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Impact of Cardiac Rehabilitation on the Exercise Function of Children With Serious Congenital Heart Disease

Jonathan Rhodes, MD; Tracy J. Curran, MS; Laurel Camil, MS; Nicole Rabideau, MS; David R. Fulton, MD; Naomi S. Gauthier, MD; Kimberlee Gauvreau, ScD; and Kathy J. Jenkins, MD, MPH

ABSTRACT. *Objectives.* **The exercise capacity of children with congenital heart disease (CHD) is often depressed. This depression is thought to be attributable to (1) residual hemodynamic defects and (2) deconditioning secondary to physical inactivity. We hypothesized that this latter component would be ameliorated by a formal cardiac rehabilitation program designed specifically for children. The objective of this study was to characterize the effect of a cardiac rehabilitation program on the exercise performance of children with CHD and to define the physiologic mechanisms that might account for any improvements that are observed.**

Methods. **Nineteen patients with CHD who were referred for exercise testing and found to have a peak oxygen consumption (VO_2) and/or peak work rate <80% of predicted were enrolled in the study. Sixteen patients (11 Fontan patients, 5 with other CHD) completed the program and had postrehabilitation exercise tests, results of which were compared with the prerehabilitation studies.**

Results. **Improvements were found in 15 of 16 patients. Peak VO_2 rose from 26.4 ± 9.1 to 30.7 ± 9.2 mL/kg per min; peak work rate from 93 ± 32 to 106 ± 34 W, and the ventilatory anaerobic threshold from 14.2 ± 4.8 to 17.4 ± 4.5 mL/kg per min. The peak heart rate and peak respiratory exchange ratio did not change, suggesting that the improvements were not attributable merely to an increased effort. In contrast, the peak oxygen pulse rose significantly, from 7.6 ± 2.8 to 9.7 ± 4.1 mL/beat, an improvement that can be attributed only to an increase in stroke volume and/or oxygen extraction at peak exercise. No patient experienced rehabilitation-related complications.**

Conclusion. **Cardiac rehabilitation can improve the exercise performance of children with CHD. This improvement is mediated by an increase in stroke volume and/or oxygen extraction during exercise. Routine use of formal cardiac rehabilitation may greatly reduce the morbidity of complex CHD.** ***Pediatrics*** **2005;116:1339–1345;** ***congenital heart defects, exercise, cardiac rehabilitation.***

ABBREVIATIONS. CHD, congenital heart disease; VO_2, oxygen consumption; VAT, ventilatory anaerobic threshold; $\Delta VE/\Delta VCO_2$, slope of the minute ventilation versus carbon dioxide production relationship below the respiratory compensation point; MVV, maximal voluntary ventilation; FEV1, the forced expiratory volume in the first second; RER, the ratio of carbon dioxide production over oxygen consumption; FVC, forced vital capacity.

From the Department of Pediatric Cardiology, Children's Hospital, Boston, Massachusetts.

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Reprint requests to (J.R.) Department of Cardiology, 300 Longwood Ave, Boston, MA 02115. E-mail: jonathan.rhodes@cardio.chboston.org

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Over the past 2 decades, medical and surgical advances have dramatically increased the survival rates of patients with congenital heart disease (CHD). Survival into adulthood is now expected for even the most complex malformations. However, despite excellent surgical outcomes, the exercise capacity of children who have had heart surgery is often depressed.[1–14] Undoubtedly, residual hemodynamic defects are partly responsible for the poor exercise capacity. However, we believe that many of these children are often (inappropriately) considered to be excessively fragile and may be unduly restricted from participation in physical activities.[15] We suspect that the inactivity and consequent deconditioning that results from the imposed restrictions exacerbate whatever exercise intolerance the children might experience as a result of residual cardiovascular disease. A formal cardiac rehabilitation program might be expected to reverse partially the effects of inactivity and deconditioning and thereby improve exercise function, as well as allow children to initiate exercise in a safe environment.

