in the results of the research supporting this article has or will confer a benefit upon the author(s) or upon any organization with which the author(s) is/are associated.

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doi:10.1016/j.apmr.2005.11.027

Table 1: American Medical Association Disability Guidelines

Designation	Functional Level at Which Symptoms Present	Oxygen Consumption (mL·kg ⁻¹ ·min ⁻¹)
Class 1	High community level	>25
Class 2	Low community level	20–25
Class 3	Household level	15–20
Class 4	At rest	<15

have elevated cholesterol levels. African-American women have a higher prevalence of diabetes and obesity. Women are counseled less often than men regarding exercise, nutrition, and weight loss. Outpatient cardiac rehabilitation programs are ideally suited to educate participants about lifestyle modifications.⁷

Hormone replacement therapy (HRT) to reduce heart disease is no longer recommended. Recent data show an increased risk for heart disease with combined estrogen-progestin HRT among healthy postmenopausal women.⁸

1.2 Clinical Activity: To plan the rehabilitation for a 66-year-old, obese, diabetic, hypertensive person who is referred on the second postoperative day after CABG.

CABG is now a routine procedure in the United States. It decreases symptoms and increases survival. Morbidity and mortality from the procedure have also markedly declined, and new less-invasive procedures are now allowing sicker patients to have surgery. The classical cardiac risk factors including hypertension, diabetes, smoking, inactivity, hypercholesterolemia, advanced age, and obesity are usually present. A primary prevention program is too late for these patients, but a cardiac rehabilitation program will prevent recurrent morbidity and mortality through secondary prevention. Education and counseling should begin before surgery, if possible.^{9–11}

The educational program must address reversible causes of ischemic heart disease (IHD). These include obesity, sedentary lifestyle, hyperlipidemia, cigarette smoking, and conditions such as diabetes mellitus and hypertension. Dietary counseling and exercise may assist in weight reduction. A decrease in dietary saturated fats and cholesterol may improve lipoprotein levels. Both behavior modification and the addition of lipid-lowering medications are proven to be beneficial in combination. Tight control of blood sugar in diabetes can achieve improved outcomes for heart disease. People who have not stopped smoking should be enrolled in cessation programs, with pharmacologic interventions as needed. Finally, appropriate treatment of hypertension with afterload reduction, β -blockade, and use of antiplatelet agents improve overall survival.^{12–14}

Mobilization after surgery must progress as rapidly as possible to prevent thromboembolism, deconditioning, decubitus ulcers, and pneumonia. Patients should be sitting in a chair on the first postoperative day; limited ambulation should be started on the second postoperative day. Ideally, patients should progress to independent functional mobility by the fifth postoperative day. Before discharge home, patients should have a complete and detailed set of exercise instructions. The physical limitations on people after CABG are usually related to the surgical approach. For median sternotomy, patients will have to limit upper-extremity loading and thoracic torque. In minimally invasive CABG and robotic surgery, there are usually no limitations to motion, because the sternum is intact.

Full exercise programs are not started with an outpatient until the person has achieved a complete recovery from the CABG. In a classical median sternotomy, this point is 6 weeks

from the surgery, to allow sternal union. In other approaches, the time is usually 3 to 4 weeks. At the time of initiation of the outpatient program, an aerobic program with gentle resistance exercise and without Valsalva's maneuver is recommended. The early program usually lasts 8 to 12 weeks, with encouragement to adhere to a lifetime lifestyle modification, and progresses thereafter.^{15–17}

Inpatient acute rehabilitation may be needed in a patient with postsurgical complications such as a stroke, peripheral vascular disease, or preexisting disabilities. In these people, a program of acute rehabilitation must include the educational modifications for secondary prevention and a component of aerobic training for cardiac health. As the general population ages and the comorbidities associated with aging accumulate, rehabilitation services will have to provide secondary cardiac prevention interventions on both an inpatient and outpatient basis.^{18–20}

1.3 Clinical Activity: To discuss the medical and rehabilitation management of a previously very active 30-year-old woman with peripartum cardiomyopathy and ejection fraction of 20%.

Congestive heart failure (CHF) is the most costly condition for Medicare, with 2 million hospitalizations and nearly 300,000 deaths each year. Five million people have CHF, with an incidence of 500,000 more each year.²¹

CHF severely limits exercise capacity. This condition has been categorized by American Medical Association Guidelines²² (table 1). There is little correlation between left ventricular ejection fraction (LVEF) and symptoms or functional capacity. Coronary artery disease is the underlying cause of CHF in two thirds of cases, the remainder being associated with a variety of causes. Adaptation to CHF includes abnormal left ventricular dilatation and hypertrophy. CHF can present with decreased exercise tolerance, fluid retention, or incidental finding of decreased LVEF. The exercise stress test with respiratory gas analysis helps to classify the severity of CHF and guide exercise prescription.

