Cardiorespiratory Responses to Aerobic Training by Patients With Postpoliomyelitis Sequelae

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We examined the cardiorespiratory responses of 16 patients with postpoliomyelitis sequelae to a 16-week aerobic exercise program. The patients exercised at 70% of maximal heart rate. Dependent variables were resting and maximal heart rates, systolic and diastolic blood pressures, maximum oxygen consumption, maximum carbon dioxide consumption, respiratory quotient, and maximum expired volume per unit time. The exercise group was superior to the control group in watts, exercise time, maximum expired volume per unit time, and maximum oxygen consumption. No untoward events or loss of leg strength occurred as a result of the exercise regimen. We conclude that the aerobic training program employed in this study is a safe, short-term procedure and that patients with postpolio sequelae respond to training in a manner similar to healthy adults.

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IT IS estimated that the second most prevalent condition that causes residual paralysis in American households is poliomyelitis.[1] There probably are 400 000 polio survivors (paralytic and "nonparalytic" persons) at risk for developing a constellation of new medical problems known as *postpolio sequelae*. After 30 or more years of medical stability following recovery from polio, survivors are reporting some or all of the following symptoms: (1) fatigue, generally described as a feeling of extreme exhaustion after minimal activities coupled with decreased endurance; (2) new joint and muscle pain; (3) progressive weakness in muscles both affected and supposedly unaffected by acute polio; (4) new respiratory difficulties, including sleep apnea, that necessitate a return to ventilatory devices; and (5) cold intolerance that contributes to muscle weakness and discoloration of limbs, with burning pain.

For editorial comment see p 3294.

Many patients who report symptoms are younger than 60 years. Beyond age 60 years, Tomlinson and Irving[2,3] and others have estimated that there is a normal age-related loss of anterior horn cells at 5% per decade. Thus, there must be other processes that contribute to this deterioration, including decreased function or loss of overworked surviving anterior horn cells or impaired neuromuscular transmission. Recently, several workers have presented evidence supporting failure of terminal axonal sprout function, causing defective transmission at the neuromuscular junction.[4-6]

Treatment of these problems presents a challenge. Overuse of muscles that operate at maximal capacity with minimal or no reserve can result in irreversible damage to muscle fibers, to terminal axonal sprouts, or even to anterior horn cells themselves.[7]

Use of exercise as a treatment modality for postpolio sequelae has come under close scrutiny by the medical community because of fear of increased muscle weakness resulting from overuse. Health practitioners have discouraged vigorous physical activity. However, recent studies[8,9] report that a reduction in prolonged strenuous activity combined with carefully prescribed progressive resistance exercises and cardiovascular conditioning has a positive effect on both muscle weakness and fatigue. Because of the paucity of research dealing with the effects of conditioning programs on postpolio survivors, we investigated the effects of a 16-week modified aerobic exercise program on the cardiorespiratory fitness levels of patients with postpolio sequelae. More specifically, we examined the changes in maximum oxygen consumption ($\dot{V}O_2max$), carbon dioxide consumption per unit time ($\dot{V}CO_2$), expired volume per unit time ($\dot{V}E$), and work capacity as a result of an aerobic conditioning program in patients with postpolio sequelae

PATIENTS AND METHODS

Patients

A total of 45 potential patients were recruited via a postpolio newsletter, from the outpatient clinic at Sister Kenny Institute, Minneapolis, Minn, and from among individuals currently living in the Minneapolis–St Paul metropolitan area who had polio between 1952 and 1959. Thirty-seven patients completed the study. The following inclusion criteria were applied: (1) a history of polio verified by hospital records, involvement in selected epidemics, and/or a history of paralysis with some subsequent recovery and stabilization of function; (2) 10 to 20 years of neurological stability following the acute onset; (3) present age between 30 and 60 years; (4) adequate strength in at least one lower extremity to pedal an exercycle (defined as quadriceps and hip flexor strength of at least 3/5 according to the manual test [3 = antigravity] and hip extensors and knee flexors that possessed at least 2 strength levels [2 = motion within a gravity-limited plane]); and (5) arm and torso strength adequate to allow safe and stable positioning on a cycle ergometer. By not including participants older than 60 years, the investigators attempted (1) to eliminate the contribution of normal aging-related loses of anterior horn cells, thus better assessing the factor of muscle overuse, and (2) to decrease the likelihood of occult cardiovascular disease.

