Effect of supplemental oxygen on supramaximal exercise performance and recovery in cystic fibrosis

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Shah, Ashish R., Thomas G. Keens, and David Gozal. Effect of supplemental oxygen on supramaximal exercise performance and recovery in cystic fibrosis. J. Appl. Physiol. 83(5): 1641–1647, 1997.—The effects of supplemental O2 on recovery from supramaximal exercise and subsequent performance remain unknown. If recovery from exercise could be enhanced in individuals with chronic lung disease, subsequent supramaximal exercise performance could also be improved. Recovery from supramaximal exercise and subsequent supramaximal exercise performance were assessed after 10 min of breathing 100% O2 or room air (RA) in 17 cystic fibrosis (CF) patients [25 ± 10 (SD) yr old, 53% men, forced expired volume in 1 s = 62 ± 21% predicted] and 17 normal subjects [25 ± 8 yr old, 59% men, forced expired volume in 1 s = 112 ± 15% predicted]. Supramaximal performance was assessed as the work of sustained bicycling at a load of 130% of the maximum load achieved during a graded maximal exercise. Peak minute ventilation (Ve) and heart rate (HR) were lower in CF patients at the end of each supramaximal bout than in controls. In CF patients, single-exponential time decay constants indicated faster recovery of HR (τHR = 86 ± 8 and 73 ± 6 s in RA and O2, respectively, P < 0.01). Similarly, fast and slow time constants of two-exponential equations providing the best fit for ventilatory recovery were improved in CF patients during O2 breathing (τVe = 132.1 ± 10.5 vs. 82.5 ± 10.4 s; τVe = 880.3 ± 300.1 vs. 368.6 ± 107.1 s, P < 0.01). However, no such improvements occurred in controls. Supramaximal performance after O2 improved in CF patients (109 ± 6% of the 1st bout after O2 vs. 94 ± 6% in RA, P < 0.01). O2 supplementation had no effect on subsequent recovery in controls (97 ± 3% in O2 vs. 93 ± 3% in RA). We conclude that supplemental O2 after a short bout of supramaximal exercise accelerates recovery and preserves subsequent supramaximal performance in patients with CF.

obstructive lung disease; gas exchange

Materials and Methods

Subjects. Seventeen patients with CF and 17 normal age- and sex-matched controls were studied. Patients with CF were recruited from the Division of Pediatric Pulmonology at Childrens Hospital Los Angeles. All patients had pulmonary symptoms, signs, and radiological changes consistent with CF and were diagnosed by pilocarpine iontophoretic sweat chloride tests. All patients with CF were receiving pancreatic enzymes, vitamin supplements, and bronchodilators. Some CF patients were also receiving antibiotics at the time of the study. Patients with CF were studied when clinically stable, either as outpatients or at the end of a 2- to 3-wk hospital stay.

Control subjects were healthy adults recruited from members of the hospital staff and their families. No control subjects smoked or had evidence of cardiopulmonary disease.

Informed consent was obtained from each subject. The study was approved by the Institutional Review Board of Childrens Hospital Los Angeles.

Pulmonary function testing. All subjects underwent pulmonary function testing in the pulmonary function laboratory at the Childrens Hospital Los Angeles, which is located at sea level (mean atmospheric pressure 751 Torr). All measurements for each subject were performed on the same day. The
vital capacity and its subdivisions were measured from a slow exhalation with a wedge spirometer (model 3000, Med-Science, St. Louis, MO). The best forced vital capacity, forced expiratory volume in 1 s, mean forced expiratory flow during the middle half of forced vital capacity, and maximal expiratory flow-volume curves obtained from forced expiration into the wedge spirometer were selected and corrected for body temperature, pressure saturated (bTPS). Functional residual capacity was measured with a body pressure plethysmograph (2800 Autobox, Sensormedics, Yorba Linda, CA) by the method of DuBois et al. (7). Residual volume and total lung capacity were calculated, and the ratio of residual volume to total lung capacity was determined from the actual values. Individual test results were analyzed and considered abnormal if they were greater than ±2 SD from available reference values appropriate for age, height, and gender (20).