Cardiomyopathies are classified according to etiology.²³ Peripartum cardiomyopathy, which is defined by the presence of heart failure in the last month of pregnancy or up to 5 months postdelivery, affects up to 1300 American women yearly. It is more common with advanced maternal age, multiparity, and being African American.²⁴

Research continues to define the role of medications to treat CHF (table 2). For example, spironolactone improves long-term outcomes, with a 35% reduction in rehospitalization from CHF.²⁵

A multidisciplinary approach in managing CHF improves quality of life and reduces health care expenditure. Educating the patient and caregivers about CHF reduces rehospitalization, lowers mortality rates, and extends survival. A worsening of CHF symptoms warrants immediate evaluation.²⁶

Table 2: Pharmacologic Management of CHF

Medications	Indication	Action
Diuretics	Fluid retention	Reduce fluid overload
Angiotensin-converting enzyme inhibitors	Limit left-ventricular remodeling	Reduce afterload
Digoxin	Inotropic incompetence	Improve contractility
β -blockers	Hyperadrenergic state	Autonomic modulation

Hypertension, diabetes, and pulmonary, lipid, and thyroid disorders should be optimally managed. Cessation of alcohol consumption and smoking are essential. Weight control improves New York Heart Association functional class by a grade or more (out of a 1–4 scale) in 90% of patients. Daily sodium intake should be 2g or less.

Sleep apnea is reported in 50% of patients with stable CHF. Continuous positive airway pressure at night is associated with reduced ventricular arrhythmias and improved ejection fraction. Other therapies in advanced CHF include biventricular pacing and insertion of an implantable cardioverter defibrillator (ICD).²⁴

The ability to perform aerobic exercise is related to cardiac output and peripheral utilization of oxygen. In healthy people, cardiac output increases 4- to 6-fold because of a 2- to 4-fold increase in heart rate and a 20% to 50% augmentation of stroke volume. In CHF, cardiac output at peak exercise may be less than 50% of normal because of a significant reduction in stroke volume. Thus, cardiac output during activity is primarily achieved with an increase in heart rate.²⁷ Peripheral vasodilation is impaired because of excessive sympathetic stimulation and reduced nitric oxide. Skeletal muscle in CHF shows decreased oxidative type 1 fibers and reduced mitochondrial enzymes.

Exercise training in CHF improves these central and peripheral abnormalities and increases exercise tolerance, duration, and peak oxygen consumption (VO_2). Exercise attenuates left ventricular remodeling,²⁸ improves myocardial perfusion and QOL, and decreases mortality and hospital admission rates. There was no evidence that exercise in this population was dangerous.²⁹ An individualized exercise prescription is recommended in CHF. There is a greater improvement in left ventricular function, peak VO_2 , and muscular strength in programs that combine aerobic conditioning and strength training.³⁰

In an exercise program for CHF, training intensity is best set at 70% to 80% of maximal oxygen consumption achieved on the pre-exercise stress test. In very debilitated patients, lower intensities can be used at first, with increases as patients become more conditioned. A Borg rating of perceived exertion scale of 12 to 13 is well tolerated in exercise.

Aerobic exercise sessions should include a prolonged warm up and cool down of 10 to 15 minutes. The exercise period should be 30 to 45 minutes, 3 to 5 times a week, and can be supplemented with walking on the off days. Combining resistance and aerobic training increases muscle strength in addition to increasing peak VO_2 . Telemetry monitoring is needed initially, and unmonitored home training can follow. Exercise benefits of training are lost within 6 months if exercise is not continued.

1.4 Clinical Activity: To consider the rehabilitation management of a 35-year-old young mother of twins who was diagnosed with primary pulmonary hypertension 3 months postpartum.

Inherited primary pulmonary hypertension (PPH) is quite rare, with 1 to 2 cases per million or 300 new cases per year in the United States.³¹ Idiopathic PPH is more common, with 163,000 hospital discharges and 3065 deaths attributed to PPH in 2000. PPH is more common in women between the ages of 21 and 40 years. Morbidity is more common with secondary pulmonary hypertension from lung disease, chronic emboli, collagen vascular disease, and heart disease. Life expectancy for severe untreated pulmonary hypertension is 2 to 3 years.³¹ Recent medical advances have improved survival. Combined with lung transplantation in refractory cases, survival is dou-

bled. Treatments producing vasodilatation of the pulmonary vascular bed have improved management of pulmonary hypertension. These include positive pressure ventilation for hypoxemic sleep apnea, supplemental oxygen, prostacyclines, calcium channel blockers, and nitric oxide modulators such as sildenafil.³² Continuous ambulatory intravenous infusion of epoprostenol markedly improves function and survival but is associated with the encumbrance and costs of the treatment. Recently, oral analogues have come into use, with the approval of bosentan in 2003. Optimal medical management of pulmonary hypertension includes arrhythmia control, anticoagulants, digoxin, and diuretics. Supplemental oxygen was shown in the nocturnal oxygen treatment trial and the long-term oxygen treatment trial to prevent the development of secondary pulmonary hypertension and is an important part of treatment of all forms of pulmonary hypertension.³³

Exercise used to be considered a high-risk activity for people with pulmonary hypertension, but after a cardiac rehabilitation program, in conjunction with optimal medical treatment, daily exercise can be safe and beneficial. Functional independence and exercise capacity in patients with severe pulmonary hypertension are similar to that of people with classes 3 and 4 CHF. Exercise benefits are similar, with improved functional status, decreased depression, and improved health status. Exercise stress testing is part of the management of pulmonary hypertension and is used to prescribe a safe exercise program. Resistance training is avoided, and aerobic exercise is performed up to 60% of maximal aerobic capacity for 20 to 30 minutes 3 times a week. Supplemental oxygen is recommended to maintain hemoglobin oxygen saturations above 90%. Often, the people affected are young, and this conditioning will facilitate either a return to work or maintenance of employment.^{34,35}

1.5 Clinical Activity: To discuss the rehabilitation management of a 65-year-old man with intermittent claudication recently diagnosed with stable angina.

