Submaximal Exercise Capacity and Maximal Power Output in Polio Subjects

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Objectives: To compare the submaximal exercise capacity of polio subjects with postpoliomyelitis syndrome (PPS) and without (non-PPS) with that of healthy control subjects, to investigate the relationship of this capacity with maximal short-term power, and to evaluate movement economy.

Design: Cross-sectional survey.
Setting: University hospital.
Participants: Forty-three polio subjects (25 PPS, 18 non-PPS) and 12 control subjects.
Interventions: Not applicable.
Main Outcome Measures: Power output, oxygen uptake, and heart rate were measured in an incremental submaximal cycle ergometry test. Maximal short-term power was measured in 5-second all-out efforts. Knee extensor strength was measured on a chair dynamometer.
Results: The mean submaximal power ± standard deviation at 80% of heart rate reserve of 83.8 ± 29.9 watts in the polio subjects was significantly less than the mean submaximal power of 142.1 ± 30.4 watts in the control group. However, expressed as a percentage of the maximal short-term power, submaximal power did not differ between the groups. Strength and maximal short-term power correlated significantly (p < .005) with submaximal power (r = .64 and .76, respectively). The oxygen uptake was higher than theoretically expected for the given submaximal power output in polio subjects, and appeared to increase with increasing asymmetry in strength and power between legs. No differences were found between PPS and non-PPS subjects.
Conclusion: The submaximal work capacity of polio subjects was severely reduced, mainly in association with the reduced muscle capacity. And, because of a reduced movement economy, their energy cost was elevated. Although muscle loads in activities such as walking and climbing stairs differ from cycling, they also may require elevated relative levels of effort, predisposing subjects to premature fatigue in sustained activity.

Key Words: Exercise test; Fatigue; Isometric contraction; Muscles; Physical endurance; Physical fitness; Poliomyelitis; Postpoliomyelitis syndrome; Rehabilitation; Work capacity evaluation.
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AFTER MANY YEARS OF NEUROLOGIC and functional stability, patients who suffered from polio earlier in their lives may develop late-onset neuromuscular symptoms, including new muscle weakness, which is referred to as postpoliomyelitis syndrome (PPS). 1 Patients with PPS report fatigue and a decline in their functional abilities, especially walking outdoors and climbing stairs, as their major problems. 2 Some studies have reported a poor cardiorespiratory condition in terms of a low maximal oxygen uptake (V\textsubscript{O\textsuperscript{2} max}) in subjects with polio residuals 3-5 and in subjects with PPS. 5 However, the maximal oxygen uptake achieved was not compared with the extent of the residual paresis from polio. This factor must be taken into account because maximal oxygen uptake also depends on the quantity of active muscle mass, shown in studies comparing 1- and 2-leg cycling. 6-7 In a recent study of predominantly PPS subjects and some subjects with late effects not classified as PPS, Willén et al 9 did find that peak oxygen uptake and peak power correlated with muscle strength of the knee extensors and flexors. However, from their results it is difficult to draw conclusions on cardiorespiratory conditioning because it appeared that the maximum was often not reached: their subjects' average maximal heart rate, 138 beats/min, was approximately 30 beats/min below the predicted maximal heart rate of 220 minus age.

Studies that focus on submaximal work capacity in polio subjects are scarce, and mainly describe training effects in polio survivors or polio subjects with new neuromuscular symptoms. 10-12 Dean and Ross 4 reported that the cardiorespiratory conditioning when walking was reduced in some polio subjects because they had higher heart rate values compared with predicted heart rate values for the oxygen uptake levels achieved.

We are not aware of any studies that have compared submaximal exercise capacity between PPS subjects and stable-functioning polio subjects without symptoms of PPS, or with healthy control subjects. It may well be that the symptoms of fatigue and problems with walking longer distances are not simply caused by poor cardiorespiratory conditioning, but primarily result from a limited physical capacity related to the degree of paresis, 13 and from the extra energy expenditure required because of reduced movement economy. 11

Because activities such as walking in daily life situations are at submaximal exercise levels, the present study focused primarily on the submaximal exercise capacity of polio subjects in steady-state cycle ergometry. We hypothesized that the sub-
maximal exercise capacity of PPS subjects would be worse than that of non-PPS subjects. The objectives of the study were: (1) to compare the submaximal exercise capacity of polio subjects with that of healthy control subjects, and also between polio subjects with and without PPS, (2) to investigate whether muscle strength and maximal short-term power output are major determinants of submaximal work capacity, and (3) to determine whether the oxygen uptake is increased in relation to a reduced movement economy.

