



Rehabilitation strategies for the complex cardiac patient

FREDRIC J. PASHKOW, MD

■ Carefully designed, monitored rehabilitation regimens can benefit patients with significant cardiac disease, such as life-threatening arrhythmias or congestive heart failure, or who have concurrent systemic disease such as diabetes. Patients with heart failure can tolerate minimal workloads but, with conditioning, they can increase their duration of exercise. Heart transplant recipients, who are usually severely deconditioned at the time of surgery, are good candidates for a comprehensive rehabilitation program; some have progressed to competition-level athletic achievements. Rehabilitation is safe for patients with arrhythmias, given appropriate monitoring, and can contribute to enhanced quality of life. Objective measures are needed to distinguish between symptomatic and functional improvement.

□ INDEX TERM: CARDIAC REHABILITATION □ CLEVE CLIN J MED 1991; 58:70-75

CARDIAC rehabilitation is increasingly an option for a growing population of patients with significant cardiac problems—including patients with heart failure, transplant recipients, and patients with life-threatening arrhythmias (Table 1). State-of-the-art programs can accommodate the special requirements of these patients, who need close supervision and monitoring.¹

Two important studies^{2,3} show significantly reduced mortality rates among patients randomized to cardiac rehabilitation, compared to those who received “usual” care; these studies included higher risk patients. Overall mortality was reduced by 20% to 25% in the rehabilitation groups, and mortality associated with sudden car-

diac death in the first year post-myocardial infarction was reduced by 35% to 43%. After the first year, the mortality rate in the rehabilitation group began to approximate that of patients receiving usual care. This implies that the effects of the 8- to 12-week rehabilitative period are not indefinite; the early efficacy probably reflects the intense supervision in this period.

SEVERE LEFT VENTRICULAR DYSFUNCTION

Aggressive cardiac care has contributed to the survival of many patients with severe left ventricular (LV) dysfunction, or resting left ventricular ejection fraction (LVEF) less than 30%. This group of patients is one of the fastest growing in the cardiac rehabilitation patient population.

These patients present a different set of complications and expectations than, for example, post-coronary bypass patients who have well-preserved LV function, and may realize different outcomes from cardiac rehabilitation. They are at higher risk of sudden

From the Department of Cardiology, The Cleveland Clinic Foundation.

Address reprint requests to F.J.P., Medical Director of Cardiac Rehabilitation, Department of Cardiology/Desk F15, The Cleveland Clinic Foundation, One Clinic Center, 9500 Euclid Avenue, Cleveland, Ohio 44195.

TABLE 1
INDICATIONS FOR REFERRAL TO MONITORED
REHABILITATION PROGRAMS

Presence of active ischemia, or an acute ischemic event occurring within the last 6 weeks
Significant left ventricular dysfunction, chronotropic incompetence, history of sustained ventricular tachycardia, episode of sudden cardiac death
Patients with implanted devices where monitoring is needed to demonstrate efficacy and safety
Significant concurrent medical problems such as unstable diabetes mellitus, pulmonary insufficiency, or renal disease that requires dialysis
Significant psychosocial problems such as depression or noncompliance

death^{4,5} and may be emotionally depressed by the long-standing ordeal of their cardiac disease and their marked limitation.⁶

Inconsistent responses

Patients with poor LV function show an inconsistent response to exercise.^{7,8} Limited exercise capacity is probably the earliest quantifiable finding in heart failure,⁹ and may actually represent functional impairment.¹⁰ The normal central and peripheral effects of exercise may not occur in these patients.¹¹ At-rest pulmonary capillary wedge pressure (PCWP) and heart rate are often elevated, and minimal effort may cause dyspnea and fatigue.¹²

In patients with severe LV dysfunction, exercise can cause an additional drop in ejection fraction, a decrease in stroke volume, and exertional hypotension. Those with the most severe dysfunction may not elevate cardiac output sufficiently to generate a dynamic exercise response.¹³ Yet, among patients with clinically evident heart failure, these hemodynamic alterations do not correlate consistently with overall exercise capacity.¹⁴ Other factors influence the status of left ventricular function as well as exercise duration and intensity. The patient may be in atrial fibrillation, for example, or have significant active ischemia. He may have forgotten to take prescribed medications, or he may be salt overloaded, dehydrated, or heat intolerant. A concurrent infection may affect LV function and exercise tolerance.