Peripheral arterial disease (PAD) and IHD often coexist, and there are management overlaps. PAD is present in up to 10 million people in the United States, and 20% of people over age 70 are affected.³⁶

The ankle-brachial index (ABI), which compares systolic blood pressure (SBP) at the ankle to brachial SBP, is used to classify the severity of PAD (table 3). Intermittent claudication is present in up to 40% of patients with PAD. Five percent of people over age 55 have intermittent claudication. Over the 5 years after diagnosis, 75% remain stable, 16% develop severe limb ischemia, 7% require lower-extremity revascularization, and 4% require amputation.

IHD is the leading cause of mortality in the United States, responsible for 20% of deaths and presenting with chronic stable angina in 50% of patients. The annual incidence of angina is 213 per 100,000 population over 30 years and there are at least 16 million people with stable angina.³⁷ On nonin-

Table 3: Classification of PAD Severity

Severity	ABI
Normal	>1.0
Mild	0.8 to 1.0
Moderate	0.6 to <0.8
Severe	0.4 to <0.6
Very severe	<0.4

Table 4: Medications for PAD and IHD

Medications	Action	Indication/Effect
Statins (hydroxymethyl glutaryl coenzyme A reductase inhibitors)	Decreased atherosclerosis Proangiogenic	Dyslipidemia Improved pain-free walking Reduced mortality
Aspirin	Antiplatelet aggregation	Decreased MI by 18% Decreased need for surgery
Clopidogrel (Plavix)	Antiplatelet aggregation	Reduced risk of MI/vascular death by 23.8% more than aspirin
β -blockers	Sympatholytic	Reduced risk of MI
Angiotensin-converting enzyme inhibitors	Afterload reduction	Reduced mortality Reduced ischemic events
Calcium channel blockers	Vasodilation	Reduced claudication Reduced angina
Nitrates	Vasodilation	Reduced claudication Reduced angina
Pentoxifylline (Trental) Cilostazol (Plental)	Red blood cell deformability	Decreased pain Increased walking distance Cilostazol improves HDL, ABI Cilostazol not to be used in CHF

NOTE. For a review of the drug therapy for PAD and IHD, see Hiatt.³⁹

vative evaluation, 20% to 60% of patients with PAD have IHD. The incidence increases to 90% with angiography.³⁸ Once intermittent claudication develops, there is a 20% to 30% 5-year mortality from IHD, and for each 0.1 decrement of the ABI, there is a corresponding 10% increase in relative risk for nonfatal MI. Cardiovascular disease is the most common cause of death in patients with PAD, accounting for up to 75% of deaths.³⁸ Rehabilitation of stable angina is usually outpatient; however, there is no Medicare reimbursement for the rehabilitation of PAD and intermittent claudication. Although patients are instructed to “go home and walk,” home-based walking programs are not proven effective in treating intermittent claudication or PAD. Identification of myocardial ischemia in patients with PAD qualifies them for monitored cardiac rehabilitation.

Because patients with intermittent claudication have difficulty walking, a treadmill exercise stress test may be of limited use, and bicycle ergometry can be used as an alternative to set a reasonable heart rate intensity for aerobic training. However, treadmill walking is essential in the exercise rehabilitation of intermittent claudication.

The primary and secondary risk factor modification in IHD and PAD are essentially the same. Smoking cessation is paramount in slowing the progression to critical leg ischemia and reduces the risk of MI and vascular deaths. Pharmacologic management of PAD and IHD are summarized in table 4.³⁹

Cardiac rehabilitation, based on the exercise stress test, is safe in stable angina, with a nonfatal rate of 1 myocardial infarction per 294,000 patient hours of exercise. Exercise intensity as guided by heart rate response is set below the double product (SBP by heart rate) in patients whose ischemic signs or symptoms were first noticed on exercise stress test. Patients should undergo monitored aerobic exercise 3 times a week for 45 to 60 minutes. Intensity is gradually increased under supervision to maintain the training heart rate. With training, vagal

tone increases and heart rate is lower at rest and submaximal levels of exercise.⁴⁰ Improved myocardial perfusion with exercise therapy in patients with stable angina is likely related to improved endothelium-dependent vasodilatation.

Exercise training also improves the sense of well-being, reduces disability, lowers lipids, limits disease progression, and may enhance disease regression. There is a clinically significant training effect that is caused by enhanced oxygen extraction by the trained muscles. Cardiac rehabilitation reduces subsequent risk of cardiac events in stable angina with a 31% reduction in mortality rate. It may be more effective in managing stable angina than percutaneous coronary intervention with stenting, with a higher event-free survival rate.⁴¹

Modification of the exercise prescription in patients with PAD, intermittent claudication, and IHD includes a training intensity that produces moderate claudication pain within the first 5 minutes of treadmill walking. The walking is interspersed with rest periods long enough for symptoms to subside. Exercise is continued in an exercise-rest-exercise interval cycle to achieve the intensity and overall duration of a normal cardiac rehabilitation program. Exercise therapy in PAD improves pain-free walking time by 189% and maximal walking time by 80% to 150% starting within 4 weeks of exercise, and benefit is maximized in 12 months.⁴² This finding exceeds the benefit seen with medication alone. There is a 31% improvement in the ability to carry out ADLs and a 62% increase in physical activity.