Criteria for exclusion were (1) a diagnosis of diabetes mellitus, compression neuropathies, radiculopathies verified by electromyography, multiple sclerosis, amyotrophic lateral sclerosis, muscular dystrophies, myasthenia gravis, cerebrovascular accident, or spinal cord injury; (2) a history of recent and/or prolonged (>3 months) steroid use; (3) a

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history of cardiovascular disease, including cardiac arrhythmia, angina, uncontrolled hypertension, myocardial infarction, valvular disease, and congenital heart disease; (4) current participation in any aerobic or muscle strength training program; and (5) abnormalities on pretraining stress testing. Patients with distal lower-extremity weakness (dorsi or plantar flexion) were not excluded as long as they wore an ankle-foot orthosis during exercise or possessed adequate foot stability when pedaling with the foot positioned properly and strapped to the pedal. On initial evaluation most patients described a mild decrease in muscle strength of affected limbs and moderate problems with fatigue and endurance. Applicants with severe progressive weakness, atrophy, and fatigue were excluded, and the participants were self-selected by their motivation to attend an ongoing exercise program.

Table 1.—Descriptive Statistics for the Control Group (n = 21) and the Exercise Group (n = 16)

Dependent Variable	Before Training		After Training	
	Mean	SD	Mean	SD
Control Group				
Resting heart rate, beats per minute	85.9	14.6	83.5	11.5
Maximal heart rate, beats per minute	162.8	15.5	164.2	18.2
Resting systolic blood pressure, mm Hg	119.5	19.2	119.8	20.1
Resting diastolic blood pressure, mm Hg	77.8	10.6	76.9	8.8
Maximum systolic blood pressure, mm Hg	186.0	23.1	185.5	20.8
Maximum diastolic blood pressure, mm Hg	92.8	8.0	86.9	7.9
Watts	123.9	52.4	131.4	52.4
Exercise time, s	474.0	151.3	482.9	161.5
Maximum expired volume, L/min	66.6	28.7	72.6	30.6
Maximum oxygen consumption, mL/min	1543.9	556.2	1603.6	609.6
Maximum carbon dioxide consumption, mL/min	1877.8	775.8	1938.1	875.8
Respiratory exchange ratio	1.21	0.22	1.17	0.14
Exercise Group				
Resting heart rate, beats per minute	80.7	9.8	75.0	8.5
Maximal heart rate, beats per minute	157.3	16.7	162.1	13.8
Resting systolic blood pressure, mm Hg	121.6	19.9	116.9	16.6
Resting diastolic blood pressure, mm Hg	77.0	8.6	75.1	7.4
Maximum systolic blood pressure, mm Hg	188.2	24.1	181.0	24.7
Maximum diastolic blood pressure, mm Hg	92.9	11.6	88.0	8.1
Watts	123.5	36.6	145.5	34.9
Exercise time, s	457.1	103.3	526.2	111.2
Maximum expired volume, L/min	55.4	11.4	71.0	14.5
Maximum oxygen consumption, mL/min	1513.6	471.9	1739.9	541.3
Maximum carbon dioxide consumption, mL/min	1791.5	510.6	2041.9	566.1
Respiratory exchange ratio	1.19	0.14	1.18	0.13

Testing

Metabolic measurements were taken at the beginning and end of the 16-week training program. All patients signed informed consent forms. Prior to initial testing, patients were measured for height and weight and were fitted with electrodes to monitor heart rate (HR) and any atypical coronary response to exercise. After sitting quietly for 5 minutes, resting HR and blood pressure were recorded.

Patients were positioned on a cycle ergometer and feet were strapped securely to the pedals. The testing protocol involved continuous exercise on an electronically braked cycle ergometer. Patients pedaled at 50 to 70 rpm throughout the exercise test. During the first minute of exercise, no resistance was applied to the cycle ergometer. After a 1-minute warm-up, workloads were increased in 1-W intervals at a rate of 20 W/min using a ramping protocol. This increase in magnitude was designed to fatigue the patients in 5 to 8 minutes. The protocol was developed specifically to eliminate local muscle fatigue as the cause for test termination prior to the attainment of $\dot{V}O_2$max.

Patients pedaled at 50 to 70 rpm continuously until at least one of the following: (1) oxygen consumption per unit time ($\dot{V}O_2$) failed to increase with increased workload; (2) the respiratory exchange ratio exceeded 1.0; (3) cardiovascular abnormalities including decreased HR or blood pressure response to increasing workloads or electrocardiographic abnormalities including frequent or multifocal premature ventricular contractions or ST-segment displacement of 2 mm or more occurred; (4) volitional fatigue; and (5) inability to maintain a pedaling cadence of at least 40 rpm. Cardiorespiratory variables were measured using an exercise laboratory (CAD NET System 2001, Medical Graphics Corp, St Paul, Minn), allowing for continuous computerized sampling. Data were recorded and averaged over 30-second intervals.