METHODS

Subjects

A group of 54 subjects from a cohort of 103 polio subjects, previously studied and described in detail elsewhere, were considered for participation in the present study. All 27 stable-functioning polio subjects from the cohort (non-PPS) and the 27 PPS subjects, who were matched to the stable subjects with regard to age and sex, formed the present study subgroup. The diagnosis of PPS was made according to the criteria of Halstead. The group of 12 healthy control subjects consisted of volunteers from the university staff and others who responded to an inhouse advertisement. All polio subjects met the following inclusion criteria applied in the cohort study: (1) a history of poliomyelitis, that is, an acute febrile illness, with paresis of 1 or more limbs followed by a partial recovery of function; (2) on physical examination, residual muscle weakness, atrophy, and hypo- or areflexia with normal sensation in at least 1 extremity; and (3) capability of walking with or without walking aids. The cohort study exclusion criteria were conditions that: (1) might hinder adequate testing, such as joint pain or muscle pain in the legs, or low back pain; (2) other neurologic conditions (eg, neuropathy, radiculopathy); or (3) endocrine disorders, cardiovascular disease, pulmonary disease, or psychiatric problems. Additional exclusion criteria for the present study were a poor lung function and inability to cycle on an ergometer.

The study was approved by the medical ethics committee of the university hospital, and written informed consent was obtained from all participants.

Polio History and Actual Status

History-taking focused on age at the acute polio stage, residual paresis, and walking ability after recovery in young adulthood, present walking ability, and physical activity level. Clinical involvement owing to polio was investigated through physical examination. Anthropometry included measurements of height, weight, and body fat from 4 skinfolds according to the method described by Durnin and Womersley. Lung function was determined with a Vica Test 5E device based on vital capacity and forced expiratory volume in 1 second (FEV1).

Testing Equipment

A cycle ergometer with 2 modes of operation was used. It could either serve as an electromagnetically braked Lode Standaard cycle ergometer, or it could be switched to an isokinetic mode in which the ergometer is driven by a 2.2kW electric motor at a constant speed, adjustable through a variable gearbox. In the latter mode, the pedal frequency remains constant, irrespective of the forces generated on the pedals. The ergometer was also modified with type 3/120xxy23s strain gauges mounted inside the pedals to measure the horizontal and vertical forces exerted. Force measurements were recorded with a sampling frequency of 150 samples/revolution and stored on a computer for off-line analysis. Power was calculated from these force measurements and simultaneous measurements of crank speed and crank and pedal angles. Calibration of these strain gauges was performed regularly with reference weights. Gas exchange variables were measured continuously with an Oxycon Gamma, and data were stored on a computer with the concomitant software program. Calibration of the analyzers with certificated calibration gases was performed regularly, and on all occasions values were found to be within the reference limits. Heart rate was monitored continuously on a Polar Sport Tester and was saved to a computer with the Polar Sport Tester interface and software.

Submaximal Exercise Test

Subjects performed an incremental exercise test with the cycle ergometer in the normal electromechanically braked mode with step durations of 4 minutes at a speed of approximately 70rpm. During the test, the subjects' feet were fixed to the pedals with toe-clips and extra bands, if necessary, if individually customized orthopedic footwear was worn. The seat height was adjusted individually to a position that the subject felt was optimal for cycling because objective criteria were often difficult to determine. For example, some subjects had leg-length discrepancies. The test ended when the subject completed the step in which he/she had cycled at or above their target heart rate, or when the pedal rate dropped below 60rpm. The target heart rate was calculated before the test, and was set at 80% of the heart rate reserve (HRR). The HRR is the difference between the maximal heart rate and the heart rate at rest. The maximal heart rate was estimated at 220 minus age, and the heart rate at rest was measured after 3 minutes of rest, with the subject lying supine. To equalize the test duration among all subjects, independent of their physical capacity, the magnitude of the increments were determined individually: each subject was expected to reach his/her target heart rate in 4 steps. The polio subjects typically started at a workload of 25 watts, compared with a workload of 50 watts for healthy control subjects. Depending on the heart rate in the last minute of each step, the increment for the next step was chosen. Typically the increments were steps of 25 or 50 watts. Throughout the test, gas exchange variables and heart rate were measured continuously. The average values of heart rate and gas exchange variables of the last 30 seconds of each step were used for analysis. The forces exerted on the pedals were measured for 15 revolutions in the third minute of each step. From these recordings the average power, generated through the entire cycle, was calculated per leg for each step.