Despite the inconsistent response, exercise has theoretical as well as documented benefits in heart failure patients.^{13,15} Patients with severe LV dysfunction can exercise safely by gradually raising their heart rates above resting level.¹⁶ With time, they are able to extract more oxygen from the blood during exercise, widening the arteriovenous oxygen difference.

The effects of exercise training on mortality for

these patients is unknown, but exercise tolerance is improved as illustrated by lower heart rates during submaximal exercise and increased maximal workloads.¹⁷ Endurance and fatigue levels must be considered in addition to aerobic capacity. Heart failure patients may be able to achieve high aerobic workloads, but then experience prolonged fatigue for hours or days following an exercise session.¹³ All of these considerations require careful program design and precautions.^{1,16}

Rehabilitation for heart failure patients

Cardiac rehabilitation helps heart failure patients accomplish significant gains in functional capacity¹⁸ and may substantially enhance the quality of their lives.¹ The ability to sustain activity at a low metabolic rate of oxygen consumption (MET) level may mean the difference between living independently and living in a chronic care facility.¹⁹

Before a heart failure patient undertakes a rehabilitation program, a thorough pre-training evaluation is critical, both to assess the patient's condition and to aid in the design of a program. In addition to the usual graded exercise study, special tests may be indicated, such as cardiopulmonary studies, measurements of PCWP during exercise, or measurements of LVEF by gated pool scanning or echocardiography during exercise. A patient who has unstable angina, decompensated heart failure, or arrhythmias that compromise hemodynamic stability is not a candidate for exercise rehabilitation.

The rehabilitation program should be designed to accommodate the patient's needs and capacities. Patients with left ventricular dysfunction can tolerate only limited workloads; but, because they can increase their duration of exercise, prolonged periods of warm-up and cool-down are appropriate. Dynamic muscle-strengthening exercises are preferable to isometrics.

The patient's target heart rate should be adjusted to 10 bpm below any significant endpoints of exercise observed in the pre-training evaluation, such as exertional hypotension, significant dyspnea, or the onset of sustained arrhythmia.

These patients need continuous supervision by staff trained to recognize the special problems associated with this population. Patients with severe LV dysfunction should be on continuous telemetry throughout warm-up, exercise, and cool-down. Data such as body weight, blood pressure, and rate response to exercise, measured routinely, are especially valuable in tracking the clinical status of these patients.

CARDIAC TRANSPLANT PATIENTS

Most transplant patients are severely deconditioned after years of low-level activity due to advanced cardiac failure.²⁰ They all require comprehensive psychological, educational, and exercise rehabilitation.^{6,21}

Cardiac output is a function of the strength of muscle contraction (inotropy) and heart rate (chronotropy), as well as the net peripheral vascular and metabolic response to exercise. A normally innervated heart is stimulated by the cardiac plexus of the sympathetic nervous system that increases inotropy and chronotropy. The catecholamines, including epinephrine and norepinephrine, hormonally cause similar increases in cardiac output.

In the transplanted heart, normal cardiac innervation is surgically interrupted. The hormonal mechanism becomes the primary mediator of hemodynamic responses.²⁰ After denervation, the donor heart becomes supersensitive to catecholamines, and the resting heart rate increases. Surgical interruption of the vagus nerve from the donor sinus node also contributes to an increased resting heart rate. Cardiac output, oxygen consumption, and physical work capacity, however, are lower in the transplant patient.²²

The transplant recipient has a characteristic electrocardiogram (ECG). Two distinct P waves may be seen, since the recipient sinoatrial node is generally left intact and may be functioning. This remnant sinoatrial node remains under the control of the recipient's intact vagus nerve. It stimulates the remaining atrial tissue, but the transmitted signal is interrupted at the line where the recipient and donor atria are joined. The donor P wave is also present and is normally conducted to the ventricles. The donor P wave is not influenced by the intrinsic (recipient) cardiac innervation and vagus nerve.

During exercise, anxiety, or other sympathetic stimulation, the rate of the recipient-generated P wave will increase in accordance with the intensity of the sympathetic stimulation. The donor sinus node responds slowly, or not at all, depending on the levels of circulating catecholamines. The net result is a recipient-generated P wave with no relationship to the donor-generated P wave and its associated QRS complex.

Transplant patients can demonstrate significant improvement with exercise conditioning,^{23,24} and there have been several reports of competition-level athletic accomplishment.²⁵ Moreover, frequent graded exercise testing may help detect early signs of rejection by the

development of an otherwise unexplained fall in exercise tolerance. Emotional lability is common in heart transplant patients, which may complicate the rehabilitation process, but many are eager to learn and to overcome their physical difficulties.