Nutritional assessment. Nutritional assessment consisting of anthropometric measurements and calculation of lean body mass and percentage of body fat was performed. Anthropometry included the triceps, biceps, anterior superior iliac, and subscapular skinfolds using Lange skinfold calipers. The average of six consecutive measurements was used in calculations. Midarm circumference, height, and weight were also measured. Lean body mass was derived from standard equations (5, 9, 14, 26).

Exercise testing. During all exercise tests, subjects breathed through a mouthpiece from which inspired and expired gas concentrations were continuously analyzed with rapid-through a mouthpiece from which inspired and expired gas concentrations (5, 9, 14, 26).

Graded maximal exercise testing. This initial test was designed to establish V˙O2max of each subject and allow determination of the exercise load to be applied during supramaximal exercise. After assessment of baseline cardiorespiratory measures at rest, the test consisted of pedaling an electronic exercise ergometer while breathing room air or 100% O2 through the mouthpiece from a 100-liter Douglas bag. After 9 min, subjects began pedaling at zero load at a frequency of ~70 rpm while still breathing the predetermined gas mixture. At 10 min the load was increased to 130% of V˙O2max, and all subjects were switched to room air. The test was discontinued when subjects could not maintain a pedaling frequency ≥ 40 rpm.

Within 2 wk of completion of this exercise protocol, subjects underwent the second series of supramaximal exercise testing. The gas mixture not given for the first series was used in the second series.

Supramaximal performance (in kpm) was assessed as the time (in minutes) subjects could sustain bicycling at 130% V˙O2max multiplied by the workload (kpm/min). The second exercise bout was expressed as a percentage of the first supramaximal exercise bout in each series.

Data analysis. Unpaired t-tests were used to compare clinical characteristics and pulmonary function values between CF patients and control subjects. Unpaired t-tests were used to compare differences in cardiorespiratory measures between the two groups (supramaximal performance, SpO2, peak HR, and peak Ve).

Individual HR recovery rates (1HR) during 100% O2 or room air breathing were assessed by computer curve-fitting procedures employing a single-exponential decay equation, as previously described (23). Nonlinear techniques were used to calculate the parameters of the equation:

\[
HR(t) = Ae^{-kt} + y_0
\]

where HR(t) is the value of HR at time t (in seconds after cessation of exercise), A is a parameter, k is the rate constant, and y0 is the asymptotic baseline value. The time constant (1HR = 1/k) was used to quantify the recovery time and indicates the time necessary to achieve 63.2% of the difference between peak and baseline HR values. A two-exponential fit was also applied to the cardiac recovery data. However, no significant improvements in the goodness of fit were found between the monoexponential equation and the two-exponential equation using the F test (12).

In contrast to HR recovery, improved goodness of the fit was found for a two-order exponential decay equation when individual Ve recovery was assessed. Thus the exponential equation used was as follows:

\[
\dot{V}_e(t) = A_1e^{-kt_1} + A_2e^{-kt_2} + y_0
\]

where y0 is the asymptotic baseline value; A1 and A2 represent parameters; k1 and k2 represent fast and slow rate constants, respectively; and t1 and t2 represent time 1 and time 2 in seconds after cessation of exercise, respectively. As mentioned above, the time constants (1/t1 Ve = 1/k1 and 1/t2 Ve = 1/k2) were used to compare Ve recovery patterns in CF and control groups.

Two-way analysis of variance for repeated measures and the Newman-Keuls multiple-range test for multiple comparisons were employed for assessment of differences between CF
and control groups. Two-tailed paired t-tests were used to evaluate potential differences within the CF group or the control group (recovery in room air vs. 100% O2 and comparison of 2nd bout vs. 1st bout). In all tests, P < 0.05 was considered significant.

RESULTS

Seventeen CF patients and 17 normal subjects were studied. Most CF patients showed moderate obstructive disease by pulmonary function testing, and all had some degree of hyperinflation. Control subjects had normal pulmonary function tests. CF patients had significantly decreased weight and lean body mass. Resting SpO2 was slightly lower in CF patients. Clinical characteristics, pulmonary function values, and anthropometric data are shown in Table 1.