Mechanisms suggested to improve walking distance and functional abilities include angiogenesis, enhanced collateral blood flow, redistribution of blood flow from inactive to active muscles, improved blood and plasma viscosity, increased endothelial-dependent vasodilatation, and increased oxidative capacity.³⁵ Exercise improves walking economy by increasing biomechanical and metabolic efficiency.

1.6 Clinical Activity: To discuss the impact of ventricular arrhythmias in a 68-year-old man with ischemic cardiomyopathy being evaluated for cardiac rehabilitation.

Arrhythmias are frequently noted in patients with IHD after MI or CABG and are also associated with cardiomyopathy. The most significant risk of cardiac rehabilitation is arrhythmia. Up to 5% of people in cardiac rehabilitation after CABG fail to complete the program because of arrhythmias. The rate of arrhythmias was similar in men and women, and arrhythmia was related to diabetes, hyperlipidemia, hypertension, and older age.⁴³

Prognosis in ischemic cardiomyopathy is poor, particularly in patients with multiple MIs and ventricular arrhythmias. There are 300,000 sudden cardiac deaths per year because of malignant ventricular arrhythmias. Without underlying heart disease, the presence of premature ventricular contractions (PVCs) does not affect activity tolerance or survival. After MI and CABG, the presence of frequent PVCs identifies patients at increased risk of developing ventricular tachycardia or sudden cardiac death. Patients can be asymptomatic or experience significant hemodynamic consequences including syncope. Patients with IHD with reduced LVEF and ventricular tachycardia are more likely to develop ventricular fibrillation. Ventricular tachycardia after MI and CABG has a 2-year mortality of 30%.

Ventricular tachycardia and ventricular fibrillation within 30 days of CABG is seen in up to 1.6% of patients but accounts for 21.7% of deaths. Detection of arrhythmias warrants a search for correctable factors, including ischemia. Pharmacologic management includes nitrates, β -blockers, and amiodarone. Ablative therapy or an ICD can be considered for life-threatening ventricular tachycardia or ventricular fibrillation. ICDs decrease mortality by 31% in patients with nonsustained ventricular tachycardia after MI complicated by reduced ejection fraction compared with pharmacologic management alone. Over a 5-year follow-up, only 4% of patients with an ICD died suddenly, whereas 60% of patients without an ICD who did not respond to pharmacologic treatment of their ventricular tachycardia or ventricular fibrillation experienced sudden death.⁴⁴

In survivors of cardiac arrest from ventricular arrhythmias, ventricular tachycardia or ventricular fibrillation reoccurs at an annual rate of up to 40%. However, exercise therapy is safe and effective in monitored settings, despite the concern that exercise is arrhythmogenic.⁴⁵ For safety in cardiac rehabilitation, ongoing ischemia must be stabilized and CHF must be optimally managed. The exercise stress test on antiarrhythmic medications is essential to screen for ventricular arrhythmia and to establish cardiac rehabilitation parameters. Target heart rate is set at a level below which ventricular arrhythmias are noted. A standard telemetry-monitored cardiac rehabilitation program is followed. Warm-up and cool-down periods decrease the frequency of arrhythmias by promoting coronary perfusion. Patients with malignant ventricular arrhythmia, minimally reduced ejection fraction, and very good exercise tolerance are far more likely to experience ventricular tachycardia or ventricular fibrillation during cardiac rehabilitation than patients with very limited exercise capacity. They should decrease exercise intensity and increase exercise duration and frequency.⁴⁴

Nearly 80% of patients with a history of ventricular arrhythmias will have a ventricular arrhythmia during inpatient cardiac rehabilitation. Life-threatening arrhythmias occur in 35% of patients, with up to 1 urgent complication in 173 patient-hours of exercise. The arrhythmias continue in the outpatient setting, with up to 30% incidence of ventricular tachycardia with up to 1 urgent complication in 138 patient-hours of exercise. How-

ever, only 1.3 fatalities per million patient-hours of cardiac rehabilitation have been reported in this population.

Comparing patients with and without an ICD, baseline physiologic parameters are similar. If patients are protected with an ICD, care must be taken to keep the peak heart rate below that which will initiate anti-tachycardia pacing and eventual ICD discharge. Because supine exercise produces a greater myocardial oxygen demand than upright exercise, it is recommended that patients prone to arrhythmias and those with ICDs exercise in the upright position (walking, biking, stair climbing) rather than with recumbent activities.⁴⁶ However, patients with an ICD make smaller absolute improvements in aerobic capacity compared with patients without an ICD, because their peak exercise is usually limited because of lower target exercise heart rate.⁴⁷ In patients with ischemic cardiomyopathy and CHF who have conduction abnormalities, biventricular pacing with leads placed in both ventricles synchronizes ventricular contractions. This increases LVEF, exercise tolerance, aerobic capacity, 6-minute walk distance, and QOL and decreases CHF exacerbations. Biventricular pacemakers, when combined with ICDs, significantly improve mortality compared with biventricular pacing alone.⁴⁸⁻⁵⁰

The initial relief of anxiety with the implantation of the ICD is often followed by anticipatory anxiety over potential firing of the ICD. This may eventually lead to depression, impaired QOL, and severe limitation of activity.⁵¹ Social support groups and cardiac rehabilitation programs can alleviate this fear and improve QOL.⁵²

1.7 Clinical Activity: To define the rehabilitation indications, goals, precautions, and prescription for a 45-year-old man who presents with a massive anterior wall MI, requiring left ventricular assist device and eventual heart transplantation.