Data from adult cardiac patients have shown that many benefits may be derived from a supervised cardiac rehabilitation program. These benefits include improvement in lipid profiles, decrease in obesity, and improvement in exercise capacity. Adult cardiac rehabilitation programs have also been shown to produce significant reductions in hospitalization costs and overall cardiac morbidity and mortality.[16,17]

Only limited data are available concerning the effect of cardiac rehabilitation on pediatric patients with CHD. Past studies have included small numbers of patients and have had mixed results. In those studies that have detected an improvement in exercise function, the mechanisms that were responsible for the observed improvements have not been elucidated.[15,18–25] We therefore undertook this study to characterize more clearly the effect of a 12-week pediatric cardiac rehabilitation program on the exercise performance and cardiopulmonary function of children with CHD and to define the physiologic mechanisms that account for any improvements that may be observed.

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METHODS

For this pilot project, we sought to enroll children who we believed would be most likely to benefit from cardiac rehabilitation and for whom exercise would pose a low risk. To accomplish this goal, our entry criteria included (1) age 8 to 17 years; (2) a nontrivial congenital heart defect of severity sufficient to have potentially caused a degree of activity restriction to be imposed on the patient by his or her doctors, parents, teachers, coaches, etc; consequently, all patients had to have had at least 1 surgical or interventional catheterization procedure and/or have a significant residual hemodynamic defect; (3) abnormal exercise function, characterized by a peak work rate and/or peak oxygen consumption (VO_2) <80% of predicted on a standard progressive bicycle ergometry test that was performed at the Children's Hospital exercise physiology laboratory within the last 6 months; and (4) a commitment to attend and participate reliably in rehabilitation sessions.

Out of concern for the safety of the patients, those who developed during their exercise tests abnormalities (eg, exercise-induced arrhythmias, ST depression, hypertension, hypotension, cardiac chest pain, or systemic desaturation <80%) that could pose a health risk during exercise were excluded from the study. In addition, patients with the following conditions were excluded:

- Documented life-threatening arrhythmias not palliated by an automatic internal cardiac defibrillator
- Moderate or severe dysfunction of either ventricle (ejection fraction <40% or qualitative)
- Pulmonary artery hypertension >40 mm Hg or requiring treatment with vasodilators
- Acute inflammatory cardiac disease
- Significant coronary artery disease (ie, documented stenoses and/or myocardial ischemia)
- Uncontrolled heart failure (ie, requiring hospitalization and/or intravenous medications within the last month)
- Resting oxygen saturation <90%
- Aortic stenosis; resting peak systolic gradient >50 mm Hg
- Pulmonic stenosis, resting peak systolic gradient >50 mm Hg
- Severe systemic atrioventricular valve regurgitation
- Systemic hypertension (>95th percentile for age)
- Acute renal disease
- Acute hepatitis

Recruitment

During the 6 months before the initiation of the rehabilitation program, 188 patients aged 6 to 17 years completed exercise tests with expiratory gas analysis in our laboratory. Of these, 71 did not have a congenital heart defect of severity sufficient to be included in the study, and 33 were excluded because they had a peak VO_2 and peak work rate >80% predicted. An additional 16 were excluded on account of 1 or more of the exclusion criteria. The remaining 68 patients were contacted by mail and asked to participate in the study. Nineteen of these patients agreed to participate in the study; geographic factors (ie, living a great distance from the facility in which the rehabilitation program was located) were the primary reasons that precluded the participation of the other 49 patients. The clinical characteristics of the respondents who did participate were similar to those of the respondents who did not participate. Three patients elected to withdraw from the program within the first 3 weeks. The remaining 16 patients completed the 12-week program and are the subject of this report. All patients were at least 6 months post their last surgical or interventional catheterization procedure, and no patient had an additional procedure during the course of the study.

This study was approved by the Committee on Clinical Investigation at Children's Hospital. All parents signed consent forms, and all children gave their assent before enrolling in the study.