Maximal exercise. Duration of maximal exercise and peak workload was lower in the CF patients than in the control group (Table 2). At end of maximal exercise, VE, maximum HR, and VO2max were lower in CF patients. Anaerobic threshold occurred at a lower VO2 in CF patients than in normal subjects. No significant O2 desaturation occurred in CF patients or control subjects (Table 2).

Supramaximal exercise. Supramaximal performance was decreased in CF patients compared with control subjects. VE and HR were also decreased in CF patients at the end of supramaximal exercise compared with normal subjects (Table 3). No O2 desaturation was observed during supramaximal exercise in CF patients or control subjects.

Recovery after supramaximal exercise. An example of VE recovery during 100% O2 and room air breathing in one CF patient is shown in Fig. 1. For CF patients and control subjects, the sum of two exponentials provided a significantly better fit of the group mean VE recovery data than did a single exponential. For CF patients, F = 236 and P < 0.00001; for controls, F = 248 and P < 0.00001. In room air the fast (τ1VE) and the slow (τ2VE) time decay constants for ventilation were longer in CF patients than in control subjects (Fig. 2). However, although no changes in τ1VE or τ2VE occurred in control subjects when O2 was administered during recovery, significant reductions in τ1VE and τ2VE were observed in CF patients, indicating faster recovery with O2 (Fig. 2).

Table 1. Characteristics of study population

<table>
<thead>
<tr>
<th></th>
<th>CF</th>
<th>Control</th>
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<tbody>
<tr>
<td>Age, yr (%women)</td>
<td>25 ± 2(47)</td>
<td>25 ± 2(41)</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.63 ± 0.02</td>
<td>1.70 ± 0.02</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>55 ± 3‡</td>
<td>70 ± 3</td>
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<tr>
<td>VC, %predicted</td>
<td>83 ± 3‡</td>
<td>110 ± 4</td>
</tr>
<tr>
<td>FEV1, %predicted</td>
<td>62 ± 4‡</td>
<td>112 ± 4</td>
</tr>
<tr>
<td>FEV25–75, %</td>
<td>31 ± 6‡</td>
<td>93 ± 6</td>
</tr>
<tr>
<td>RV/TLC</td>
<td>0.44 ± 0.02‡</td>
<td>0.19 ± 0.01</td>
</tr>
<tr>
<td>Resting SpO2, %</td>
<td>97 ± 9*</td>
<td>99 ± 0</td>
</tr>
<tr>
<td>LBM, kg</td>
<td>44 ± 2*</td>
<td>53 ± 2</td>
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</table>

Values are means ± SE of 17 in cystic fibrosis (CF) patients and normal subjects. VC, vital capacity; FEV1, forced expiratory volume in 1 s; FEV25–75, forced expiratory flow from 25 to 75% of vital capacity; RV, residual volume; TLC, total lung capacity; SpO2, arterial O2 saturation; LBM, lean body mass. *P < 0.05; †P < 0.01; ‡P < 0.001.

Similarly, time decay constants for HR (τHR) also indicated faster recovery of HR in CF patients during O2 breathing, whereas this effect was absent in controls (Fig. 3). However, no correlations were found between pulmonary function (mean forced expiratory flow during the middle half of forced vital capacity and forced expired volume in 1 s) and time recovery constants τ1VE, τ2VE, and τHR in room air or O2.

No O2 desaturation was observed during recovery from supramaximal exercise in CF patients or control subjects.

Subsequent supramaximal performance. In CF patients, supramaximal performance decreased from the first bout to the second bout during recovery in room air, whereas it improved albeit slightly after recovery in O2 (Fig. 4, Table 3). In normal subjects no performance differences were found in the bout after recovery in O2 or room air (Fig. 4, Table 3; CF vs. control: P < 0.001, 2-way analysis of variance).

DISCUSSION

We have demonstrated that administration of supplemental O2 after supramaximal exercise accelerates recovery of HR and VE in CF patients. Also, subsequent supramaximal exercise performance improves in CF patients compared with control subjects when 100% supplemental O2 is administered between anaerobic exercise bouts. In control subjects, such beneficial effects of O2 were absent.