Maximal Short-Term Power Measurements

Subjects performed all-out maximal cycling efforts for 5 seconds with the ergometer set in the isokinetic mode and the subject positioned and fixed as in the submaximal exercise test. Before a maximal effort was made, subjects were instructed to start pedalling until they reached the preset revolution speed and then to hold this speed without exerting extra force. Then, after a 3-second countdown, subjects had to make an all-out effort for 5 seconds in an attempt to accelerate the pedals. To optimize performance, subjects were verbally encouraged during each effort. First, 2 maximal efforts were made at 60rpm to warm up, followed by 1 maximal effort at 80rpm. The resting interval between efforts was 3 minutes. From the pedal forces recorded at 80rpm, the mean power generated by each leg for each revolution was calculated and the highest value was included in the analysis.
coefficients were calculated by means of Pearson’s correlation. Differences in power between polio subjects and healthy control subjects, and between PPS subjects and stable polio subjects, were analyzed with the chi-square test and ordinal data with the Wilcoxon’s signed-ranks test. Dichotomized variables were analyzed with the Student’s t-test and with the Mann-Whitney U test for not normally distributed data. Independent variables used were diagnosis of polio, gender, power delivered at the final increment of the submaximal test, lean body mass (body weight × [1 − fat%]), and asymmetry between the legs in power delivered at the final increment. The probability of F-to-enter was set at less than .05. Residual analysis was performed to search for violations of necessary assumptions in multiple regression in terms of linearity, equality of variance, independence of error, and normality.

RESULTS

Study Group

Of the 54 polio subjects considered for participation in the study, 7 were unable to cycle for the following reasons: arthrosis of the hip (1 PPS, 1 non-PPS), insufficient strength (4 non-PPS), and problems in fixing a shoe to the pedal (1 PPS). Furthermore, 2 non-PPS subjects were excluded because they found cycling painful, 1 subject was excluded on the grounds of poor lung function (vital capacity, 1.5L), and 1 subject was unwilling to comply with the protocol. Eventually, 25 PPS subjects, 18 non-PPS subjects, and 12 healthy control subjects, were included. No differences in subject characteristics existed between polio subjects and healthy control subjects.

Data Analysis

To compare the power delivered in the tests and the distribution in power between legs with quadriceps strength and the difference in quadriceps strength between legs, we calculated strength sum scores and asymmetry scores for power and strength, respectively, as the sum of the values of both legs and as the absolute difference between the values of both legs, divided by the sum of both legs. Data are presented for the strongest and the weakest sides, respectively.

To compare the oxygen uptake required at the final increment of the submaximal test, the actual oxygen uptake was expressed as the percentage of the expected oxygen uptake for that power output, based on the Astrand monogram. As a measure of movement economy, delta economy was calculated as the change in VO2/W from the first and the second workload in the submaximal test, because the resting metabolism was not measured and the absolute workloads differed considerably between polio subjects and healthy control subjects.

Statistical analysis was performed with SPSS statistical software package. For normally distributed data, differences between polio subjects and healthy control subjects, and between PPS subjects and stable polio subjects, were analyzed with Student’s t-test and with the Mann-Whitney U test for not normally distributed data. Dependent samples were analyzed with Wilcoxon’s signed-ranks test. Dichotomized variables were analyzed with the chi-square test and ordinal data with the Mann-Whitney U test, with correction for ties. Correlation coefficients were calculated by means of Pearson’s correlation test. An alpha level of p less than .05 was used for all tests of significance. To assess the factors that are independently associated with the oxygen uptake at the final increment of the submaximal test, we used multivariate linear regression with a stepwise forward selection procedure. Exploratory variables used were diagnosis of polio, gender, power delivered at the final increment, lean body mass (body weight × [1 − fat%]), and asymmetry between the legs in power delivered at the final increment.

Table 1: Subject Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Control Subjects (n = 12)</th>
<th>Polio Subjects</th>
<th>Non-PPS (n = 18)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All (n = 43)</td>
<td>PPS (n = 25)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Non-PPS (n = 18)</td>
<td></td>
</tr>
<tr>
<td>Age (yr)</td>
<td>45.3 ± 8.4</td>
<td>45.4 ± 5.8</td>
<td>46.3 ± 5.9</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>3/9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Length (cm)</td>
<td>172.0 ± 7.4</td>
<td>167.5 ± 6.8</td>
<td>168.4 ± 7.0</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>67.0 ± 10.1</td>
<td>70.2 ± 11.7</td>
<td>71.5 ± 15.6</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>28.0 ± 6.5</td>
<td>32.4 ± 7.7</td>
<td>33.1 ± 8.9</td>
</tr>
<tr>
<td>Vital capacity (L)</td>
<td></td>
<td>3.7 ± 0.7</td>
<td>4.0 ± 0.8</td>
</tr>
<tr>
<td>FEV1 (%)</td>
<td></td>
<td>85.6 ± 6.8</td>
<td>81.8 ± 10.7</td>
</tr>
</tbody>
</table>

NOTE. Values are mean ± standard deviation (SD) (range). Missing data: Fat%, 3 (2 controls, 1 non-PPS); lung function, 5 (2 PPS, 3 non-PPS).