Rehabilitation Program

Rehabilitation sessions were conducted for 1 hour twice a week for 12 weeks. They were conducted in a 30′ × 15′-room at a suburban satellite clinic facility. The sessions were staffed by a pediatric cardiologist (J.R.) and 1 or 2 exercise physiologists (T.J.C., L.C., and N.R.) who led the sessions, participated in the activities, and monitored the patients. The patients were divided by age into 2 groups (8- to 13-year-olds and 13- to 17-year-olds). Each session began with 5 to 10 minutes of stretching exercises, followed by ~45 minutes of aerobic and light weight/resistance exercises. Activities included aerobic dance, step aerobics, calisthenics (sit-ups, crunches, jumping jacks, push-ups, etc), kick boxing, and jumping rope. When the weather permitted, outdoor games such as capture the flag and relay races were conducted. Resistance exercises were performed with 3- and 5-lb barbells, light elastic bands, and cords. Games, rubber balls, music, and simple, age-appropriate prizes (eg, baseball cards) were incorporated into the activities to promote enthusiasm and motivation. Attempts were also made to vary activities and to accommodate to the moment-to-moment desires of the patients to optimize participation and interest. The last 5 to 10 minutes of each session were devoted to cooling down and stretching. Patients were also encouraged to exercise at home on at least 2 additional occasions per week. This message was reinforced at each rehabilitation session, but a specific home exercise program was not prescribed and compliance was not monitored.

Heart rate was checked (manually) at the start of each session and on 2 or 3 additional occasions during each session. The patients were encouraged to exercise at an intensity sufficient to raise their heart rates to levels equal to that associated with the ventilatory anaerobic threshold (VAT; as determined on their baseline exercise tests). At each session, the patients also rated the intensity of exercise using the Borg rate of perceived exertion score.[26] As a safety precaution, oxygen, a pulse oximeter, and an external defibrillator were available at each rehabilitation session. At the last rehabilitation session, all participants were awarded a certificate of attendance and a pedometer.

Exercise Tests

The patients performed progressive, symptom-limited exercise tests using a stationary, upright cycle ergometer with a continuous graded workload adjusted for each patient according to the methods of Wasserman et al.[27] Electrocardiographic monitoring and breath-by-breath expiratory gas analysis were performed with a Medical Graphics exercise testing system (Medical Graphics Corp, St Paul, MN). As mentioned previously, all patients had undergone exercise testing as part of clinical care, within 6 months before the start of the rehabilitation program. Within 2 weeks of the end of the 12-week rehabilitation program, all patients performed a second progressive bicycle ergometry exercise test with expiratory gas analysis, using a ramp rate identical to that used for the prerehabilitation test. A monetary award was issued to each patient at the time of the postrehabilitation exercise test.

Predicted values were calculated on the basis of regression equations that take into account age, gender, and size.[28] The VAT was determined by the V-slope method.[29] The slope of the minute ventilation versus carbon dioxide production relationship below the respiratory compensation point ($\Delta VE/\Delta VCO_2$) was calculated as previously described.[30]

Spirometric measurements were obtained on every patient before each exercise test. The maximal voluntary ventilation (MVV) was estimated by multiplying the forced expiratory volume in the first second (FEV1) by 40.[31] The breathing reserve was calculated from the following equation: Breathing Reserve (%) = 100 × (MVV − peak VE)/MVV.

Statistical Analysis

Student's paired *t* test was used to compare continuous variables, and McNemar's test was used to compare dichotomous variables before and after rehabilitation. The relationships between indexes of exercise performance and other continuous variables were evaluated using Pearson's correlation coefficient.

RESULTS

The demographic and clinical features of the patients are summarized in Table 1. Eleven of the patients had had Fontan procedures, and all but 3 patients were maintained on at least 1 cardiac medication. On preparticipation questionnaires, 11 of 16 patients indicated that their physical activities were limited on account of concern about their heart problems. The participants attended a mean of 18.2 ± 3.8 (range: 10–22) of the 24 sessions. They achieved their

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TABLE 1. Clinical Characteristics