Aerobic and anaerobic exercise performances are decreased in CF (4, 6, 13, 15, 18). Major emphasis has been given to the improvement of aerobic exercise capacity in CF (19). Anaerobic exercise, rather than aerobic exercise, may be of more practical importance in CF, since routine daily activities often involve bouts of short anaerobic exercise. In addition, aerobic exercise training may not always improve baseline pulmonary function or increase weight gain in CF patients (27). Anaerobic exercise training such as weight lifting, however, significantly improves weight and muscle strength (24, 27). Similarly, interval training, which employs repeated bouts of anaerobic exercise, leads to improvement of aerobic exercise performance in ath-
letes and in patients with airway obstruction (25). Therefore, optimization of anaerobic training conditions in CF patients by accelerating cardiopulmonary recovery using supplemental O2 could ultimately lead to improved performances and exercise tolerance, as well as reduced discomfort during daily tasks.

The effects of administering supplemental O2 during aerobic exercise testing in CF patients has been studied, although the data available are not extensive. Previous work with CF patients from our laboratory indicated that supplemental O2 at an inspired O2 fraction of 0.3 during graded exercise increased VO2max and O2 pulse and reduced the severity of O2 desaturation (15). In a similar study, Nixon et al. (18) found that supplemental O2 administered during graded exercise minimized O2 desaturation in CF patients, who would otherwise normally desaturate during exercise. However, in contrast to the report of Marcus et al. (15), Nixon and co-workers found no improvements in peak work capacity or VO2 in CF. Nixon and co-workers did note that, for a given workload, CF patients given supplemental O2 during exercise performed at lower VE and HR during exercise than those breathing room air. Although impractical during daily activities, it appears that supplemental O2 during exercise in patients with obstructive lung disease may be beneficial in reducing HR and ventilatory outputs.

Despite widespread use of supplemental O2 by professional athletes during inactivity periods in a competition, data on the effects of supplemental O2 on the recovery from aerobic exercise and on subsequent performance are scarce. Robbins et al. (21) administered 100% O2 after bouts of submaximal and maximal aerobic exercise in healthy male athletes and found that breathing 100% O2 had no significant effect on the recovery kinetics of HR or VE. Furthermore, subsequent exercise performance was unaltered by O2 supplementation (21). The study by Robbins et al., however, involved healthy athletes. In well-trained individuals, supplemental O2 would not be expected to accelerate

**Table 3. Peak supramaximal exercise measurements and performance in CF patients and matched control subjects**

<table>
<thead>
<tr>
<th></th>
<th>AnP, kpm</th>
<th>VE, l/min</th>
<th>HR, beats/min</th>
<th>T, min</th>
<th>Workload, W</th>
<th>SpO2, %</th>
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<tbody>
<tr>
<td><strong>Before room air</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CF</td>
<td>1.240 ± 157*†</td>
<td>53 ± 4†</td>
<td>155 ± 2†</td>
<td>1.30 ± 0.12</td>
<td>147 ± 10†</td>
<td>95 ± 1†</td>
</tr>
<tr>
<td>Control</td>
<td>2.005 ± 204‡</td>
<td>77 ± 6†</td>
<td>163 ± 2†</td>
<td>1.27 ± 0.07</td>
<td>248 ± 18†</td>
<td>98 ± 0†</td>
</tr>
<tr>
<td><strong>After room air</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>CF</td>
<td>1.076 ± 110*†</td>
<td>56 ± 4†</td>
<td>161 ± 3</td>
<td>1.22 ± 0.12</td>
<td>147 ± 10†</td>
<td>95 ± 1†</td>
</tr>
<tr>
<td>Control</td>
<td>1.837 ± 183†</td>
<td>81 ± 7†</td>
<td>167 ± 4</td>
<td>1.15 ± 0.05</td>
<td>248 ± 18†</td>
<td>97 ± 0†</td>
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<tr>
<td><strong>Before O2</strong></td>
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</tr>
<tr>
<td>CF</td>
<td>1.111 ± 124†</td>
<td>53 ± 5†</td>
<td>151 ± 3</td>
<td>1.20 ± 0.12</td>
<td>147 ± 10†</td>
<td>96 ± 1</td>
</tr>
<tr>
<td>Control</td>
<td>1.961 ± 1.96†</td>
<td>83 ± 7†</td>
<td>159 ± 3</td>
<td>1.25 ± 0.05</td>
<td>248 ± 18†</td>
<td>97 ± 0</td>
</tr>
<tr>
<td><strong>After O2</strong></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>CF</td>
<td>1.174 ± 129†</td>
<td>55 ± 4†</td>
<td>159 ± 3†</td>
<td>1.27 ± 0.12</td>
<td>147 ± 10†</td>
<td>97 ± 1*</td>
</tr>
<tr>
<td>Control</td>
<td>1.815 ± 199‡</td>
<td>87 ± 8†</td>
<td>170 ± 2†</td>
<td>1.22 ± 0.07</td>
<td>248 ± 18†</td>
<td>99 ± 0*</td>
</tr>
</tbody>
</table>