The technique of orthotopic cardiac transplantation has improved, and 5- and 10-year survival rates are now 82% and 74%, respectively. Typical cardiac transplant patients are middle aged and have often suffered from months of preoperative deconditioning, general muscle weakness, depression, and anxiety. Transplantation resolves the cardiac disability, but a comprehensive rehabilitation approach is necessary.

Pretransplant management of the patient includes secondary prevention programs and an exercise conditioning program. Any of the several models of left ventricular assist device (LVAD) and intravenous inotropes sustain cardiac function and allow mobilization of these patients. Some inpatient acute rehabilitation units can accommodate patients who have either inotropes or LVAD in a comprehensive inpatient cardiac rehabilitation program. This facilitates earlier successful transplantation through more rapid mobilization.^{10,53,54}

After cardiac transplantation, vagal inhibition to the sinoatrial node is lost, resulting in a resting tachycardia near 100bpm.^{55,56} Stroke volume may be reduced because of diastolic dysfunction from increased myocardial stiffness in the new heart. Possible causes of this dysfunction include myocardial ischemia from accelerated atherosclerosis, side effect of immunosuppressants, and prolonged ischemic time between heart explantation and reimplantation.^{53,54} Fortunately, cardiac output can be increased by nonneural mechanisms such as circulating catecholamines inducing a chronotropic and inotropic response.^{10,55,57} The sympathetic denervation causes a blunted heart rate response to an incremental exercise stress test, with peak heart rates 20% to 25% lower than age-matched controls. Resting hypertension is common, caused in part by the renal effects of antirejection medications.⁵⁶

A 10% to 50% loss of lean body mass from lack of activity and a high-dose of steroids in the perioperative period can be seen. A strengthening program is needed to recover the lost lean body mass. The cardiac rehabilitation program in transplant patients must address their overall conditioning as well as their cardiac function. Walking, jogging, and cycling are recommended exercises, with individualized guidelines for transplant patients. Finally, the education program also must include the new medication regimen and education about immunosuppression and rejection. A properly structured cardiac rehabilitation program can provide the ideal setting for this.

1.8 Clinical Activity: To discuss the rehabilitation program for a 17-year-old immigrant from South America who presents with cyanotic heart disease due to an uncorrected congenital ventricular septal defect and subsequent Eisenmenger's syndrome.

Patients with congenital heart disease (CHD) initially are cared for by pediatric physicians. With advances in cardiac medical and surgical management of CHD, more patients reach adulthood and may require physiologic treatments. Most of the principles of rehabilitation in CHF and pulmonary hypertension apply to patients with CHD. Significant concerns include sudden death and hemodynamic instability. Specific exercise parameters are determined based on the original defect(s) and the type(s) of corrective surgery. Guidelines from the treating cardiac team and recent cardiac evaluations are essential.⁵⁸

CHD classification helps to plan medical treatments and exercise. CHD is divided into cyanotic and noncyanotic disease, further defined by the presence or absence of shunting in the heart. Pulse oximetry and echocardiography help to classify patients and assign precautions. Safe exercise depends on matching the type of CHD with the correct form of exercise. Dynamic isotonic exercise presents a volume load that is contraindicated in restrictive cardiac diseases, and static isometric exercise presents a pressure load to the ventricles that is contraindicated in CHD with shunting. Specific concerns in patients with Marfan syndrome include contact sports and high-impact activities, because these patients may experience sudden death from aortic rupture.⁵⁹⁻⁶¹ Tables 5 and 6 summarize specific conditions related to activity guidelines.

1.9 Clinical Activity: To discuss the management of a 78-year-old man with cognitive deficits who has had CABG.

Patients presenting for CABG may have preexisting cognitive deficits. IHD and dementia often coexist and are associated with aging. Prior slow cognitive decline may be undetected without formal cognitive testing. Marginally functioning patients are vulnerable to further neurologic impairment after CABG and preexisting cognitive deficits can be unmasked. Full cognitive recovery often does not occur.

Cognitive impairments after CABG include defects associated with attention, concentration, short-term memory, fine motor function, and speed of mental and motor responses. Impairments may be transient or more persistent, with a reported incidence from 20% to 80%.

Encephalopathy, defined as delirium, coma, or seizure at any time after the first 24 hours after surgery, has a prevalence of 6.9%. Stroke is reported in 2.7% of patients. Length of stay and mortality are increased with encephalopathy and/or stroke after CABG.⁶² Factors associated with encephalopathy include increasing age, previous stroke, hypertension, diabetes, carotid bruit, and cardiopulmonary bypass time. Each additional hour on cardiopulmonary bypass is associated with doubling the prob-

ability of postoperative encephalopathy. Predictors for CABG-associated stroke include increasing age, previous stroke, and hypertension.