Age/Gender	Cardiologic Diagnoses	Cardiologic Procedures	Years Since Last Procedure	Cardiac Medications
11/M	Tricuspid atresia, d-transposition	Bidirectional Glenn, Fontan	11	ACEi, Coumadin
12/M	Hypoplastic left heart syndrome	Norwood, bidirectional Glenn, Fontan	4	Aspirin, ACEi
16/M	d-transposition, VSD	Blalock-Taussig shunt, pulmonary artery band, Senning, pacemaker	6	Aspirin, ACEi
8/F	Unbalanced atrioventricular canal defect, hypoplastic LV, coarctation	Coarctation repair, pulmonary artery band, bidirectional Glenn, Stansel, Fontan	5	Digoxin, diuretic, aspirin, ACEi
13/M	Tricuspid atresia, d-transposition	Fontan, resection of subaortic stenosis	2	Aspirin
11/M	Double-outlet RV, pulmonic stenosis	Balloon pulmonary valvuloplasty, transannular repair	10	Digoxin, diuretic
11/F	d-transposition, hypoplastic RV	Blalock-Taussig shunt, bidirectional Glenn, Fontan	3	Aspirin, ACEi
13/M	Double-outlet RV, hypoplastic LV, coarctation	Coarctation repair, pulmonary artery band, coarctation balloon, bidirectional Glenn, Fontan	10	ACEi, Coumadin
13/M	Pulmonary atresia/intact ventricular septum	RV outflow tract patch	13	None
12/M	Hypoplastic left heart syndrome	Norwood, bidirectional Glenn, Fontan	8	Aspirin, ACEi
15/F	Heterotaxy, double outlet RV, hypoplastic LV	Stansel, Blalock-Taussig shunt, Fontan, pacemaker	4	Aspirin, digoxin
10/M	VSD	VSD repair	9	None
10/M	Double-outlet RV, hypoplastic LV	Pulmonary artery band, bidirectional Glenn, Fontan, resection of subaortic stenosis, Fen closure, pacemaker	1	Aspirin, ACEi, sotalol
13/M	Ebstein's, Wolff-Parkinson-White syndrome	Radiofrequency ablation (2 separate procedures)	3	None
13/F	Double-outlet RV, hypoplastic LV	Pulmonary artery band, bidirectional Glenn, tracheal reconstruction, Fontan	3	Aspirin, ACEi, diuretic
13/M	Double-outlet RV, hypoplastic LV	Pulmonary artery band, bidirectional Glenn, Fontan	9	Aspirin, ACEi, diuretic
11.9 ± 2.1; 12M/4F			6.3 ± 3.7	

ACEi indicates angiotensin-converting enzyme inhibitor; LV, left ventricle; RV, right ventricle; VSD, ventricular septal defect.

target heart rates at 92.3 ± 4.1% of the sessions and reported Borg RPE scores of 15.1 ± 3.0. No patient experienced a rehabilitation-related complication during the study. On no occasion was it necessary to use the aforementioned safety equipment.

Effect of the Cardiac Rehabilitation Program on Peak Exercise

Before the rehabilitation program, the patients' peak VO_2, work rate, heart rate, and oxygen pulse (the amount of oxygen consumed per heart beat, an index equal to stroke volume times oxygen extraction[32]) were abnormally low (Table 2). After rehabilitation, there was a significant increase in peak VO_2 and peak work rate. One or both of these parameters increased in 15 of 16 patients (Fig 1). The improved exercise function was not attributable solely to somatic growth, because absolute values as well as weight normalized, and percentage of predicted values for each parameter also increased. Furthermore, the patients' peak respiratory exchange ratio (RER; the ratio of VCO_2 over VO_2; the magnitude of the peak RER roughly reflects the effort expended by the patient at peak exercise.[33,34]) and peak heart rate on the postrehabilitation study did not differ from prerehabilitation values, indicating that the improvements in exercise performance were not attributable to an increased effort. The improvements in exercise function, however, were almost without exception accompanied by a significant increase in peak oxygen pulse (Fig 2).

The peak exercise systolic blood pressure was not affected by cardiac rehabilitation. However, the patients' peak exercise diastolic blood pressure was significantly higher after cardiac rehabilitation.

As would be expected, the increase in peak VO_2 after rehabilitation was associated with comparable increases in peak VCO_2 and VE. The increase in VE was mediated by a rise in the tidal volume at peak exercise (when expressed in liters). When expressed as a percentage of the resting forced vital capacity (FVC), the tidal volume also increased ($P = .06$). The respiratory rate at peak exercise did not differ significantly from prerehabilitation values. The rise in VE at peak exercise resulted in a statistically insignificant decline in the breathing reserve. Moreover, the patients' breathing reserve remained substantial after the rehabilitation program. Cardiac rehabilitation had no effect on peak exercise oxygen saturation.