Values are means ± SE. AnP, supramaximal performance. T, duration of supramaximal bout. *P < 0.01, 1st bout vs. 2nd bout in CF. †P < 0.01, CF vs. control. ‡P < 0.001, 1st bout vs. 2nd bout in control. Two-way analysis of variance for repeated measures revealed that O2 modified 1st bout vs. 2nd bout AnP in CF but not in controls (P < 0.001).

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![Fig. 1. Minute ventilation (VE) at 15-s intervals during recovery in room air (■) and 100% O2 (○) in a cystic fibrosis patient. Lines correspond to 2-exponential decay equations calculated for each recovery. Fast time constants (t1VE) were 47.8 and 35.1 s in room air and O2, respectively; slow time constants (t2VE) were 178.3 and 158.7 s in room air and O2, respectively.](image)

![Fig. 2. Mean t1VE and t2VE in cystic fibrosis (CF) patients and matched controls (CON) after supramaximal exercise, as derived from individual 2-exponential curve-fitting equations. Values are means ± SE. *Room air vs. 100% O2 in CF for t1VE, P < 0.01. #Room air (RA) vs. 100% O2 in CF for t2VE, P < 0.03.](image)
recovery, since exercise is not primarily limited by pulmonary mechanics.

Assessment of the kinetics of recovery from a short bout of exercise indicates that time constants depend on the intensity of the exercise being performed (1). Indeed, Armon et al. (1) found significant increases of $\tau_{VE}$ with increasing work intensity in healthy adults and also reported that when the workload corresponded to 125% of the anaerobic threshold, a two-exponential rather than a single-exponential equation provided a better fit for recovery. Our findings in this study indeed concur with such data (1). Similar increases in $\tau_{HR}$ with workload were also found (2). Thus precaution to match individual exercise workloads as a constant percentage of the maximal load achieved during the graded exercise test appears essential whenever comparisons of recovery kinetics are contemplated across experimental groups.

In this study we addressed the effects of supplemental O$_2$ on recovery from anaerobic exercise and subsequent anaerobic performance. As in the study by Robbins et al. (21), no significant improvements in HR or ventilatory kinetics were found in the control group. In contrast, CF patients in our study achieved faster recovery of HR and VE when O$_2$ was administered, and they were able to enhance their subsequent performance. Although hyperoxic inhibition of peripheral chemoreceptor tone could be advanced as one possible explanation for accelerated decreases in cardioventilatory outputs, this explanation appears unlikely, since it would be expected to occur in CF patients and control subjects. However, accelerated recovery rates were present in CF patients only. Another alternative explanation for the faster HR recovery could involve improvements in cardiac output associated with O$_2$ breathing in CF patients. This possibility is unlikely, since there was no evidence of cardiac limitation during exercise and $\tau_{HR}$ values in room air were similar in CF patients and controls. O$_2$-induced alterations in regional capillary bed resistance such that blood flow would be preferentially directed to exercised muscles could lead to faster lactate removal and pH normalization, which in turn could reduce muscle afferent nerve input and sympathetic recruitment.