Despite the benefits, there is potential for several serious problems. For example, it was hoped that rehabilitation would lead to a decline in the incidence of accelerated graft atherosclerosis (AGAS). AGAS is the leading cause of death among cardiac transplant patients after the first year, and may be more related to the use of immunosuppressive agents than to traditional coronary risk factors.²⁶ While the cause of the atherogenic process is incompletely understood, reducing secondary risk factors such as smoking and cholesterol seems appropriate.

Another problem is that only one-third of transplant patients return to work, despite their relatively young age.²⁷ One factor may be the time away from the job prior to transplant surgery. This speculation is based on observations of return-to-work experience in patients after coronary bypass surgery and valve replacement.²⁴ If preoperative time away from work is a factor, the problems of early identification of patients as transplant candidates and the protracted waiting periods for donor hearts must be addressed. Rehabilitation can help here.

CARDIAC ARRHYTHMIAS

The risk of cardiac arrest during rehabilitation exercise is very low. From 1980 to 1984, one arrest per 112,000 patient hours of exercise was reported.²⁸ The incidence from 1960 to 1977 was one arrest per 33,000 patient hours.²⁹ This trend toward fewer cardiac arrests reflects the improved medical and surgical treatment of ischemia, a precipitant of many ventricular arrhythmias associated with exercise.³⁰

Monitoring guidelines

Standards and guidelines are evolving in the United States for continuous *v* intermittent ECG monitoring during exercise rehabilitation.³¹ The financial reimbursement system has indirectly encouraged monitoring,³² but this is likely to change. Furthermore, since the risk of death during cardiac rehabilitation has significantly decreased,²⁸ program guidelines may limit continuous ECG monitoring to those patients who are at high risk. This includes patients who have had an acute event within 6 weeks, or who have active ischemia, significant left ventricular dysfunction, or a history of

sustained ventricular tachycardia. Patients who have had episodes of sudden cardiac death and are not yet stabilized on therapy should also be continuously monitored.

Implantable cardioverter defibrillators

Drugs for the suppression of lethal or potentially lethal arrhythmias have proven inadequate in many patients, whereas an implantable device that uses direct current shocks has had surprising success. The implantable cardioverter defibrillator,^{33,34} initially limited to patients with histories of sudden death,^{35,36} is now used in individuals who have susceptible arrhythmia substrates, such as an extensive prior myocardial infarction, and in whom sustained ventricular tachycardia can be induced in the electrophysiologic laboratory.³⁷

Most of the algorithms that control these devices are based on rate dependency; therefore the patient must undergo exercise stress testing to ensure that his intrinsic rate will not exceed the device's threshold rate, which would cause it to discharge unnecessarily during physical activity. This is unlikely to occur, since many patients are receiving concomitant drug therapy, but testing is necessary nevertheless. Most patients are aware that they may be shocked by the device during exercise and are appropriately apprehensive.³⁸

These patients are excellent candidates for monitored programs, at least until the likelihood of inadvertent defibrillation has been ruled out with sufficient experience. They also benefit from the group support and the socialization in a monitored program.³⁸ The implantation of such a potent device into an individual can have significant psychological ramifications. Formal group psychotherapy can be offered to those who are identified as having significant adjustment problems.³⁸ The opportunity to share experiences is, for many, a significant relief.

The staff's knowledge of how the device can be temporarily turned off (usually by magnet), together with an understanding of the underlying disease process and thorough preparation for the management of emergencies leads to an air of confidence that is very reassuring to these vulnerable patients.

Cardiac pacemakers

About 20% of patients in cardiac rehabilitation programs have pacemakers. The number of patients with rate incompetence is increasing and the indications for bradycardia pacing are changing.³⁹ At one time, the rehabilitation of a patient with an implanted

pacemaker simply involved reassurance and education about the device. The patient was cautioned to avoid high-level physical activity since the pacemaker's fixed rate could not respond to exertion.¹⁹

There has been enormous progress in the design and manufacture of cardiac pacing devices. The changes relevant to the rehabilitation of severely disabled patients result from the miniaturization of electronic components by the use of large-scale integrated circuitry. This allows the emulation of normal rate and rhythm responses under varying physiologic conditions and levels of metabolic demand.⁴⁰

The impact of these devices on functional capacity is significant. Patients who are limited to a sedentary lifestyle may achieve the cardiac output necessary to accomplish most activities of daily living.^{39,41} This increase in work capacity allows for the performance of such essential activities as shopping and food preparation, light housework, or care of an invalid spouse—and may sometimes make the difference between institutionalization and independence.³⁹