Improvements in peak exercise function were observed in all 11 Fontan patients. The only patient whose peak exercise function did not improve after the rehabilitation program was an 11-year-old boy with severe pulmonary regurgitation after transan-

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TABLE 2. Effect of Cardiac Rehabilitation on Peak Exercise Parameters

	Before Rehabilitation	After Rehabilitation	% Change	Mean Difference	*P* Value
VO_2, mL/min	1182 ± 422	1439 ± 490	22	257	<.001
VO_2, mL/kg/ per min	26.4 ± 9.1	30.7 ± 9.2	16	4.4	.005
VO_2, % predicted	61.3 ± 15.1	71.6 ± 16.8	17	10.3	.003
VCO_2, mL/kg	29.9 ± 10.8	34.9 ± 11.1	17	4.9	.006
Work rate, W	93 ± 32	106 ± 34	14	13	<.001
Work rate, W/kg	2.09 ± 0.66	2.27 ± 0.69	9	0.19	.01
Work rate, % predicted	63.2 ± 18.2	69.0 ± 19.1	9	5.8	.02
RER	1.13 ± 0.08	1.13 ± 0.04	0	0	NS
HR, bpm	158 ± 24	158 ± 28	0	0	NS
Oxygen pulse, % predicted	76.0 ± 20.1	89.9 ± 22.5	18	13.9	.01
Minute ventilation, L/min	51.8 ± 17.2	63.0 ± 21.3	22	11.3	<.001
Breathing reserve, %	41.6 ± 12.2	36.1 ± 12.6	−13	−5.6	NS
Tidal volume, L	1.04 ± 0.38	1.17 ± 0.41	13	0.14	.004
Tidal volume, % FVC	44.1 ± 9.9	47.3 ± 10.9	7	3.2	.06
Respiratory rate, breaths/min	52.5 ± 15.5	56.6 ± 17.5	8	4.1	NS
Systolic BP, mm Hg	133 ± 16	135 ± 8	2	2	NS
Diastolic BP, mm Hg	63 ± 12	71 ± 8	13	8	.01
Peak O_2 saturation, %	92.0 ± 4.6	91.8 ± 5.1	0	−0.1	NS

BP indicates blood pressure; HR, heart rate; NS not significant; bpm, beats per minute.

nular repair of double-outlet right ventricle with pulmonary stenosis.

Impact of Cardiac Rehabilitation on Submaximal Exercise Function

The effect of cardiac rehabilitation on the patients' cardiopulmonary function at the VAT was similar to that observed at peak exercise. After rehabilitation, the VO_2 and the work rate at the VAT rose significantly. These changes were associated with a concomitant increase in the oxygen pulse; the heart rate at the VAT did not change. Neither the ventilatory equivalents for oxygen and carbon dioxide at the VAT nor the $\Delta VE/\Delta VCO_2$ (indices that reflect the efficiency of gas exchange during exercise) was affected by the rehabilitation program (Table 3).

Low-grade atrial and/or ventricular ectopy developed in 8 of 16 patients during the prerehabilitation exercise test. Ectopy was not detected during the postrehabilitation exercise test in 5 of these patients. None of the patients without ectopy on the prerehabilitation study subsequently developed ectopy on the postrehabilitation study ($P = .06$ by McNemar's test).

Impact of Cardiac Rehabilitation on Measurements at Rest

Between the times of the pre– and post–cardiac rehabilitation exercise tests, small but statistically significant increases in body weight and height were observed. The FVC and FEV1 also increased slightly. These increases were attributed to the patients' somatic growth, as there was no change in the percentage of predicted FVC and FEV1. Statistically significant changes were also not observed in the patients' body mass index, FEV1/FVC, resting oxygen saturation, or blood pressures (Table 4).