In individuals with CLD and congestive heart failure (CHF), altered skeletal muscle metabolism has been demonstrated using $^{31}$P nuclear magnetic resonance spectroscopy (22, 28). Studies have shown reduced phosphocreatine-to-inorganic phosphate ratios in exercising muscle of individuals with CLD and CHF, which may reflect impaired oxidative metabolism (28). Arterial O$_2$ output (cardiac output multiplied by arterial O$_2$ content) has also been found to be reduced in CHF and CLD (28). It has been suggested that the reduced muscle metabolism in these subjects may be secondary to a number of factors, including reduced blood flow, impaired O$_2$ delivery to muscle, and reduced mitochondrial oxidative capacity (28). In healthy individuals, energy for anaerobic exercise can come from aerobic as well as anaerobic sources (16). The energy needed for anaerobic exercise in individuals with chronic respiratory impairments may come from earlier activation of anaerobic glycolysis and increased proportion of energy from anaerobic glycolysis compared with normal individuals (11). Therefore, in individuals with CLD, a larger proportion of the overall energy produced originates from anaerobic glycolysis, such that increased O$_2$ debt will be incurred during short, exhaustive bouts of exercise. Supplemental O$_2$ could accelerate recovery by helping overcome some of these deficits. For example, an immediate consequence of O$_2$ supplementation would be a significant increase in arterial O$_2$ content if our CF patients had demonstrated significant O$_2$ desaturations during supramaximal exercise, in contrast to control subjects, in whom the increase in arterial O$_2$ content would have been of minor proportions. However, major decreases in O$_2$ saturation did not occur in our CF patients, suggesting that mechanisms other than limitations in blood O$_2$ content could be operative in this context. Higher arterial PO$_2$, during the recovery period could accelerate the rate of muscle energy stores repletion, which might be compromised in CF because of intrinsic mechanisms inherent to CF or, more likely, as a result of severe deconditioning in these patients. Although our study design cannot provide definitive answers regarding the mechanisms underlying improved recovery from supramaximal exercise in CF, several mechanisms are postulated. Among these, improved muscle microcirculatory flows due to shifts in

![Fig. 3. Mean heart rate recovery time constants ($\tau_{HR}$) in CF patients and matched controls after supramaximal exercise, as calculated from single-exponential equation curve fitting. Values are means ± SE. *Room air vs. 100% O$_2$ in CF for $\tau_{HR}$, P < 0.01.](image1)

![Fig. 4. Subsequent supramaximal performance (% AnP) after recovery in room air or 100% O$_2$ in CF patients and matched controls. Performance is expressed as percentage of 1st supramaximal bout. Values are means ± SE. *Room air vs. 100% O$_2$ in CF, P < 0.01.](image2)
vascular bed resistances with preferential flow delivery to exercised muscles, improved lactate removal kinetics from exercised muscles, thereby reducing metabolotropic type III/IV fiber recruitment more rapidly and thus diminishing ventilatory stimulation by such fibers, improved intracellular muscle energy recovery kinetics, and/or differences in central drive changes due to O₂ breathing could be involved in O₂-associated faster recovery kinetics in CF. Delineation of which of these potential mechanisms underlies the improvement in cardiorespiratory recovery in CF patients with supplemental O₂ during the recovery period awaits further study.

Several methodological issues deserve comment. Improvements in lung function in our CF patients during the period between the two supramaximal exercise series could exert profound effects on overall performance. However, our CF patients were clinically stable and were randomly assigned to receive O₂ or room air during the recovery period. Indeed, 9 of the 17 CF patients received O₂ in their first series. Similarly, a training effect could have induced changes in performance over time. Such an effect is unlikely, since the only improvements in performance were those associated with O₂ supplementation. Finally, a placebo effect could modify the motivation during the exercise bouts. However, CF patients and control subjects were unaware of the gas mixture being administered, and there were absolutely no visual cues in the laboratory setting that may have been conducive to an assumption in either direction.

In summary, supplemental O₂ does not modify the recovery from supramaximal exercise and will not modify subsequent supramaximal performance in healthy individuals. However, in CF patients, accelerated recovery and improved supramaximal performance ensue. We speculate that optimization of anaerobic exercise training by the administration of supplemental O₂ during recovery may not only enhance compliance with training schedules but also lead to increased anaerobic and aerobic exercise tolerance, resulting in overall improvements in the quality of life.

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