Table 2: Residual Effects From Acute Polio and Present Status

<table>
<thead>
<tr>
<th>Polio Symptoms</th>
<th>PPS (n = 25)</th>
<th>Non-PPS (n = 18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at acute polio</td>
<td>1.0 (0.3–10)</td>
<td>2.0 (0.3–5)</td>
</tr>
<tr>
<td>Number of extremities with residual paraplegia</td>
<td>1 (1–3)</td>
<td>1 (1–2)</td>
</tr>
<tr>
<td>Residual paraplegia in 1 leg/2 legs</td>
<td>22/3</td>
<td>17/1</td>
</tr>
<tr>
<td>Walking distance in early adulthood*</td>
<td>4 (3–4)</td>
<td>4 (2–4)</td>
</tr>
<tr>
<td>Present status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of clinically affected extremities</td>
<td>1 (1–3)</td>
<td>1 (1–3)</td>
</tr>
<tr>
<td>Clinical involvement of 1 leg/2 legs</td>
<td>19/6</td>
<td>13/5</td>
</tr>
<tr>
<td>Present walking distance*</td>
<td>2 (1–4)</td>
<td>3.5 (1–4)</td>
</tr>
</tbody>
</table>

NOTE. Values are median (range). *Walking distance was classified in 4 categories: <250m; 250m–1km; 1–5km; >5km. **p < .05, Mann-Whitney U test with correction for ties (PPS vs non-PPS); ††p < .001, Wilcoxon’s signed-rank test (present walking distance vs walking distance after recovery).
between the groups (table 1). Nor was there any difference between the age at onset of acute polio and the number of extremities with residual paresis between PPS and non-PPS subjects. In all subjects, at least 1 leg showed signs of clinical involvement. Both groups of polio subjects did not differ significantly with regard to the use of assistive devices or braces. Except for 1 PPS subject who needed a cane, all polio subjects walked inside their houses without assistive devices. When walking outside, as was performed by all subjects, 5 PPS and 4 non-PPS subjects used a cane or a crutch; 1 non-PPS subject used 2 crutches. A knee-ankle-foot orthosis was used by 7 subject (6 PPS, 1 non-PPS), and customized shoes by 18 subjects (6 PPS, 1 non-PPS). PPS subjects reported a significantly reduced walking ability, compared with the non-PPS subjects, as well as compared with their own walking ability in early adulthood (table 2). The level of physical activity did not differ significantly between groups. About half of all polio subjects (13 PPS, 10 non-PPS) performed exercise once or twice a week (swimming, 10; disabled sports, 5; cycling, 3; cycling and swimming, 2; fitness, 2; tennis, 1; jogging, 1). In the control group, 5 subjects exercised once or twice a week (tennis, 2; fitness, 2; cycling and swimming, 1) and 2 subjects exercised more than 2 times a week (cycling and running, 1; aerobics and fitness, 1).

Submaximal Exercise Capacity

The power output and oxygen uptake at the final increment were significantly lower in the polio subjects than in the control subjects (table 3). The polio subjects achieved their previously set target heart rate values in response to the final increment on the ergometer. In contrast, the control subjects achieved heart rates that were significantly higher than the target values in response to the final increment. This difference occurred because the control subjects had a higher increment between steps in the protocol. To improve comparability, adjusted values for power output and \( V_{\text{O}_2} \) at the final increment were calculated for the control subjects as the intermediate values of the final 2 increments, which would reflect a 25-watt increment, as imposed on the polio subjects. The adjusted values for power output and \( V_{\text{O}_2} \) at the final increment were still 45% \( (p < .005) \) and 18% \( (p < .05) \) higher in the control subjects than in the polio subjects, respectively. Because the difference in \( V_{\text{O}_2} \) between polio subjects and control subjects might result mainly from the difference in power output, \( V_{\text{O}_2} \) was also expressed as a percentage of expected \( V_{\text{O}_2} \). In control subjects, the oxygen uptake at the final increment did not differ from the expected \( V_{\text{O}_2} \). However, \( V_{\text{O}_2} \) at the final increment in the polio subjects was 18% higher than expected \( (p < .005) \) (table 3). At the final increment, the adjusted respiratory exchange ratio (RER) was significantly higher in the polio subjects, compared with the control subjects.