Correlates of the Improvement in Peak Exercise Function

The improvement in percentage of predicted peak VO_2 correlated strongly with the improvement in percentage of predicted peak oxygen pulse ($r = 0.75$; $P < .001$). Pre–post rehabilitation changes in peak VO_2 did not correlate with age, baseline exercise function, Borg RPE score, or attendance. Although the patients' $\Delta VE/\Delta VCO_2$ correlated negatively with their peak VO_2, changes in the $\Delta VE/\Delta VCO_2$ from

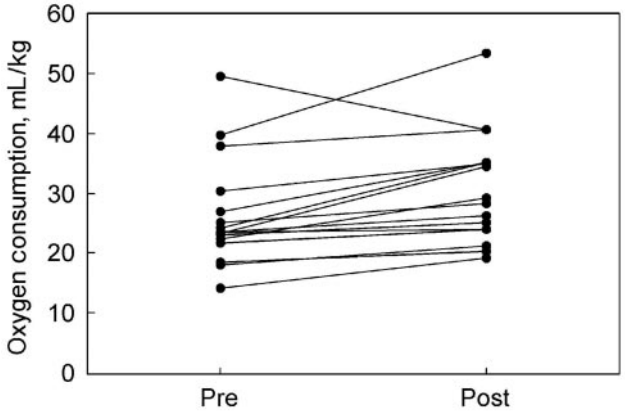

Fig 1. Effect of cardiac rehabilitation on peak oxygen consumption. Peak oxygen consumption rose in 15 of 16 patients after cardiac rehabilitation. Pre, before rehabilitation; post, after rehabilitation.

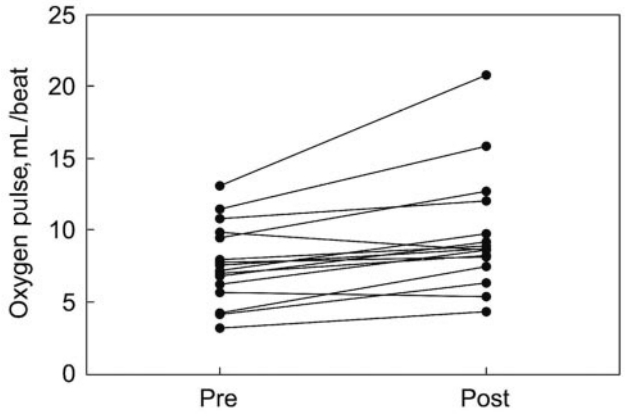

Fig 2. Changes in peak oxygen pulse after cardiac rehabilitation. The peak oxygen pulse declined in the 1 patient whose peak oxygen consumption declined. It fell slightly in 1 other patient and rose in the remaining 14 patients.

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TABLE 3. Effect of Cardiac Rehabilitation on Submaximal Exercise Parameters

	Before Rehabilitation	After Rehabilitation	% Change	Mean Difference	*P* Value
VO_2 at VAT, mL/min	649 ± 259	836 ± 267	29	199	<.001
VO_2 at VAT, mL/kg per min	14.2 ± 4.8	17.4 ± 4.5	23	4.0	.001
VO_2 at VAT, % predicted peak VO_2	32.8 ± 8.1	42.7 ± 10.5	30	10.8	.001
Work rate at VAT, W	45.1 ± 21.6	54.1 ± 15.6	20	9.9	.02
Work rate at VAT, W/kg	0.99 ± 0.44	1.16 ± 0.41	17	0.23	.02
Work rate at VAT, % predicted peak work rate	29.7 ± 11.0	36.6 ± 15.6	23	7.7	.05
HR at VAT, bpm	118.0 ± 23.4	123.5 ± 22.0	5	5.5	NS
O_2 pulse at VAT, % predicted peak O_2 pulse	55.2 ± 15.0	68.5 ± 18.2	24	14.8	.001
VE at VAT, L/min	23.3 ± 7.5	28.7 ± 7.3	23	5.7	<.001
Breathing reserve at VAT, %	72.8 ± 6.5	69.1 ± 7.7	−5	−4.3	NS
Tidal volume at VAT, L	0.72 ± 0.27	0.89 ± 0.21	24	0.16	.002
Respiratory rate at VAT, breaths/min	33.9 ± 7.9	32.5 ± 5.4	−4	−0.4	NS
VE/VO_2 at VAT	38.3 ± 10.2	34.8 ± 5.5	−9	−3.7	NS
VE/VCO_2 at VAT	38.9 ± 7.8	36.5 ± 4.9	−6	−2.7	NS
$\Delta VE/\Delta VCO_2$	36.1 ± 5.1	38.6 ± 8.3	7	2.4	NS