A diagnostic search for neurologic damage after CABG shows a significant prevalence of injury. Diffusion-weighted magnetic resonance imaging identified new cerebral ischemic lesions in 26% of cases. However, the presence of lesions does not correlate with impairments on neurologic and neuropsychologic testing. Abnormalities in magnetic resonance spectroscopy, an estimation of cerebral metabolism, correlate with postoperative neuropsychologic deficits. This suggests transient metabolic neuronal disturbance as a cause of postoperative cognitive changes.⁶³

Neuropsychologic deficits are most marked at day 3 after CABG, and these usually recover over the following week.⁶⁴ Mechanisms responsible for neuropsychologic deficits include hypoperfusion during cardiopulmonary bypass; venous hypertension caused by manipulation of the heart during surgery; microemboli of air, atheroma, fat, and platelet aggregates originating from the cardiopulmonary bypass circuit and the ascending aorta; and a systemic inflammatory process that produces cerebral swelling.

Off-pump CABG surgical techniques were developed to minimize the incidence of cognitive deficits. Whereas on-pump techniques require a median sternotomy and the use of a pump-oxygenator, minimally invasive procedures are performed through smaller lateral chest wall incisions without the need for a pump-oxygenator. Currently, 25% of CABG surgeries are performed off-pump. Overall, outcomes of off- and on-pump surgeries have remained the same, but cognitive function has been better in patients undergoing off-pump surgery.⁶⁵ Ten weeks after CABG, 10% of off-pump patients remain impaired compared with 40% of on-pump patients. Cognitive changes may still be noted 5 years after CABG. The number of cerebral microemboli during cardiopulmonary bypass, postoperative short-term cognitive change, and the degree of cognitive recovery at 8 weeks are predictors of cognitive score at 5 years.⁶⁶ Cognitive performance after CABG is an independent predictor of mortality at 8 to 10 years after CABG. Similar cognitive changes and outcomes are also seen after valve surgery, with more significant decline after valve replacement compared with valve repair.⁶¹

A neurocognitive evaluation should be part of the routine assessment of a comprehensive cardiac rehabilitation program. It is best completed before hospital discharge, and if cognitive decline is detected, patients should be followed up with serial evaluations. A program of cognitive remediation is recommended, but outcome studies evaluating efficacy are lacking. Patient and family education is essential, because cognitive deficits may influence safe functioning at home. Differentiating cognitive deficits from anxiety and depression disorders is important, because they can both present with temporary cognitive changes or may mask underlying neurocognitive decline related to CABG.

1.10 Educational Activity: To provide risk factor modification advice to a 57-year-old, obese, diabetic, hypertensive patient with IHD and CHF.

This patient represents a classic presentation of someone with the multiple metabolic syndrome or syndrome X. The risk factors for high cardiac mortality are encompassed by multiple metabolic syndrome, a process characterized by obesity and associated with hypertension, diabetes, hypercholesterolemia, and dyslipidemia. The syndrome correlates directly with obesity, and weight loss corrects many of the abnormalities. Unfortunately, no single weight loss program is most effective, but a combination of

Table 5: Summary of Congenital Defects and Clinical Findings

Anomaly	Symptom/Finding	Activities/Treatments	Comments
Shunts (ASD, VSD, PDA)	L to R: fatigue, dyspnea R to L: dyspnea, cyanosis, fatigue	Small to moderate L to R: participate in most activities R to L: low-level activity; PA pressures determining factor	R to L: present at load with CHF End stage: presents with L to R in Eisenmenger's physiology
Hypertrophic cardiomyopathy	Dyspnea Sudden death Family history, screening echocardiogram	β -blocker Calcium channel blocker Diuretics Surgical septostomy No competitive athletics Limit to 60% to 70% maximum exertion	1/500 in population Septal thickening most common finding
Stenotic bicuspid aortic valve	Dyspnea CHF Rare sudden death	Asymptomatic mild: no restriction Asymptomatic moderate: low level activity Symptomatic moderate or severe: low-level activity, consider correction Symptomatic unrepaired severe: maintain fitness through walking/low-level activity	Incidence 1%: most common congenital anomaly Moderate stenosis or regurgitation: consider correction
Coronary artery anomalies	Sudden death often first symptom Occasionally identified by cardiac catheterization	Avoid strenuous activity until corrected: especially if left coronary artery between the aorta and pulmonary artery	Rare, but seen in 20% of sudden death with sports Can present with LV dysfunction in a child or young adult
Tetralogy of Fallot (VSD, RV hypertrophy, overriding aorta, PS)	Dyspnea Cyanosis	Repaired with good hemodynamics: normal activity Significant RV failure or PH: limited	Usual correction with systemic to pulmonary shunt PH major limiting factor in some
Marfan syndrome (congenital defect in fibrillin)	Chest pain Aortic dissection Sudden death	No impact on activities unless aortic root repaired Otherwise normal activities	Dilation of ascending aorta and risk of dissection Repair via aortic root replacement
Tricuspid atresia	Dyspnea Intolerance of high-level activity	Low- to moderate-level activity CPET for high-level activity Consider supplemental oxygen	Usually paired with ASD Fontan repair (right atrium to pulmonary artery)
Ebstein's anomaly (apical displacement of the tricuspid valve)	Dyspnea Cyanosis Atrial arrhythmias	Mild: unlimited Moderate: CPET determined Severe: only low-level exercise	Decreased RV function Up to 85% with ASD Echocardiogram determines extent of lesion
Patent ductus arteriosus	Dyspnea CHF from volume overload	Intolerant of high-volume activity Limited by pulmonary pressures	Usually a L to R shunt Behaves like mitral regurgitation
Pulmonary valve stenosis	Fatigue Dyspnea Syncope	Mild or repaired: high-level activity Severe: avoid volume activity	Surgical correction usually achievable
Aortic coarctation (congenital narrowing of the descending aorta)	Different BP between arms or arms and legs Hypertension	Avoid isometric static exercise Low-level exercise until corrected	Associated with bicuspid aortic valve

Abbreviations: ASD, atrial septal defect; BP, blood pressure; CPET, cardiopulmonary exercise test; L, left; LV, left ventricle; PA, pulmonary artery; PDA, posterior descending artery; PH, pulmonary hypertension; PS, pulmonary stenosis; R, right; RV, right ventricle; VSD, ventricular septal defect.

