Aerobic Conditioning in Mild Asthma
Decreases the Hyperpnea of Exercise
and Improves Exercise and Ventilatory Capacity*

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Study objective: To determine the effect of an aerobic conditioning program on fitness, respiratory physiology, and resting lung function in patients with mild asthma.

Design: Prospective cohort study.

Setting: Outpatient rehabilitation facility.

Methods: Five patients with mild intermittent asthma and five normal control subjects completed a 10-week aerobic conditioning program. Pulmonary function studies and noninvasive cardio-pulmonary exercise tests were performed before and after the conditioning program.

Results: After aerobic conditioning, there were significant gains in maximum oxygen consumption ($V_{\text{O}}_{2\text{max}}$: 22.73 mL/kg/min vs 25.29 mL/kg/min, $p = 0.01$, asthma; 22.94 mL/kg/min vs 27.85 mL/kg/min, $p = 0.03$, control) and anaerobic threshold (0.99 L/min vs 1.09 L/min, $p = 0.03$, asthma; 0.89 L/min vs 1.13 L/min, $p = 0.01$, control) in both groups. Although FEV$_1$ was unchanged, the maximum voluntary ventilation (MVV) improved in the asthma group (96.0 L/min vs 108.2 L/min, $p = 0.08$, asthma; 134.0 L/min vs 131.2 L/min, $p = 0.35$, control). During exercise, minute ventilation ($V_e$) for each level of work was decreased in the asthma group after conditioning, while little change occurred in the control group (68.48 L/min vs 51.70 L/min at initial $V_{\text{O}}_{2\text{max}}$, $p = 0.02$, asthma; 65.82 L/min vs 63.12 L/min at initial $V_{\text{O}}_{2\text{max}}$, $p = 0.60$, control). A significant decrease in the ventilatory equivalent ($V_e$/oxygen consumption, 40.8 vs 30.4 at $V_{\text{O}}_{2\text{max}}$, $p = 0.02$, asthma; 37.2 vs 35.8 4 at $V_{\text{O}}_{2\text{max}}$, $p = 0.02$, control) and the dyspnea index ($V_e$/MVV) at submaximal (0.44 vs 0.38, $p = 0.05$, asthma; 0.32 vs 0.38, $p < 0.01$, control) and maximal exercise (0.72 vs 0.63, $p = 0.03$, asthma; 0.49 vs 0.62, $p = 0.02$, control) occurred in the asthma group.

Conclusions: Exercise rehabilitation improves aerobic fitness in both asthmatic and nonasthmatic participants of a 10-week aerobic fitness program. Additional benefits of improved ventilatory capacity and decreased hyperpnea of exercise occurred in patients with mild asthma.

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Key words: asthma; exercise-induced bronchospasm; rehabilitation

Abbreviations: MVV = maximum voluntary ventilation; $P_{\text{ETCO}}_2$ = end-tidal carbon dioxide pressure; $V_{\text{CO}}_2$ = carbon dioxide production; $V_d/V_t$ = Bohr dead space ratio; $V_e$ = minute ventilation; $V_{\text{O}}_2$ = oxygen consumption; $V_{\text{O}}_{2\text{max}}$ = maximum oxygen consumption; $V_t$ = tidal volume

The physiologic effect of aerobic training in adults with asthma remains to be clearly delineated. The notion that conditioning is beneficial in asthmatics dates to the mid-19th century. ¹ Aerobic conditioning improves fitness and pulmonary symptoms in individuals with asthma. In children with asthma,
aerobic conditioning improves resting lung function, dyspnea scores, and social development scores, and decreases exercise-induced bronchospasm and peak expiratory flow variability.\textsuperscript{2–5} In adults with asthma, conditioning decreases exercise-induced bronchospasm and improves exercise tolerance and quality of life.\textsuperscript{6–9} The fundamental basis of these effects of aerobic conditioning in adults with asthma remains unclear. Improvement in peak expiratory flow variability and decreased medication use in children suggests a decline in airway inflammation; however, this has not been demonstrated directly. Adults and children increase their exercise tolerance after training without demonstrable changes in airway obstruction.\textsuperscript{8–13} Individuals with asthma are limited during exercise by a low maximum voluntary ventilation (MVV) and a high minute ventilation (Ve) for a particular workload.\textsuperscript{6,14,15} The MVV is decreased in individuals with asthma, either directly from fixed airflow obstruction or from increased airway hyperresponsiveness that causes a decline in airway conductance during the MVV maneuver.\textsuperscript{16} Ventilatory efficiency is reduced, reflected by an increased Ve for a particular workload.\textsuperscript{5} These factors are described together in the dyspnea index (Ve/MVV). In mild to moderate asthma, the dyspnea index is elevated, although not to the degree that would conventionally define ventilatory limitation.\textsuperscript{14,15} This increase in the degree of dyspnea during aerobic activities may affect exercise tolerance and lead to deconditioning.\textsuperscript{6,11,13,14} Despite these barriers, many individuals with asthma take part in aerobic activities, even at the highest levels of competition.\textsuperscript{17}