A comparison of cardiorespiratory parameters between control subjects and polio subjects was made at 50 watts because all subjects cycled at this workload except 2 polio subjects and 1 control subject whose increments did not contain this workload step. At 50 watts, heart rate, \( V_{\text{E}} \), and RER values were all significantly higher in the polio subjects than in the control subjects, whereas \( V_{\text{O}_2} \) did not differ (table 4).

### Table 3: Power Output at the Final Increment and Cardiorespiratory Parameters

<table>
<thead>
<tr>
<th>Exercise Parameters</th>
<th>Control Subjects (n = 12)</th>
<th>Polio Subjects</th>
<th>Non-PPS (n = 18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Power output (W)</td>
<td>142 ± 30 (^a)</td>
<td>84 ± 30</td>
<td>88 ± 33</td>
</tr>
<tr>
<td>Target heart rate (beats/min)</td>
<td>154 ± 7</td>
<td>154 ± 6</td>
<td>154 ± 6</td>
</tr>
<tr>
<td>Achieved heart rate (beats/min)</td>
<td>162 ± 12 (^d)</td>
<td>153 ± 14</td>
<td>155 ± 12</td>
</tr>
<tr>
<td>( V_{\text{O}_2} ) (L/min)</td>
<td>2.03 ± 0.35 (^b)</td>
<td>1.54 ± 0.40</td>
<td>1.65 ± 0.37</td>
</tr>
<tr>
<td>( V_{\text{O}_2} ) (% of expected) (L/min)</td>
<td>101 (96–114) (^c)</td>
<td>118 (88–163)</td>
<td>119 (96–163)</td>
</tr>
<tr>
<td>( V_{\text{E}} ) (L/min)</td>
<td>58 ± 11 (^f)</td>
<td>48 ± 13</td>
<td>51 ± 13</td>
</tr>
<tr>
<td>RER (L/min)</td>
<td>1.00 ± 0.05 (^g)</td>
<td>1.02 ± 0.06</td>
<td>1.03 ± 0.06</td>
</tr>
</tbody>
</table>

**NOTE.** Values are mean ± SD except for \( V_{\text{O}_2} \) (% of expected), which is median (range). Missing data: heart rate, 1 (recording failure; PPS subject); power, 1 (recording failure; PPS subject).

\(^a\) Adjusted values were calculated as the mean of the final and the previous workload, because achieved heart rate was significantly above target heart rate.

\(^b\) \( p < .05 \) (control subjects vs polio subjects; Student’s t test).

\(^c\) \( p < .005 \) (control subjects vs polio subjects; Mann-Whitney U test).

\(^d\) \( p < .005 \) (control subjects vs all polio subjects; Student’s t test).

\(^e\) \( p < .005 \) (achieved heart rate vs target heart rate; paired t test).

### Table 4: Cardiorespiratory Parameters at 50-Watt Steady-State Cycling

<table>
<thead>
<tr>
<th>Exercise Parameters</th>
<th>Control Subjects (n = 12)</th>
<th>Polio Subjects</th>
<th>Non-PPS (n = 17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Power output (W)</td>
<td>54 ± 3</td>
<td>52 ± 4</td>
<td>53 ± 5</td>
</tr>
<tr>
<td>( V_{\text{O}_2} ) (L/min)</td>
<td>1.08 ± 0.12</td>
<td>1.11 ± 0.17</td>
<td>1.14 ± 0.20</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>112 ± 17 (^*)</td>
<td>133 ± 17</td>
<td>134 ± 17</td>
</tr>
<tr>
<td>( V_{\text{E}} ) (L/min)</td>
<td>28 ± 3 (^*)</td>
<td>34 ± 7</td>
<td>34 ± 9</td>
</tr>
<tr>
<td>RER</td>
<td>.85 ± .04 (^*)</td>
<td>.95 ± .07</td>
<td>.95 ± .06</td>
</tr>
</tbody>
</table>

**NOTE.** Values are mean ± SD. Missing data: technical failures, 2 (power recording in 1 PPS, heart rate recording in 1 PPS).

\(^*\) \( p < .005 \) (controls vs all polio subjects; Student’s t test).
We found no differences for any value between PPS subjects and non-PPS subjects, whether at target workload or at 50 watts. Nor did we find any differences between exercising subjects and nonexercising subjects.