Table 6: Exercise Limits in Selected Conditions

Defect	Normal Exercise	Low- to Moderate-Intensity Exercise	Very-Low-Intensity Exercise
Atrial septal defect	Normal pulmonary pressure	Moderate PH	Eisenmenger's physiology
Ventricular septal defect	Small to moderate with no symptoms	Large	Eisenmenger's physiology
Patent ductus arteriosus	Small	Moderate	Large, unrepaired
Pulmonary stenosis	Mild with no symptoms	High gradient with no symptoms	High gradient with symptoms
Aortic stenosis	Mild	Moderate with no symptoms	Severe aortic stenosis
Tetralogy of Fallot	Normal pulmonary pressures	Moderate PH	Uncorrected or severe PH

Abbreviation: PH, pulmonary hypertension.

dietary restraint and moderate exercise seems to achieve the best long-term sustainable weight reduction. Intriguingly, multiple metabolic syndrome and cardiac disease often coexist. Treatment of obesity alone will achieve both primary and secondary cardiac risk factor modification. In people with multiple metabolic syndrome, there is a clear association of the severity of the syndrome and heart disease.^{67,68} Dietary interventions for multiple metabolic syndrome and IHD both are focused on reducing cholesterol, controlling diabetes, lowering weight, and controlling hypertension.

Optimal lipid management is achieved by combining exercise and lipid-lowering agents. A diet with less than 300mg of cholesterol per day, low saturated fats, and avoidance of *trans*-saturated fats is effective at modifying IHD risk.⁶⁹⁻⁷¹

Optimal diabetic management is achieved by combining exercise with weight loss. This includes increasing complex carbohydrates and avoiding refined sugars to reduce insulin resistance and hyperinsulinemia.⁶⁶

Optimal management of hypertension and fluid status is essential in CHF and IHD. Strict control of salt intake leads to better control of hypertension and CHF and modifies the course of IHD. Guidelines include sodium restriction below 2g/d for CHF and below 4g/d for hypertension and IHD.

Even people in the high-normal weight groups to low-level overweight groups (body mass index [BMI], 24–28kg/m²) are at risk of multiple metabolic syndrome.^{72,73} Primary and secondary prevention aimed at this group is effective. Identifying multiple metabolic syndrome and continuing patient nutrition education through cardiac rehabilitation programs is an essential part of treating people with heart disease.

1.11 Educational Activity: To provide counseling to a 42-year-old uninsured day laborer who was referred for rehabilitation after presenting to the emergency department with an MI.

Reimbursement for outpatient cardiac rehabilitation is often not available. Even well-insured patients often have limited coverage. Uninsured patients may have no access to services. However, the greatest limitation to access is lack of physician referral. Medicare will only reimburse for outpatient cardiac rehabilitation after CABG, MI, and stable angina, despite its demonstrated benefit for patients with CHF, cardiac transplant, pulmonary hypertension, CHD, postangioplasty, post-valve replacement, and arrhythmias. Out-of-pocket expenses for cardiac rehabilitation are usually \$100 to \$150 per session, making a total cost of \$2400 to \$5400 for a customary program of 24 to 36 sessions. There is no insurance coverage for maintenance or wellness programs. Most programs offer these services at an out-of-pocket cost of \$50 to \$100 a month to people who have completed outpatient cardiac rehabilitation.⁷⁴⁻⁷⁶

Coverage varies by locality and by state, and the rules and regulations are constantly changing. Coverage for cardiac re-

habilitation is being sought for CHF and other conditions at this time. Additionally, despite the fact that cardiac and pulmonary rehabilitation have been recognized in the prospective payment system coding for inpatient rehabilitation, they will no longer qualify as rehabilitation diagnoses under the recently issued reimbursement guidelines from the Center for Medicare and Medicaid Services.⁷⁷ In the future, cardiac disease will still be seen in the inpatient setting, but primarily as a comorbidity to other diseases, or in the setting of qualifying noncardiac complications of primary heart disease.

1.12 Clinical Activity: To discuss primary prevention of coronary heart disease in a 45-year-old woman with paraplegia who smokes.

Longer life expectancy in patients with spinal cord injury (SCI) is associated with an increased risk for mortality from cardiovascular disease (CVD). Cardiac disease is the primary cause of death in nearly 20% of patients with SCI and contributes to nearly 25% of all deaths over 6 years after SCI. CVD is the leading cause of death (46%) for patients with SCI for more than 30 years and in patients over the age of 60 years (35%).

Patients with SCI are reported to have multiple risk factors for IHD including smoking (54%), high total cholesterol (32%), high low-density lipoprotein (41%), HDL (52%), and diabetes (7%). HDL is reduced further in those who have complete, higher-level, and longer-duration SCIs.⁷⁸ Prevalence of hypertension in people with SCI is similar to that in the general population. As in able-bodied populations, people with SCI can benefit from dietary and lifestyle modification, which will reduce their risk of developing IHD.