To determine the effect of aerobic conditioning on exercise tolerance and pulmonary physiology, we prospectively enrolled a group of patients with mild asthma and a group of normal control subjects in a 10-week conditioning program. We hypothesized that an aerobic exercise program would have beneficial effects on exercise tolerance, fitness, ventilatory efficiency, and lung function.

**Materials and Methods**

**Subjects**

A group of nine adult patients with mild intermittent asthma as defined by the National Asthma Education and Prevention Program, Expert Panel Report 2\textsuperscript{18} were recruited for this study. The asthma group was restricted to nonsmoking, sedentary individuals who required only intermittent short-acting \( \beta_2 \)-agonist therapy in the 3 months preceding the study. Seven sedentary individuals without a history of asthma were recruited for the control group. Of these 16 individuals initially screened for the study, 6 individuals were excluded during the run-in period due to poor compliance with the exercise regimen and were not included in the study. Five individuals in each group entered the 10-week conditioning program. The study protocol was approved by the human subjects review committee of the Maine Medical Center.

**Lung Function and Exercise Testing**

Baseline lung function testing, including FEV\textsubscript{1} and MVV based on a 12-s trial, was performed on a pneumotachograph (model 6200; SensorMedics; Yorba Linda, CA), according to American Thoracic Society guidelines.\textsuperscript{19} Each participant underwent a noninvasive, progressive exercise trial on a cycle ergometer (Ergoline 580S; SensorMedics) to maximum oxygen consumption (VO\textsubscript{2max}). Individuals with asthma were instructed not to use short-acting \( \beta_2 \)-agonists during the 4 h preceding the test. The initial workload and rate of progression were selected, such that each participant would reach VO\textsubscript{2max} after approximately 12 min. To standardize the stimulus for exercise-induced bronchospasm, the participants continued to cycle at a power output of 60 W after the maximum workload was achieved for a total of 15 min of exercise. After completing the exercise trial, FEV\textsubscript{1} was obtained at 3, 6, 10, and 15 min after exercise.

Respiratory rate, tidal volume (V\textsubscript{T}), heart rate, Ve, oxygen consumption (VO\textsubscript{2}), carbon dioxide production (VCO\textsubscript{2}), and end-tidal carbon dioxide pressure (PETCO\textsubscript{2}) were measured on a metabolic cart (model 2900; SensorMedics). The anoxic threshold was determined by the V-slope method using the point of divergence of the slopes of V\textsubscript{CO}\textsubscript{2} and VO\textsubscript{2} expressed in terms of VO\textsubscript{2}\textsubscript{max}.\textsuperscript{20} The ventilatory equivalent was calculated by dividing Ve by V\textsubscript{CO}\textsubscript{2}. The dyspnea index at 75% of maximum and at VO\textsubscript{2max} was calculated by dividing the Ve at each level of VO\textsubscript{2} by the measured MVV.\textsuperscript{14,15} Respiratory rate, V\textsubscript{T}, Ve, VCO\textsubscript{2}, and Bohr dead space ratio (VD/VT) were determined at 20% increments of VO\textsubscript{2max}. VD/VT was determined using the PETCO\textsubscript{2} substituted for PaCO\textsubscript{2} in the VD/VT equation.\textsuperscript{20}