Quadriceps Strength and Maximal Short-Term Power

The maximal voluntary force of the quadriceps muscles of the weakest side and the maximal short-term power produced by the weakest leg was significantly reduced, by 64% and 61%, respectively, in polio subjects compared with control subjects. Although all strength and power values were lower by 16% to 21% and 4% to 5%, respectively, in PPS subjects compared with non-PPS subjects, the differences were not significant (table 5).

The strength of both quadriceps muscles added together correlated significantly with the measured power output achieved at the final increment ($r = .64$, $p < .005$). The asymmetry in quadriceps strength between legs correlated significantly with the asymmetry in power delivery between legs in the final increment ($r = .86$, $p < .005$). The maximal short-term power correlated significantly with the power at the final increment ($r = .76$, $p < .005$). The asymmetry in maximal short-term power between legs was reflected by the asymmetry in power at the final increment ($r = .92$, $p < .005$) (fig 1).

In the submaximal exercise test, the polio subjects differed considerably in the contribution of each leg to the power delivered both at 50 watts and at the final increment. When cycling at 50 watts, the polio subjects, as a result of a reduced maximal short-term power, used their legs at 16% of their maximal capacity, which was significantly higher than the 10% found in the control subjects (table 6). However, at the final increment, which was determined by the target heart rate, the power delivered as the percentage of maximal short-term power did not differ between polio subjects and control sub-

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**Table 5: Quadriceps Strength and Maximal Short-Term Power**

<table>
<thead>
<tr>
<th>Strength and Power</th>
<th>Control Subjects ($n = 12$)</th>
<th>Polio Subjects</th>
<th>Non-PPS ($n = 18$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quadriceps strength strongest side (N)</td>
<td>500 ± 112</td>
<td>438 ± 160</td>
<td>482 ± 198</td>
</tr>
<tr>
<td>Quadriceps strength weakest side (N)</td>
<td>467 ± 119</td>
<td>169 ± 148*</td>
<td>192 ± 165</td>
</tr>
<tr>
<td>Quadriceps strength sum (N)</td>
<td>966 ± 230</td>
<td>607 ± 260*</td>
<td>673 ± 318</td>
</tr>
<tr>
<td>Quadriceps strength asymmetry</td>
<td>.03 (.00–.11)</td>
<td>.50 (.09–1.0)†</td>
<td>.39 (.09–1.00)</td>
</tr>
<tr>
<td>Maximal ST power strongest side (W)</td>
<td>297 ± 80</td>
<td>257 ± 85</td>
<td>265.1 ± 96.3</td>
</tr>
<tr>
<td>Maximal ST power weakest side (W)</td>
<td>284 ± 81</td>
<td>111 ± 82*</td>
<td>113.7 ± 90.5</td>
</tr>
<tr>
<td>Maximal ST power both sides (W)</td>
<td>581 ± 160</td>
<td>386 ± 146*</td>
<td>378.8 ± 166.2</td>
</tr>
<tr>
<td>Maximal ST power asymmetry</td>
<td>2.6 (0.5–3.9)</td>
<td>39.4 (4.7–112.8)†</td>
<td>35.6 (7.2–112.8)</td>
</tr>
</tbody>
</table>

NOTE. Values are mean ± SD or median (range). No strength and power data were available for 2 PPS subjects (not willing for personal reasons). Missing data for muscle strength in 1 control subject (painful knee).

Abbreviation: ST, short-term.

* $p < .005$ (control subjects vs all polio subjects; Student’s t test).

† $p < .005$ (control subjects vs all polio subjects; Mann-Whitney U test).
Abbreviation: ST, short-term.

With 1 passive leg were excluded from analysis because no percentage of maximal power could be calculated.

NOTE. Values are mean ± SD. Missing data: 2 not cycled at 50W, 1 technical problem, 2 no maximal ST power measured. Four subjects cycling with 1 passive leg were excluded from analysis because no percentage of maximal power could be calculated. Abbreviation: ST, short-term.

*p < .005 (control subjects vs polio subjects; Student’s t test).

*p < .05.

Subjects—who even performed above their target heart rate—being 24% and 26%, respectively. In the polio subjects, the absolute power delivered at the final increment by the weakest side was over 50% less than the absolute power delivered by the strongest side (57 ± 22W vs 25 ± 14W). However, when expressed as a percentage, both the weakest and the strongest legs of the polio subjects delivered 24% of the maximum power available from that leg. No differences for any value were found between PPS subjects and non-PPS subjects. In the exercising polio subjects, the relative power at the final increment was 26% ± 12% for the weakest leg and 25% ± 8% for the strongest leg, compared with, respectively, 21% ± 7% and 23% ± 6% in the nonexercising polio subjects; these differences were not significant (p = .11, .41, respectively). No differences in relative power output were observed between exercising and nonexercising control subjects.