The physiologic aspects of exercise in these patients is identical to any other.⁴² What is unique is the way in which their physiology integrates with the implanted devices. The relative contribution of chronotropy to cardiac output is generally appreciated, but the relationship between rate and atrioventricular synchrony during progressive levels of exercise is less well understood. The ability to develop an appropriate heart rate response during exercise accounts for about two-thirds of the cardiac output. Stroke volume, consisting of preload and afterload, accounts for the other third.⁴² As heart rate increases, the relative contribution from atrioventricular synchrony decreases.⁴²

Pacemakers are now available that provide atrioventricular synchrony as well as dynamic adjustment of the heart rate to match varying levels of metabolic demand.^{40,43,44} Cardiac synchrony is maintained by the placement of two pacing leads, one in the atria, and a second in the ventricle. A rate-adaptive pacer uses a parameter or physiologic variable other than the natural ones to drive one's sinus node to control the pacing rate.⁴⁵

HEART VALVE REPLACEMENT

Physical training after heart valve replacement improves cardiorespiratory fitness as evidenced by improved oxygen consumption.⁴⁶ Training also increases physical work capacity by 60% and decreases rate-pressure product and perceived exertion by about 15%.⁴⁷

Physical fitness alone does not guarantee a return to work, however. Age and time away from the job before operation are also important factors.²⁴ Many patients require anticoagulation therapy after valve surgery and have special requirements for low-impact exercise (to avoid bruising or hemarthrosis) and special educational needs; eg, to avoid risk of falls associated with climbing ladders or riding motorcycles or horses.

SERIOUS COEXISTING SYSTEMIC DISEASE

Chronic pulmonary disease

Patients with advanced chronic obstructive pulmonary disease can benefit from rehabilitation programs.^{48,49} Patients with combined pulmonary and cardiac disease should be referred to the appropriate rehabilitation program, depending on which problem (cardiac or pulmonary) is more limiting and on what is available in the community. When these patients are followed in the cardiac setting, provisions should be made to noninvasively monitor oxygen saturation. Pulse oximetry with a compact portable device can be helpful to monitor exercise-induced hypoxia. These patients invariably are taking xanthine drugs, such as theophylline, and other adrenergic agents with narrow margins of therapeutic safety. Tachyarrhythmias are common in this patient population, so continuous electrocardiographic monitoring is indicated.

Diabetes

There is significant potential for risk factor modification for diabetic patients with concurrent heart disease.⁵⁰ In addition to its positive conditioning

effects,⁵¹ exercise may reduce the insulin requirement.⁵² Patients with unstable diabetes require carefully designed exercise regimens. The exercise must be consistent so that caloric consumption can be balanced.⁵³ Other considerations include the management of hypoglycemia that may occur during exercise, and the prevention and management of exercise-related foot problems.¹

Renal disease

Cardiovascular disease is a common concurrent disorder in patients with advanced renal disease. Uremic patients on dialysis have a higher cardiovascular death rate than nonuremic patients with equivalent lipid abnormalities.⁵⁴ Rehabilitation programs that include exercise have been successful for these patients.⁵⁵ In addition to the benefits of conditioning, programs for the renal patient have effectively modified important risk factors. For example, in one group studied, 12 months of exercise resulted in a 23% fall in triglycerides, a 21% increase in high-density lipoprotein cholesterol, and an 18% decrease in glucose.⁵⁶

NEED FOR OBJECTIVE MEASURES

Reports of functional improvement with cardiac rehabilitation are largely subjective. Valid, objective measures of functional status are rarely used in clinical practice, and those that are used are often ambiguous.⁵¹ The measures need to discriminate between symptomatic status and functional performance, and their application will require more attention and commitment from investigators, providers, and regulators.