**Conditioning Program**

Study participants were enrolled in an aerobic conditioning program consisting of step aerobics three times a week for 10 weeks. The fitness program was supervised by a physical therapist or an exercise physiologist. Both the asthma and control groups exercised together. Each participant learned to measure his or her heart rate at the start of the conditioning program. During each session, heart rate was monitored such that a target heart rate equal to that required for 70% VO\textsubscript{2max} was achieved. During each session, participants attempted to maintain their target heart rate for at least 30 min. Participants with asthma were allowed to use \( \beta_2 \)-agonist therapy as needed during the exercise program. During the fitness program, both groups were asked to maintain a diary of medication use, daytime and nighttime asthma symptoms, and cough.

At the conclusion of the exercise program, lung function and

<table>
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<th>Table 1—Baseline Characteristics</th>
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<td>Characteristics</td>
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<td>(n = 5)</td>
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<tr>
<td>Average age, yr</td>
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<tr>
<td>Sex, male/female</td>
</tr>
<tr>
<td>Average weight, kg</td>
</tr>
<tr>
<td>Average height, cm</td>
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<tr>
<td>History of exercise-induced asthma</td>
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<td>History of atopy</td>
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exercise testing were repeated according to the baseline studies. The postconditioning dyspnea index was calculated at 75% of maximum and at \( \dot{V}O_2 \) based on the measured \( \dot{V}O_2 \) during the postconditioning exercise trial. Respiratory rate, \( V_t \), \( \dot{V}e \), \( \dot{V}co_2 \), and \( V_d/V_t \) were recorded at 20, 40, 60, 80, and 100% of the pretraining \( \dot{V}O_2 \) during the postconditioning exercise trial.

**Statistical Analysis**

Spirometric values (FEV₁, MVV, and decline in FEV₁ after exercise) before and after the conditioning program were compared using a paired t test. The level of significance was based on a two-tailed distribution, except in the case of MVV, in which a one-tailed critical value is justified by prior studies.\(^7,13\) Comparison of physiologic variables during exercise, before and after conditioning, were made using paired \( t \) tests at each level of \( \dot{V}O_2 \).

**RESULTS**

**Participant Characteristics**

The five participants in each group were similar in age, height, weight, and gender (Table 1). All patients in the asthma group reported a history of atopy, and four patients reported a history of exercise-induced bronchospasm. The participants in the asthma group used inhaled short-acting \( \beta_2 \)-agonists (eg, albuterol) on average 2.8 times per week and reported episodic wheezing and occasional cough.

**Spirometry and Respiratory Symptoms**

Baseline lung function in the asthma group was normal, except for a reduction in the MVV. The asthma group demonstrated a postexercise reduction in FEV₁ of 6.1% (range, 0 to 18%; \( p = 0.01 \) vs control). After the 10-week conditioning program, there was no change in FEV₁, FEV₁/vital capacity ratio, or exercise-induced bronchospasm in either group (Table 2), although there was a trend toward improvement in the MVV in the asthma group (Fig 1). There was no significant change in bronchodilator use, daytime or nocturnal asthma symptom scores, or cough after the conditioning program.

**Fitness**

Both groups made significant gains in measures of fitness after the 10-week conditioning program (Table 3). Comparable gains in \( \dot{V}O_2 \)max and anaerobic threshold were realized in the asthma and control groups (Fig 2, 3).

**Respiratory Physiology**

At 75% of maximum and at maximum exercise, the dyspnea index was elevated in the asthma group during the baseline exercise trial (Table 3). After 10 weeks of conditioning, the dyspnea index was significantly reduced at 75% of maximum and maximum exercise in the asthma group, while the dyspnea index rose in the control group (Fig 4, 5). Prior to conditioning, the ventilatory equivalent for oxygen at maximum exercise was elevated in the asthma group, compared to the control group (Table 3). After the