Movement Economy and Asymmetry

In the submaximal exercise test, delta economy (ΔVO2/W) from the first to the second workload step correlated significantly with the asymmetry in power delivered by both legs (r = .46, p < .005) (fig 2). In some subjects the asymmetry score was even larger than 1, which resulted from a negative net power for the weakest, usually severely paralyzed, passively moved leg. In a linear regression model, 87% of variance in VO2 at the final increment of the submaximal exercise test could be explained by the power delivered at the final increment, the asymmetry between legs in power delivered, and the lean body mass, whereas gender and diagnosis of polio were not independent contributors (table 7). The model predicted that for a subject who generates all power with 1 leg (asymmetry score = 1.0) the additional oxygen uptake will be .30 L/min.

DISCUSSION

The present study shows that the reduced leg power output of the polio subjects in the final increment of the exercise test, aimed at an exercise intensity of 80% of the HRR, was predominantly the effect of reduced leg muscle function. There are 2 observations that support this conclusion. First, the power output at the final increment correlated with the isometric strength of the quadriceps muscles, which is in agreement with the findings of Willén et al. The power output at the final increment correlated even better with the maximal short-term power output. This is not surprising because the paresis in polio subjects varies greatly in number and extent of muscles involved, and measurements of maximal short-term power of the legs provide an overall measure for leg capacity. Moreover, unlike isometric strength, maximal short-term power and power measurements in incremental tests are both dynamic measurements obtained in equal test positions.

Table 6: Between-Leg Power Output Distribution in Submaximal Cycling in 34 Polio Subjects

<table>
<thead>
<tr>
<th>Leg Power Output</th>
<th>Control Subjects (n = 12)</th>
<th>Polio Subjects (n = 34)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Power strongest side (W)</td>
<td>29 ± 3</td>
<td>38 ± 7*</td>
</tr>
<tr>
<td>Power weakest side (W)</td>
<td>24 ± 3</td>
<td>14 ± 7*</td>
</tr>
<tr>
<td>Power strongest side (% of maximal ST power)</td>
<td>11 ± 4</td>
<td>17 ± 6*</td>
</tr>
<tr>
<td>Power weakest side (% of maximal ST power)</td>
<td>9 ± 2</td>
<td>15 ± 8*</td>
</tr>
</tbody>
</table>

NOTE. Four subjects were excluded from analysis because of missing data (technical problem with recording power in 1 polio subject; no fat measurements in 2 controls and 1 polio subject). Factors used in regression analysis: diagnosis of polio, gender, power delivered at the final increment (Power_fiinal), lean body mass (body weight × [1 – fat%]), asymmetry between the legs in power delivered at the final increment (Power asymmetry_fiinal), lean body mass (body weight × [1 – fat%]), asymmetry between the legs in power delivered at the final increment (Power asymmetry_fiinal)

Abbreviation: CI, confidence interval.

*p < .001.

Fig 2. Correlation between asymmetry in power at the final increment and ΔVO2/W between the first 2 workload steps of the submaximal test for the polio subjects. Legend: ○, PPS subjects; □, non-PPS subjects. The correlation coefficient is significant (p < .01).

Table 7: Regression Model for Oxygen Uptake in Steady-State Cycling at the Final Increment in 51 Subjects

<table>
<thead>
<tr>
<th>Predictors</th>
<th>B (95% CI)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO2, (L/min)</td>
<td></td>
<td>.515</td>
</tr>
<tr>
<td>Power_fiinal</td>
<td>.095 (.090 to .101)</td>
<td>.000</td>
</tr>
<tr>
<td>Lean body mass (kg)</td>
<td>.014 (.007 to .022)</td>
<td>.000</td>
</tr>
<tr>
<td>Power asymmetry_fiinal</td>
<td>.265 (.147 to .442)</td>
<td>.000</td>
</tr>
</tbody>
</table>

NOTE. Four subjects were excluded from analysis because of missing data (technical problem with recording power in 1 polio subject; no fat measurements in 2 controls and 1 polio subject). Factors used in regression analysis: diagnosis of polio, gender, power delivered at the final increment (Power_fiinal), lean body mass (body weight × [1 – fat%]), asymmetry between the legs in power delivered at the final increment (Power asymmetry_fiinal). Abbreviation: CI, confidence interval.