Diagnosis of IHD in SCI may be difficult, because patients with high SCI may not experience chest pain.⁷⁹ An arm ergometry exercise stress test with nuclear imaging is useful to detect ischemia in patients with paraplegia.⁸⁰ For higher-level lesions, pharmacologic stress testing is required.

Exercise training responses in SCI are limited by reduced cardiac preload, decreased muscle mass, and impaired myocardial autonomic control. Active compared with inactive patients with SCI have significantly elevated aerobic capacity and stroke volumes. Improvements in aerobic capacity and cardiac function are reported after performing arm crank ergometry for 40 minutes, 3 times a week, at 70% of peak aerobic capacity.⁸¹ However, aerobic capacity achieved with arm ergometry may be up to 40% lower than aerobic capacity with leg exercise. Leg functional electric stimulation (FES), usually recumbent cycling, in combination with arm ergometry augments aerobic capacity.⁸² FES walking and FES rowing also improve aerobic capacity.⁸³ FES-enhanced exercise programs improve myocardial function, insulin sensitivity, and lipid profiles.

Primary prevention is important in the reduction of IHD in men and women. The Nurses' Health Study⁸⁴ identified smoking as the most important IHD risk factor. Smoking 15 or more cigarettes

daily has a relative risk of 5.48 for IHD. Smoking contributes to 41% of coronary events. After 1 year of smoking cessation, risk for IHD is reduced 50%. Age, ethnicity, income, and education affect success of smoking cessation. Among those with 9 to 11 years of education, 39.1% of women smokers quit. Among the smokers with more than 16 years of education, 66.2% quit. Women are less successful at smoking cessation, and concern about weight gain is a frequent reason for relapse.⁸⁵ Combination risk factor modification is essential. If women optimally reduce all risk factors, relative risk for IHD declines to 0.17, and potentially 82% of all cardiac events are preventable.

Individual and group counseling increase success of smoking cessation. A problem-solving approach with social support is beneficial. Cognitive behavioral intervention for women hospitalized with IHD has produced a 50% quit rate at 3 years.⁸⁶ There are 4 approved methods of nicotine replacement (gum, inhaler, nasal spray, patch) and 1 psychotropic drug, bupropion, to treat nicotine addiction.⁸⁷ Nicotine overdose has been reported in those who continue to smoke while using nicotine replacement.

Dietary advice is essential for reducing IHD risk, focusing on reducing saturated fat, total fat, and overall calories. A BMI less than 25kg/m² is ideal. Screening for lipid abnormalities and diabetes is also important, because adequate pharmacologic management can prevent atherosclerosis. Blood pressure monitoring is simple and effective to identify those at risk of IHD. As well as physical activity and medications, treatment for hypertension includes dietary modification and weight loss.

Aspirin is effective in prevention of cardiac events in those at risk of IHD. However, for people at low (1%–3%) 5-year risk, increased incidence of hemorrhagic stroke and gastrointestinal bleeding outweighed IHD benefits.⁸⁸

Physical activity significantly improves most risk factors for IHD. The American Heart Association recommends 20 to 60 minutes of aerobic exercise at moderate intensity on most days of the week. This is sufficient to produce cardiovascular aerobic conditioning, weight reduction, and improvements in lipid and glucose profiles and to lower blood pressure.

1.13 Educational Activity: To provide counseling to a 50-year-old woman with newly diagnosed IHD who has an interest in pursuing complementary and alternative medicine (including Yoga, Tai Chi, and Chinese herbal medications) for the treatment of her cardiac disease.

As complementary and alternative medicine becomes more popular, clinicians are frequently faced with patients' questions about it. Its integration with modern medical techniques is poorly understood by lay people at this time. Discussing these issues gives the clinician a chance to educate and possibly intervene in the prevention of unwanted side effects. The efficacy of most of the traditional herbal remedies has not been verified, and the potential for serious side effects exists. Interactions among medications, such as warfarin, digoxin, and herbal agents have been documented. Examples of beneficial effects of supplements include lowering lipids with fish oil supplementation (ω -3 fatty acids) and the beneficial effects of coenzyme Q-10, polyunsaturated fatty acids, and carnitine on lipid metabolism.⁸⁹⁻⁹¹ The role of antioxidants has not yet been clarified. Negative effects of herbal treatments are mostly anecdotal but can include arrhythmias, arteritis, cardiac glycoside overdose, chest pain, CHF, hypertension, hypotension, MI, over-anticoagulation, pericarditis, and death. Agents that have been implicated in these adverse effects include aconite, ephedra, and licorice. Only with rigorous study can the full picture of the benefits and complications of complementary and alternative medicine be known. An open discussion with patients about potential risks

and a review of what is known about the specific agents that they wish to take may enlighten both the physician and the patient.

Nontraditional exercise and relaxation techniques may also have a role in the care of people with cardiac disease. Once again, the literature supporting these interventions is limited and mostly anecdotal. Tai Chi has been safely done in small groups of patients with CHF, IHD, and after CABG. The benefits listed are decreased stress, improved balance and gait, increased endurance, and improved strength. However, because the studies had limited numbers of participants, further study is needed. Finally, incorporation of Yoga, Tai Chi, and acupuncture as a part of a comprehensive rehabilitation program is likely to be beneficial without clear side effects.^{92,93}

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