REFERENCES

- Pashkow FJ. Complicating conditions. In: Pashkow FJ, Pashkow P, Schafer M, eds. *Successful Cardiac Rehabilitation: The Complete Guide for Building Cardiac Rehab Programs*. Loveland, Colo: Heart Watchers Press; 1988; 228–247.
- O'Connor GT, Buring JE, Yusuf S, et al. An overview of randomized trials of rehabilitation with exercise after myocardial infarction. *Circulation* 1989; 80:234–244.
- Oldridge NB, Guyatt GH, Fischer ME, Rimm AA. Cardiac rehabilitation after myocardial infarction: combined experience of randomized clinical trials. *JAMA* 1988; 260:945–950.
- Kannel WB, Plehn JF, Cupples LA. Cardiac failure and sudden death in the Framingham Study. *Am Heart J* 1988; 115:869–875.
- Packer M. Sudden unexpected death in patients with congestive heart failure: a second frontier. *Circulation* 1985; 72:681–685.
- Christopherson LK. Cardiac transplantation: a psychological perspective. *Circulation* 1987; 75:57–62.
- Hoffman A, Duba, J, Lengyel M, et al. The effect of training on the physical working capacity of MI patients with left ventricular dysfunction. *Eur Heart J* 1987; 8:43–49.
- Sullivan MJ, Higginbotham MB, Cobb FR. Exercise training in patients with severe left ventricular dysfunction. *Circulation* 1988; 78:506–515.
- Jennings GL, Esler MD. Circulatory regulation at rest and exercise and the functional assessment of patients with congestive heart failure. *Circulation* 1990; 81(Suppl II):II-5–II-13.
- Lipkin DP, Poole-Wilson PA. Symptoms limiting exercise in chronic heart failure. *Br Med J* 1986; 292:1030–1031.
- Arvan S. Exercise performance of the high risk acute myocardial infarction patient after cardiac rehabilitation. *Am J Cardiol* 1988; 62:197–201.
- Reddy HK, Weber KT, Janicki JS, McElroy PA. Hemodynamic, ventilatory and metabolic effects of light isometric exercise in patients with chronic heart failure. *J Am Coll Cardiol* 1988; 12:353–358.