The following metabolic variables were recorded: (1) resting HR; (2) resting blood pressure; (3) maximal HR; (4) maximal blood pressure; (5) $\dot{V}O_2$max; (6) maximum carbon dioxide consumption ($\dot{V}CO_2$max); (7) respiratory exchange ratio; and (8) maximum expired volume ($\dot{V}E$max). At the completion of the exercise test, patients were instructed to continue pedaling against no resistance for 2 to 3 minutes and were then placed in a sitting position for recovery.

Training Period

After the preliminary stress test, patients were assigned randomly to either a control or an exercise group. This allowed both the attending physician and the technician responsible for data collection to be blinded observers. Two patients were excluded after the initial test: one demonstrated a hypertensive response to exercise and the other showed significant ST-segment depression. The control group was instructed to continue with normal daily activities and to refrain from starting any form of aerobic or muscle strengthening exercise programs for the duration of the study.

Based on the pretest results, each patient assigned to the exercise group was provided with an individualized exercise prescription. All patients trained on a cycle ergometer at an intensity of 70% to 75% of the reserve HR plus resting HR. Target HR was calculated using the following formulas:

$$\text{Reserve HR} = \text{Maximal HR} - \text{Resting HR}$$
$$\text{Target HR} = (\text{Reserve HR} \times k) + \text{Resting HR}$$

In the second formula, k varied between 0.70 and 0.75.

This protocol is used routinely in our clinic and seems to represent the highest intensity of exercise that can be tolerated safely by this population. Patients were instructed to monitor intensity by checking their pulse rates at the carotid or radial artery during the first 10 seconds after exercise. This value was multiplied by 6 to estimate exercise HR. Patients were spot-checked for accuracy throughout the training period. Each patient reported to the laboratory for training once per week, with the remaining sessions performed on a cycle ergometer in the home.

Patients were instructed to exercise for 15 to 30 minutes per session. Most patients started exercising 15 minutes per session; after 4 weeks all had increased the duration to 20 minutes. To avoid the risk of overuse abuse prob-

Table 2.—Descriptive Statistics for Change Scores

Dependent Variable	Control Group		Exercise Group	
	Mean	SD	Mean	SD
Resting heart rate, beats per minute	−2.4	16.0	−5.7	11.6
Maximal heart rate, beats per minute	1.4	10.5	4.8	6.9
Resting systolic blood pressure, mm Hg	0.3	20.8	−4.7	11.7
Resting diastolic blood pressure, mm Hg	−0.9	11.0	−1.9	6.6
Maximum systolic blood pressure, mm Hg	0.5	15.6	−7.2	16.1
Maximum diastolic blood pressure, mm Hg	−5.9	10.9	−4.9	7.3
Watts	7.5	14.0	22.0*	15.2
Exercise time, s	8.9	30.9	69.1*	42.9
Maximum expired volume, L/min	6.0	8.6	15.6*	14.2
Maximum oxygen consumption, mL/min	59.7	215.0	226.3*	289.5
Maximum carbon dioxide consumption, mL/min	60.3	297.3	250.4	436.1
Respiratory exchange ratio	−0.04	0.22	−0.01	0.17

*Indicates variables that were improved significantly ($P<.05$) in the exercise group.

lems of muscle fatigue, the exercise sessions were divided into prescribed bouts of 2 to 5 minutes interspersed with 1-minute rest periods. Patients whose cardiovascular endurance levels on initial evaluation exceeded 6 metabolic equivalents of oxygen consumption were instructed to begin with 4- to 5-minute exercise bouts, whereas those at less than 6 metabolic equivalents of oxygen consumption began with 2- to 3-minute exercise bouts. The frequency of exercise sessions was three times per week (except two patients who exercised four times weekly) distributed over the entire week.

For each exercise session, patients monitored and recorded the duration of each exercise bout, the number of minutes exercised during the session, the HR at cessation of each bout, and the perceived exertion at the conclusion of each bout. Patients rated perceived exertion according to the Borg Scale of Perceived Exertion. Both this and exercise HR were considered in determining the need to modify each patient's exercise prescription.