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**Table 2—Spirometric Characteristics Before and After 10 Weeks of Conditioning**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Asthma Group (n = 5)</th>
<th>Control Group (n = 5)</th>
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<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>FEV₁, L</td>
<td>3.10 (0.22)</td>
<td>3.12 (0.30)</td>
</tr>
<tr>
<td>FEV₁, % predicted</td>
<td>97.0 (6.4)</td>
<td>97.8 (8.8)</td>
</tr>
<tr>
<td>Maximum decline in FEV₁</td>
<td>6.1 (7.4)</td>
<td>7.8 (8.5)</td>
</tr>
<tr>
<td>after exercise, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MVV, L/min</td>
<td>96.0 (15.6)</td>
<td>108.2 (10.3)</td>
</tr>
<tr>
<td>MVV, % predicted</td>
<td>83.0 (13.0)</td>
<td>93.6 (10.6)</td>
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*Data are presented as mean (SD).
conditioning program, the ventilatory equivalent at 75% of maximum and maximum exercise decreased significantly in the asthma group, while only minor changes occurred in the control group (Table 3). The maximum values for work rate, $\dot{V}E$, $\dot{V}O_2$, $\dot{V}E/\dot{V}O_2$, $V_t$, respiratory rate, and $V_d/V_t$ before and after the conditioning program are presented in Table 3. During exercise, the $\dot{V}E$ at each level of $\dot{V}O_2$ was reduced in the asthma group after the conditioning program, while little change occurred in the control group (Fig 6). The $V_t$s during exercise were similar for both trials in the asthma and control groups. The respiratory rate at each level of $\dot{V}O_2$ was reduced in both groups after the conditioning program, but the magnitude of this difference was greater in the asthma group (Fig 7). Measured $\dot{V}CO_2$ was reduced for each level of $\dot{V}O_2$ in both groups after the conditioning program; however, the magnitude of this decrease was greater in the asthma group (Fig 8). The $V_d/V_t$ ratio declined during exercise in both groups, and there was no difference between the groups. There was a trend in the partial pressure of $P_{ETCO_2}$ toward an increase in the asthma group after the conditioning program, while no change occurred in the control group (Fig 9).

**Discussion**

This study demonstrates that exercise rehabilitation improves aerobic fitness and decreases the hyperpnea of exercise in patients with mild asthma. After 10 weeks of aerobic conditioning, patients with asthma and a control group composed of nonasthmatic individuals significantly increased their $\dot{V}O_2_{max}$ and increased their anaerobic threshold. While baseline FEV₁ remained unchanged, the asthma group showed an increase in the MVV to within the normal range. After the conditioning program, there was a decrease in $\dot{V}E$ for each level of work that occurred only in the asthma group. There was a reduction in the respiratory rate and a rise in the $P_{ETCO_2}$ during exercise. Conditioning improved the ventilatory efficiency in the asthma group, reflected by a decrease in the ventilatory equivalent for oxygen and a reduction in the dyspnea index at 75% of maximum and maximum exercise.
submaximal and maximal exercise. These results show that in addition to improving fitness, aerobic conditioning increases ventilatory capacity and decreases the hyperpnea of exercise in patients with mild asthma.

Respiratory symptoms may cause asthmatics to avoid exercise, resulting in aerobic fitness that is below that of their peers.\textsuperscript{6,13} Disease severity judged by FEV\textsubscript{1} is not the primary determinant of fitness in individuals with asthma, and aerobic capacity can improve without a change in resting lung function.\textsuperscript{11,21} Exercise tolerance is reduced primarily from an increased sensation of dyspnea during exercise.\textsuperscript{6,14,15} For a given workload, deconditioned individuals with asthma maintain higher \( \dot{V}e \) than similarly deconditioned control subjects without asthma.\textsuperscript{6,22} The capacity to increase \( \dot{V}e \) may also be diminished in individuals with asthma. The summation of these physiologic parameters is described in the dyspnea index, which is increased in individuals with asthma during exercise and likely represents the primary barrier to aerobic activities in most asthmatics.\textsuperscript{14,15} These data demonstrate that an aerobic conditioning program improves the MVV and decreases the \( \dot{V}e \) at a given workload, resulting in a decreased dyspnea index and ventilatory equivalent after conditioning in patients with mild asthma.