TABLE 4. Effect of Cardiac Rehabilitation on Measurements at Rest

	Before Rehabilitation	After Rehabilitation	% Change	Mean Difference	*P* Value
Height, cm	150 ± 13	153 ± 13	2	3	<.001
Weight, kg	46.8 ± 16.2	49.2 ± 17.8	5	2.4	.01
BMI, kg/m^2	20.1 ± 4.8	20.3 ± 4.9	1	0.2	NS
Systolic BP, mm Hg	106 ± 13	108 ± 9	2	2	NS
Diastolic BP, mm Hg	68 ± 10	68 ± 9	0	0	NS
FVC, L	2.40 ± 0.86	2.54 ± 0.87	6	0.13	.07
FVC, % predicted	82.2 ± 16.0	81.1 ± 14.6	−1	−1.0	NS
FEV1, L	2.08 ± 0.74	2.23 ± 0.78	7	0.16	<.001
FEV, % predicted	81.7 ± 16.3	82.2 ± 15.8	1	0.5	NS
FEV1/FVC	0.87 ± 0.10	0.89 ± 0.08	2	0.01	NS
O_2 saturation, %	95.8 ± 3.0	95.1 ± 3.1	−1	−0.7	NS

pre– to postrehabilitation tests were not associated with the corresponding changes in peak VO_2.

DISCUSSION

This study demonstrates that the exercise function of many patients with palliated CHD can be significantly improved by a 12-week cardiac rehabilitation program. In this series, the improvement seems to have been mediated by an increase in the oxygen pulse during exercise. Because the oxygen pulse is the product of stroke volume and oxygen extraction, an increased oxygen pulse could result from improvements in either or both of these parameters. Studies in adults indeed have found that an increase in oxygen extraction is 1 mechanism by which exercise function is enhanced by cardiac rehabilitation.[35] Past studies, in children and adults, have also demonstrated that exercise training can enhance (and that inactivity can have a deleterious effect on) the stroke volume and cardiac output response to exercise.[16,36] The significant increase in our patients' peak-exercise diastolic blood pressure after cardiac rehabilitation is consistent with a rehabilitation-induced augmentation of the cardiac output (and because the heart rate was unchanged, the stroke volume) at peak exercise and suggests that this second mechanism was at least partially responsible for the observed improvement in exercise function.

The improvements in exercise capacity and cardiovascular function achieved by the patients of this study we believe are related to and underscore the important bidirectional nature of the interaction between the cardiovascular system and the skeletal muscles. Although the cardiovascular system provides the blood flow necessary to accommodate the metabolic requirements of the skeletal muscles during exercise, the pumping action of the skeletal muscles in turn helps to maintain ventricular preload. This pumping action makes an important contribution to the normal increase in cardiac output during exercise.[37] We speculate that the improvements identified in this and other studies are related, at least in part, to a rehabilitation-induced increase in the strength, mass, and pumping capacity of the skeletal muscles.

We believe that this interaction between the skeletal muscles and the cardiovascular system is an important clinical concept that may not be fully appreciated. An implication of this interaction is that interventions that address only 1 component of the skeletal muscle–cardiovascular unit may have a limited impact on exercise capacity, unless they are combined with programs that are designed to improve the function of the other component. This phenomenon probably explains the common observation that surgical and/or interventional catheterization procedures that achieve dramatic hemodynamic results often, in the short term, have a relatively minor impact on exercise function.[3,13,21,38–42] In our opinion, these observations and the results of our study provide strong arguments for the incorporation of cardiac rehabilitation into the care of many patients with CHD. Indeed, the improvements achieved in this study equal or exceed those obtained with many other medical or surgical interventions. Furthermore, no adverse events were encountered in conjunction

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with our rehabilitation program, suggesting that, in this class of patients, the risks associated with cardiac rehabilitation are low and the risk:benefit ratio for this form of therapy is favorable.

In light of recent studies that have identified the $\Delta VE/\Delta VCO_2$ to be a strong predictor of mortality in patients with heart failure,[30,43] an important correlate of exercise function in patients who have undergone surgical repair of tetralogy of Fallot,[9,44] and to be elevated after the Fontan procedure,[45] it is interesting to note that rehabilitation had no effect on this parameter and that the rehabilitation-related rise in peak VO_2 did not correlate with concurrent changes in $\Delta VE/\Delta VCO_2$. Furthermore, rehabilitation seemed to have no effect on other indices of pulmonary function, such as baseline spirometric measurements or the ventilatory equivalents for oxygen or carbon dioxide at the VAT. Indeed, our data point primarily to an improvement in cardiovascular function as the source for the rehabilitation-related improvement in exercise performance.