As indicated previously, patients were required to exercise once per week under the supervision of the investigator to document and reinforce adherence to the exercise program and to make necessary adjustments in the exercise program. During the initial stages of training, a number of patients required a reduction in the intensity and/or duration of the exercise bouts. Exercise HRs were reduced by 6 to 12 beats per minute for some patients, whereas others, particularly those whose exercise prescriptions began with 4- to 5-minute exercise bouts, were instructed to reduce the duration of the bout to facilitate more rest within each exercise session. The absolute amount of exercise remained unchanged as patients added more bouts of exercise to accommodate the reduced duration.

All patients were able to exercise at their initially prescribed intensities of exercise by the end of the fourth week. Patients were instructed to increase the duration of the exercise bouts as their muscles tolerated this change. Exercise diaries were kept and reviewed weekly. All changes were discussed and approved by the investigator at the weekly supervised exercise sessions. No exercise or control subjects participated in other aerobic exercise programs during the study. At the completion of the 16-week training program, all patients were retested on the cycle ergometer.

RESULTS

The mean duration of the exercise bouts over the 16-week training program was 4.21 minutes. Each patient trained an average of 20.33 minutes per session for 2.89 sessions per week. The mean training HR was 128.9 beats per minute, which represented 69.2% of the reserve HR plus resting HR.

Six patients withdrew from the study, although none withdrew as a result of adverse effects of the increased levels of physical activity during training. Six patients in the exercise group were eliminated from the study for the following reasons: (1) failure to attend a single exercise session; (2) serious illness of a family member; (3) fractured hip at home; (4) the inability to complete final testing because of respiratory viral infection; (5) extreme pain in pedaling caused by a preexisting failed fusion of forefoot bones; and (6) overtime at work. Descriptive statistics for the cardiorespiratory variables are presented in Table 1 for the control and exercise groups.

Change scores were calculated for each patient by subtracting the pretest score from the posttest score. The means and SDs for the change scores for the exercise and control groups are presented in Table 2.

The change scores were grouped into two categories for analysis. The first category included the following cardiovascular variables: (1) resting HR, (2) maximal HR, (3) resting systolic pressure, (4) resting diastolic pressure, (5) maximal systolic pressure, and (6) maximal diastolic pressure. The second category included the following work and respiratory variables: (1) watts, (2) exercise time, (3) respiratory exchange ratio, (4) $\dot{V}E$max, (5) $\dot{V}O_2$max, and (6) $\dot{V}CO_2$max. The relative changes were compared using multivariate analysis of variance, and the statistical test performed was Hotelling's T^2. The analysis for the cardiovascular variables failed to demonstrate any significant effect; therefore, no further analysis was applied to the first category.

For the second category, the Hotelling's T^2 value of 1.334 was significant ($P<.05$), which indicated a need for further analysis. Thus, unpaired Student's t tests were performed to determine which of the individual variables responded to the exercise program. The results of those analyses revealed that the exercise group was significantly ($P<.05$) superior to the control group in watts, exercise time during testing, $\dot{V}E$max, and $\dot{V}O_2$max.

COMMENT

During testing, only 2 of 37 patients failed to attain a respiratory exchange ratio of at least 1.00. The lowest recorded ratio was 0.97 and the highest was 1.37, with a mean of 1.20. In addition, every patient achieved at least one of the predetermined cardiovascular end points for test termination. The mean maximal HR attained during testing was 161.86 beats per minute. This maximal HR closely approximated the age-predicted values for normal patients on a cycle ergometer.[10] These results provide strong evidence to suggest that despite their compromised neuromuscular status, patients with postpolio sequelae can achieve a workload sufficient to elicit a true $\dot{V}O_2$max.

The continuous ramping protocol, which involves increasing the workload by 20 W/min in 1-W increments, was effective in eliciting $\dot{V}O_2$max prior to the onset of muscular fatigue. None of the patients presented any overuse symptoms associated with a one-time maximal effort on the cycle ergometer. No difference between the bulbar and nonbulbar patients in $\dot{V}E$ was observed at rest or during maximal exercise. During initial testing, the mean $\dot{V}E$max for bulbar patients was 65.2 L/min and for nonbulbar, 65.4 L/min. The exercise and control groups were comparable on all dependent variables at the beginning

of the study.

Although six patients withdrew from the exercise group, none failed to complete the training program because of any adverse effects associated with the exercise protocol. In pretesting and posttesting of knee flexion and extension using isokinetic strength measurements (Cybex), not one experimental patient experienced any strength decrement after training. The six experimental patients who dropped out of the study did so either for personal reasons or because of illness or injury unrelated to the experiment.