*p < .001.
Second, at the final increment the polio subjects used the same percentage of their maximum short-term power. This finding suggests that exercise capacity is the result of a reduction in the maximum leg muscle power, of which 25% can be used during sustained exercise at an exercise level of 80% of HRR. Interestingly, in the polio subjects the relative power output of the weakest and the strongest leg was also the same at the final increment. Apparently, the distribution of power output between legs is dependent on the muscular capacity of the legs. This observation is also supported by the strong correlation of the asymmetry of power generated at the final increment with the asymmetry in isometric quadriceps strength and with the asymmetry in maximal short-term power. These findings are similar to those previously reported for patients with unilateral disuse atrophy after immobilization of 1 leg. This finding is surprising because the muscles of polio patients are not merely atrophied, but often show extensive abnormalities, such as a reduced number of motor units that are greatly enlarged through collateral sprouting with muscle fiber hypertrophy.

The present study also showed that one cannot conclude from either elevated heart rates or low oxygen uptake alone that cardiorespiratory conditioning is reduced. One has to take into account the muscle capacity in terms of strength or power. It has been shown from experiments with 1- and 2-leg cycling that the heart rate is elevated if the same oxygen uptake is achieved with a smaller muscle mass. The observed difference in heart rate between polio subjects and control subjects at a workload of 50 watts may result from this phenomenon, and does not necessarily imply a worse cardiorespiratory condition in the polio subjects. Moreover, given that maximal stroke volume and arteriovenous oxygen difference in polio subjects would be smaller if their cardiorespiratory condition was less significantly than the relative output in the exercising polio subjects, physical activity may be relevant in maintaining cardiorespiratory condition. Other arguments for a low aerobic capacity of polio subjects have been found in muscle biopsy studies of polio subjects, which reported lower concentrations of some oxidative enzymes, compared with control subjects, whereas other oxidative enzymes were within normal ranges. The clinical significance of these findings has been debated by others.

Although we did not measure any enzymatic activities, our results do not suggest that the actual performance in aerobic exercise is limited significantly because of factors other than a limited muscle mass.

In daily life, many tasks, such as walking, impose a certain more or less absolute workload on the individual. If a person’s capacity is reduced as a consequence of muscle paresis from polio, performing these activities will require more of the available capacity. The relative power output of the polio subjects in cycling at 50 watts was higher than the relative power output of the control subjects, and this higher relative workload was also reflected by the higher VE and RER values. That the muscles of polio subjects are commonly used at relatively higher loads in daily life is also supported by findings of extensive type 1 fiber predominance and fiber hypertrophy. Another factor that may have profound consequences for performing activities in daily life is the finding that for a given power output, the energy cost (in terms of V\textsubscript{O\text{2}}) was higher than expected in the polio subjects. In the multivariate regression analysis, asymmetry in power (or muscle strength) between legs appeared to be an independent explanatory factor. An increased asymmetry results in a higher V\textsubscript{O\text{2}} and consequently indicates a reduced movement economy.

**CONCLUSION**

In this study, PPS subjects experienced more limitations in walking than stable polio subjects, which could not be explained by limitations with sustained activities caused by a worse cardiorespiratory condition. Although, in general, the results of the PPS subjects were worse than those of the stable polio subjects, no objective significant difference was found between the 2 polio groups. Several explanations can be suggested—for example, the large variation in values between subjects found in both groups or the fact that other locomotory overuse symptoms, such as pain, may play a role that was not taken into account. Another point that must be considered is the difficulty in making the clinical diagnosis of PPS, which lacks clear objective criteria because the patients were not diagnosed on the basis of objective alterations over time. Therefore, the relevance of the results is not that PPS subjects do not differ from stable polio subjects, but that the submaximal work capacity of many subjects with residuals from polio is severely reduced, mainly because of their reduced muscular capacity. Together with the elevated energy cost for a given power output, it is understandable that fatigue and walking difficulties are major complaints of many polio survivors because they imply a higher relative effort. Results from cycling cannot be simply generalized to walking because muscle loads will be task dependent and the pattern of paresis may affect walking differently than cycling. Further research should aim at measuring the actual energy cost and muscle load of physical activities in more valid circumstances, that is in performing functional activities, and preferably in a daily life environment.

**References**


Suppliers
c. HBM Benelux, Plettenburg 7, 3439 LW Nieuwegein, The Netherlands.
e. Polar Electro OY, Professorintie 5, FIN-90440 Kempele, Finland.
f. Version 10.0.5; SPSS Inc, 233 S Wacker Dr, 11th Fl, Chicago, IL 60606.