The results of this study compare favorably with most previous studies of cardiac rehabilitation in children. Bradley et al[19] studied the effect of a 12-week rehabilitation program on the exercise function of 11 patients with CHD (9 of whom completed the program) and found improvements in peak VO_2 and endurance time comparable to those attained in our series. However, these investigators noted a significant increase in heart rate on the postrehabilitation exercise test but did not report their patients' RER before and after rehabilitation. It therefore was unclear whether the effect that they observed was in fact attributable to an increased effort rather than an objective improvement in exercise function. They also did not detect a significant increase in their patients' VE, which, in the absence of an (implausible) improvement in the lungs' efficiency of gas exchange, seems to be incompatible with the observed increase in peak VO_2. Balfour et al[18] reported data from 6 patients who had CHD and completed a 3-month rehabilitation program. They also found improvements similar to those achieved in our study. However, their study was small, was plagued by a high dropout rate, and included patients with a relatively low acuity of disease. They also did not provide data regarding the patients' heart rate and RER. Neither of these studies provided insights into the mechanisms by which the patients' exercise function might have improved.

Other investigators have reported more modest improvements after cardiac rehabilitation. Goldberg et al,[20] in a study of 26 patients with repaired tetralogy of Fallot or ventricular septal defects, found that a 6-week home exercise program using stationary bicycles improved peak work capacity but had no effect on peak VO_2. Ruttenberg et al[23] studied 12 patients with a variety of congenital heart defects and found that a 9-week program based on a jogging and walking regimen improved treadmill endurance time but did not improve peak VO_2. Similarly, Fredriksen et al[25] found that 55 patients who had a wide spectrum of congenital heart defects and participated in a training program that introduced them to a variety of sports and other physical activities achieved a small (<5%) improvement in endurance time and no improvement in peak VO_2 normalized for body weight. Minamisawa et al,[22] in a study of 11 children and young adults who had had Fontan procedures, found that a 2- to 3-month home exercise program produced only small (~7%) improvements in peak VO_2 and peak work rate. They did not detect a significant change in oxygen pulse or respiratory function.

We believe that the favorable outcomes obtained in our study are related to the flexibility of our program, the use of age-appropriate incentives, the low patient:staff ratio, the opportunity for prompt feedback and encouragement, and the high priority that was placed on pursuing activities that accommodated the individual needs/desires of our patients. We also believe that our patients were motivated by the opportunity to exercise in a child-oriented environment, with children their own age and carrying similar diagnoses. In our opinion, cardiac rehabilitation facilities should incorporate these features into their pediatric programs to maximize the likelihood that patients will be properly motivated and derive optimal benefit from their rehabilitation sessions.

Limitations

This study examined only the immediate impact of cardiac rehabilitation on children with CHD. Additional studies are necessary to determine whether the benefits identified in this study are sustained over an extended period of time and whether they produce improved activity levels and lifestyles. The number of patients who were involved in this study was also relatively small. Although sufficient to provide persuasive data regarding the benefits of cardiac rehabilitation in children, the study was not large enough to characterize definitively the risks associated with this form of therapy. Additional studies are also needed to establish the optimal design, duration, and intensity of a pediatric cardiac rehabilitation program.

It must also be noted that the patients in this study were a highly selected group. The results of this study therefore may not be applicable to patients with milder degrees of disability or to patients with medical conditions that were excluded from this study.

CONCLUSION

This study has demonstrated that a 12-week, semiweekly cardiac rehabilitation program with encouragement of additional home exercise can significantly improve the exercise performance of children with serious CHD. This improvement seems to be mediated by an increase in stroke volume and/or oxygen extraction during exercise. Routine use of formal cardiac rehabilitation may reduce the morbidity of complex CHD.

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Jonathan Rhodes, Tracy J. Curran, Laurel Camil, Nicole Rabideau, David R. Fulton, Naomi S. Gauthier, Kimberlee Gauvreau and Kathy J. Jenkins

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