It was, however, necessary to make minor modifications in the exercise prescriptions during the first 4 weeks of training. The duration of the individual exercise bouts was reduced to 2 to 3 minutes for several patients whose original prescriptions called for bouts of 4 to 5 minutes. Although the original exercise prescriptions for duration were based on the metabolic equivalent of oxygen consumption levels attained during initial testing, this procedure was not effective. It is recommended, therefore, that in an aerobic training program such as this for patients with postpolio sequelae, a more conservative approach be followed and initial bouts range between 2 and 3 minutes in duration.

During the first several weeks of training, three patients were unable to maintain throughout the entire bout the prescribed training intensity of 70% of the reserve HR plus resting HR. Consequently, the initial intensity was reduced to approximately 60% and then increased gradually. All patients were able to complete their exercise bouts at an intensity of 70% of reserve HR plus resting HR by the fourth week.

Three exercise sessions per week provided an adequate training stimulus to demonstrate gains in (1) watts attained during testing, (2) duration of testing, (3) $\dot{V}E$, and (4) $\dot{V}O_2$max. With the exception of two patients who exercised 4 days per week, at least 1 day of rest was interposed between training sessions. The two patients who trained four times weekly improved their $\dot{V}O_2$max values by just 4%, whereas the mean gain for the exercise group was 15%. This result was unanticipated in that the initial $\dot{V}O_2$max values for the two patients who trained four times per week were 1167 and 1154 mL/min, whereas the overall mean for the exercise group was 1513 mL/min. This suggests that those two patients began training at a lower level of conditioning than their fellow patients and would, therefore, be expected to demonstrate a higher response to the training. It is possible that although their initial $\dot{V}O_2$max values were considerably below the mean, those two patients were actually closer to their $\dot{V}O_2$max values at the beginning of training than their fellow patients in the exercise group. Another possible explanation is that for patients with postpolio sequelae, it is important that at least a full day of rest be provided between vigorous exercise sessions to allow for full recovery from the previous session.

The 15% improvement in $\dot{V}O_2$max by the exercise group is consistent with the results for healthy adult men of approximately the same age. For example, Ribisl[11] reported an increase in $\dot{V}O_2$max of 14% following 20 weeks of training, Wilmore et al[12] reported an increase of 10% after 10 weeks, and Naughton and Nagle[13] reported an increase of 18% after 28 weeks. In each of these studies, the mean age of the patients was 40 to 41 years, and exercise sessions were held three times per week. Thus, the results of the present study indicate that patients with postpolio sequelae respond to an aerobic training program with increases in $\dot{V}O_2$max of a magnitude similar to normal, healthy adults of the same age range.

Of 16 patients in the exercise group, 4 had significant preexisting muscular atrophy and weakness in one leg, 1 of whom required bracing for exercise. None of these patients demonstrated any overuse abuse symptoms during the training period. To the contrary, patients with preexisting atrophy seemed to experience the greatest amount of benefit from the training program. Their relative improvement in $\dot{V}O_2$max was 26% compared with a mean improvement of 15% for the entire group. Thus, it seems that patients with postpolio sequelae who possess sufficient strength to pedal a cycle ergometer can profit from an aerobic training program such as the one employed in this study, and neither preexisting atrophy nor a need for bracing during exercise is a contraindication for participation. All exercise patients thought that endurance had improved and that fatigue with normal daily activities had decreased. No one reported additional muscle weakness and most thought that lower extremity strength had increased, which was confirmed by isokinetic strength measurements.

Both the use of (1) a day of rest between exercise sessions and (2) minirests of 1 minute between exercise bouts can decrease the likelihood of overuse effects. This might be sufficient time for metabolic recovery of the anterior horn cells, axonal sprouts, neuromuscular junction, and muscle fibers themselves. This lack of sustained demand might allow a patient with postpolio sequelae to gain cardiorespiratory endurance while minimizing chances of terminal axonal damage as well as allowing sufficient recovery/reinnervation. The effects of aerobic conditioning also might improve muscular and neuronal oxygenation that can delay the onset or progression of postpolio sequelae.

Currently, there is little knowledge of the effects of long-term aerobic exercise participation on the development or progression of postpolio syndrome. We plan to study patients with postpolio syndrome using the protocol over several years to determine the benefits or risks of aerobic conditioning. This study indicates that patients with postpolio sequelae and adequate strength to pedal a cycle ergometer can exercise safely at an HR of at least 70% of the reserve HR plus resting HR. Such patients seem to experience improvement in cardiorespiratory fitness as a consequence of exercise comparable with that of age-matched healthy counterparts.

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