The capacity to increase \( \dot{V}e \), as quantified by the MVV, may be limited in individuals with asthma. The MVV may be diminished as a direct consequence of airflow obstruction, but may be further reduced due to airway hyperresponsiveness.\textsuperscript{16} The MVV/FEV\textsubscript{1} ratio is a reflection of increased airway hyperrespon-
siveness, and the MVV maneuver causes a decrease in airway conductance in individuals with asthma, but not normal subjects.\textsuperscript{16} Aerobic conditioning improves the MVV in patients with asthma,\textsuperscript{7,13} although the mechanism of this improvement remains unclear. Increased respiratory muscle strength has been cited as a possible mechanism for improvement in the MVV after conditioning\textsuperscript{7}; however, this mechanism is not supported by the present data, since a similarly deconditioned control group did not show comparable gains in MVV. Improvement in the MVV could also reflect subtle changes in lung function or airway reactivity not detected by the FEV\textsubscript{1} maneuver. In children, conditioning reduces air trapping.
placing the diaphragm in a more advantageous position mechanically. Conditioning also decreases peak expiratory flow variability, asthma symptom scores, and medication use in children, suggesting a decrease in airway inflammation; however, it is unclear how conditioning could affect airway inflammation. In the present study, there was no evidence of a change in disease activity, likely reflecting the mild intermittent nature of the disease in our study population.

Individuals with asthma maintain a high $V_{E}$ during exercise. These data and previous studies demon-
strate that conditioning decreases \( V_e \) per level of work in patients with asthma.\(^6\)\(^,\)\(^9\)\(^,\)\(^22\) Aerobic conditioning results in a modest decrease in \( V_e \) in all subjects through an improvement in anaerobic threshold; however, the magnitude of this decrease is greater in individuals with asthma,\(^6\) and these data show a reduction in \( V_e \) prior to the anaerobic threshold. \( V_e \) was reduced by a decline in the respiratory rate with maintenance of the preconditioning \( V_t \) and an increase in the \( \text{PETCO}_2 \). No change was noted in the \( V_d/V_t \). These data suggest that a blunted ventilatory response to exercise occurs in response to conditioning in individuals with mild asthma. Reductions in the ventilatory response to exercise have also been noted in well-trained athletes, likely representing an adaptation to conditioning.\(^23\) Further study is necessary to determine if a change in central respiratory drive occurs in response to conditioning.

Figure 8. \( V_{\text{CO}_2} \) at each level of \( VO_2 \), expressed as a percentage of initial \( VO_2\text{max} \) before (▲) and after (△) 10 weeks of aerobic conditioning in patients with mild intermittent asthma and in normal control subjects.
$V^\circ_e$ is an important determinant of the amount of exercise-induced bronchospasm. The severity of exercise-induced bronchospasm was similar in both trials, reflecting a similar maximum $V^\circ_e$ in both the preconditioning and postconditioning trials. These data are consistent with the results of other conditioning programs that show no change in the severity of exercise-induced bronchospasm after a postcon-

**Figure 9.** $\text{PETCO}_2$ at each level of $\text{VO}_2$, expressed as a percentage of initial $\text{VO}_2\text{max}$ before (▲) and after (△) 10 weeks of aerobic conditioning in patients with mild intermittent asthma and in normal control subjects.

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ditioning maximal exercise test in which the participants achieved a higher level of work.25–27 The amount of exercise-induced bronchospasm declines after conditioning if the amount of work is held constant in the postconditioning exercise test due to the lower resultant VE.10,28 If the total VE is kept constant in the postconditioning trial, a small improvement occurs in the amount of exercise-induced bronchospasm.7,8 These data reinforce the importance that a reduction in VE and an improvement in ventilatory efficiency play in the ability of patients with asthma to exercise effectively.

Exercise rehabilitation in patients with mild intermittent asthma improves aerobic fitness and ventilatory efficiency. Aerobic conditioning is well tolerated and leads to fitness gains similar to those in nonasthmatic individuals. An improvement in ventilatory capacity and a decrease in the hyperpnea of exercise that was present prior to conditioning in asthmatics occurred after aerobic conditioning in patients with asthma, but not in normal control subjects. We conclude that physical training results in beneficial adaptations that allow individuals with mild asthma to participate comfortably in aerobic activities. Further study is necessary to determine the underlying basis of these adaptations.

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