13. Dubach P, Froelicher VF. Cardiac rehabilitation for heart failure patients. *Cardiology* 1989; **76**:368-373.
14. McKirman MD, Sullivan M, Jensen D, et al. Treadmill performance and cardiac function in selected patients with coronary heart disease. *J Am Coll Cardiol* 1984; **3**:253-261.
15. Shabetai R. Beneficial effects of exercise training in compensated heart failure. *Circulation* 1988; **78**:775-776.
16. Mathes P. Physical training in patients with ventricular dysfunction: choice and dosage of physical exercise in patients with pump dysfunction. *Eur Heart J* 1988; **9**(Suppl F):67-69.
17. Lee AP, Ice R, Blessey R, et al. Long-term effects of physical training in coronary patients with impaired ventricular function. *Circulation* 1979; **60**:1519.
18. Wenger NK. Left ventricular dysfunction, exercise capacity and activity recommendations. *Eur Heart J* 1988; **9**(Suppl F):63-66.
19. Wenger NK. Future directions in cardiovascular rehabilitation. *J Cardiopulmonary Rehabil* 1987; **7**:168-174.
20. Fink A. Exercise responses in the transplant population. *Cardiopulmonary Record* 1986; **1**(3):7-10.
21. O'Brien V. Psychological and social aspects of heart transplantation. *Heart Transplantation* 1985; **4**:229-231.
22. Arthur E. Rehabilitation of potential and cardiac transplant patients. *Cardiopulmonary Record* 1986; **1**(3):11-13.
23. Kavanagh T, Yacoub MH, Mertens DJ, Kennedy J, Campbell RB, Sawyer P. Cardiorespiratory responses to exercise training after orthotopic cardiac transplant. *Circulation* 1988; **77**:162-171.
24. Roos R. Exercise training for heart transplant patients. *The Physician and Sports Medicine* 1986; **14**(9):165-174.
25. Kavanagh T, Yacoub MH, Campbell R, Mertens D. Marathon running after cardiac transplantation: a case history. *J Cardiac Rehab* 1986; **6**:16-20.
26. Billingham ME. Graft coronary disease: the lesions and the patients. *Transplant Proc* 1989; **21**:3665-3666.
27. Meister ND, McAleer MJ, Meister JS, Riley JE, Copeland JG. Returning to work after heart transplantation. *J Heart Transplant* 1986; **5**:154-161.
28. Van Camp S, Peterson R. Cardiovascular complications of outpatient cardiac rehabilitation programs. *JAMA* 1986; **256**:1160-1163.
29. Haskell W. Cardiovascular complications during exercise training of cardiac patients. *Circulation* 1978; **57**:920-924.
30. Falcone C, de Servi S, Poma E, et al. Clinical significance of exercise-induced silent myocardial ischemia in patients with coronary artery disease. *J Am Coll Cardiol* 1987; **9**:295-299.
31. Health and Public Policy Committee, American College of Physicians. Cardiac rehabilitation services. *Ann Intern Med* 1988; **109**:671-673.
32. Hossack K, Hartwig R. Cardiac arrest associated with supervised cardiac rehab. *J Cardiac Rehab* 1982; **2**:402-408.
33. Mirowski M. The automatic implantable cardioverter defibrillator: an overview. *J Am Coll Cardiol* 1985; **6**:461-466.
34. Mirowski M, Mower MM, Reid PR, Watkins L, Langer A. The automatic implantable defibrillator: new modality for treatment of life-threatening ventricular arrhythmias. *PACE* 1982; **5**:384-400.
35. Echt DS, Armstrong K, Schmidt P, Dyer PE, Stinson EB, Winkle RA. Clinical experience, complications, and survival in 70 patients with the automatic implantable cardioverter defibrillator. *Circulation* 1985; **71**:289-296.
36. Kelly PA, Cannon DS, Garan H, et al. Implantable cardioverter defibrillator: efficacy, complications, and survival in patients with malignant ventricular arrhythmias. *J Am Coll Cardiol* 1988; **11**:1278-1286.
37. Winkle RA, Mead RH, Ruder MA, et al. Long-term outcome with the automatic implantable cardioverter-defibrillator. *J Am Coll Cardiol* 1989; **13**:1353-1361.
38. Pycha C, Gullede AD, Hutzler J, Kadri N, Maloney JD. Psychological responses to the implantable defibrillator. *Psychosomatics* 1986; **27**:841-845.
39. Pashkow F. Rate responsive pacing: practical applications. *Cardio* 1989; **6**(6):89-99.
40. Rickards A. Rate-Responsive Pacing. In: Barold S, ed. *Modern Cardiac Pacing*. Mt. Kisco, NY: Futura Publishing Co; 1985: 799-809.
41. Rickards AF, Donaldson RM. Rate-responsive pacing. *Clin Prog Pacing Electrophysiol* 1983; **1**:12.
42. Kristensson BE, Rydin L, Amman K, et al. The haemodynamic importance of atrioventricular synchrony and rate increase at rest and during exercise. *Eur Heart J* 1985; **6**:773-778.
43. Humen DP, Kostuk WJ, Klein GJ. Activity sensing, rate-responsive pacing: improvement in myocardial performance with exercise. *PACE* 1985; **8**:52-59.
44. Nordlander R, Hedman A, Pehrsson SK. Rate-responsive exercise capacity: a comment. *PACE* 1989; **12**:749.
45. Faerstrand S, Breivik K, Ohm DJ. Assessment of the work capacity and relationship between rate response and exercise tolerance associated with activity-sensing rate-responsive ventricular pacing. *PACE* 1987; **10**:1277.
46. Newell JP, Kappagoda CT, Stoker JB, Deverall PB, Watson DA, Linden RJ. Physical training after heart valve replacement. *Br Heart J* 1980; **44**:638-649.
47. Sire S. Physical training and occupational rehabilitation after aortic valve replacement. *Eur Heart J* 1987; **8**:1215-1220.
48. Berman LB. Exercise for the pulmonary patient. *J Cardiopul Rehab* 1986; **6**:52-61.
49. Casaburi R, Wasserman K. Exercise training in pulmonary rehabilitation. *N Engl J Med* 1985; **314**:1509-1511.
50. Niederhauser HU, Oesch W, Keel A, Stettbacher N. Can factors of prognostic significance be identified during rehabilitation of cardiac infarct patients? *Schweiz Med Wochenschr* 1984; **114**:1751-1756.
51. Tedesco C, Manning S, Lindsay R, Alexander C, Owen R, Smucher ML. Functional assessment of elderly patients after percutaneous aortic balloon valvuloplasty: NYHA classification v functional status questionnaire. *Heart Lung* 1990; **19**:118-125.
52. Koivisto V, Sherwin R. Exercise in diabetes: therapeutic implications. *Postgrad Med* 1979; **66**:87-96.
53. Richter EA, Rudermann NB, Schneider SH. Diabetes and exercise. *Am J Med* 1981; **70**:201-209.
54. Lazarus JM. Cardiovascular disease in uremic patients on hemodialysis. *Kidney Int* 1975; **2**(Suppl):S167-S175.
55. Painter PL. Exercise in end-stage renal disease. *Exerc Sport Sci Rev* 1988; **16**:305-339.
56. Goldberg AP, Geltman EM, Gavin JR, et al. Exercise training reduces coronary risk and effectively rehabilitates hemodialysis patients. *Nephron* 1986; **42**:311-316.