Maximal Strength Training of the Legs in COPD: A Therapy for Mechanical Inefficiency

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ABSTRACT

HOFF, J., A. E. TJØNNA, S. STEINSHAMN, M. HØYDAL, R. S. RICHARDSON, and J. HELGERUD. Maximal Strength Training of the Legs in COPD: A Therapy for Mechanical Inefficiency. *Med. Sci. Sports Exerc.*, Vol. 39, No. 2, pp. 220–226, 2007. **Purpose:** A diminished mechanical efficiency (work/O_2 consumed) accompanies chronic obstructive pulmonary disease (COPD), and increased mechanical efficiency has been attained by maximal strength training (MST) with an emphasis on the maximal rate of force mobilization in the concentric phase in healthy subjects. This study combined these observations and evaluated the impact of short-term MST on patients with COPD. **Methods:** Twelve patients with COPD ($FEV_1 = 1.1 \pm 0.1$) were pretested and then randomly assigned to either an MST group ($N = 6$) or a normal activity control group ($N = 6$). Within each MST training session (three times per week for 8 wk), patients performed four sets of seated leg presses with a focus on the rate of force development at an intensity that only allowed the performance of five repetitions. **Results:** Patients who performed MST significantly improved their rate of force development ($105 \pm 22.8\%$), mechanical efficiency ($32 \pm 7\%$), and FEV_1 ($21.5 \pm 6.8\%$), whereas these variables were unchanged in the controls. Neither group changed either peak oxygen consumption ($\dot{V}O_{2peak}$) or body mass. **Conclusion:** In combination with the observed improvement in FEV_1, these data certainly support the therapeutic role for MST in the treatment of COPD. **Key Words:** CHRONIC OBSTRUCTIVE PULMONARY DISEASE, SKELETAL MUSCLE, RATE OF FORCE DEVELOPMENT, EXERCISE, EFFICIENCY

In a disease that impacts O_2 transport, such as chronic obstructive pulmonary disease (COPD), a diminished mechanical efficiency (work/O_2 consumed) would be expected to exacerbate the impact of this illness. Indeed, although it is not always clearly identified in all subjects in all studies (1), there is growing evidence that a reduced mechanical efficiency may often accompany COPD (2,3). Initial evidence, collected primarily by assessing O_2 use across the whole body, suggested that the increased cost of breathing associated with COPD (17,26) might account for this mechanical inefficiency (2,3). However, in the wake of the recent interest in skeletal muscle changes associated with COPD, several studies have used a catheter-based approach to directly study isolated locomotor muscle and have revealed significantly reduced mechanical efficiency in these patients, which clearly is not a consequence of an increased cost of breathing (24,25).

The use of strength training with an emphasis on the maximal rate of force mobilization in the concentric phase (MST), consisting of high loads and few repetitions, has been reported to improve work economy by approximately 5–20%, even in subjects who were already exercise trained (10,11,20). Although the mechanisms behind the effect of MST on mechanical efficiency have not been completely elucidated, there is evidence that changes in the power–load and velocity–load relationships lead to a longer relaxation period within the duty cycle, enhancing recovery between contractions (19). Additionally, this may reduce the duration of vascular compression during the contraction phase of muscular work, improving muscle perfusion across the duty cycle and, thus, facilitating the ever important transport of O_2 at the muscular level (23). Such potential improvements are certainly germane to patients with COPD, who may experience both attenuated O_2 transport and reduced mechanical efficiency during exercise (24,25). It is not known whether MST significantly enhances mechanical efficiency in patients with COPD. In addition, there are several indications in the literature supporting a relationship between peripheral strength-training responses and pulmonary function in COPD (14,27,30). If these findings are in any way linked to the breathing demands of strength training, the forced and exaggerated breathing resulting from MST may especially challenge the respiratory muscles as well as the main locomotor muscle target.

Consequently, this study was designed to investigate whether MST can enhance mechanical efficiency during exercise in patients with COPD while also improving lung function via improvements in respiratory muscle function.

Specifically, we tested the following hypotheses: 1) Patients with COPD will increase maximal strength and improve their maximal rate of force development in response to 8 wk of MST; 2) improvements in strength and rates of force development will directly translate into increased mechanical efficiency in patients with COPD, as measured during submaximal cycle exercise; and 3) the parallel training effect on the respiratory muscles during MST of the locomotor muscles will improve pulmonary function in patients with COPD, as measured by FEV_1.

METHODS

Subjects

Twelve patients with COPD (eight males and four females) were studied; six patients were assigned to the MST group and six were assigned to the control group. Randomization within genders facilitated groups that were balanced for gender (two females per group). Inclusion criteria were a clinical definition of COPD according to GOLD guidelines (1), patients aged between 40 and 70 yr, $FEV_1/FVC < 70\%$, and $FEV_1 < 60\%$ of predicted. Exclusion criteria were a history of cardiovascular disease, lung disease other than COPD, diabetes mellitus or other metabolic diseases, malign disease, pregnancy, corticosteroid use in the last 6 months, and a respiratory tract infection within the last 4 wk. Medication was monitored and did not change during the experimental period. Written informed consent was obtained from all subjects, and the regional medical ethics committee approved the study. Subject characteristics are presented in Table 1.

Testing Procedures

Pre- and posttests were performed within 3 d of the training intervention. The order of testing and protocols for the pre- and posttests were identical. The pulmonary function tests (Master Screen Pneumo; Jaeger GmbH and Co, Germany), blood samples for [hemoglobin] and hematocrit (Coulter STKS, Coulter Electronics Ltd, England), and resting EKG were performed approximately 1 h before the exercise tests. Immediately before the exercise tests, resting heart rate (HR) (Polar Sport Tester, Polar Electro, Finland), arterial oxygen saturation by oximetry (SaO_2) (507E scholar II, Critcare Systems Inc.), and finger-stick lactate concentration [La] (YSI 1500 Sport Lactate Analyser) were measured. One-repetition maximum (1RM) was measured on a horizontal leg press (Super Gym, Taiwan) at a knee angle of 90°, with force development and peak force assessed via the force platform (9286AA, Kistler, Switzerland) installed on the leg press device with data collected at 2000 Hz (Bio-ware v3.06b, Kistler, Switzerland). Each patient performed two dynamic rate-of-force-development efforts using a load corresponding to 70% of 1RM, and the best attempt was recorded. All rate-of-force-development measurements were made with a knee angle of 90° and were measured from 10 to 90% of the peak force in the concentric phase of the leg press. The patients rested for 2 min between each rate-of-force-development and peak force measurement (Fig. 1).

After the completion of the strength testing, patients commenced the assessment of mechanical efficiency ([VO_2 − resting VO_2]/W). Both VO_2 (MetaMax II Cortex, Leipzig, Germany) and watts were converted to kilojoules to allow the calculation of percent mechanical efficiency (22), which was measured during the last 3 min of a 5-min standardized workload (40 W, 60 rpm) on a cycle ergometer (Ergomedic 839, Monark Exercises, Sweden). The cycle exercise paradigm was selected for its accepted and reliable work/oxygen consumption relationship, uncomplicated by body weight. Thereafter, the workload was increased by 10 W every minute until $\dot{V}O_{2peak}$ was reached. These increases in work rate were achieved by increasing resistance at a constant cadence of 60 rpm. After every work period, a blood sample was drawn from the finger, and unhemolyzed [La] was analyzed immediately. Heart rate and SaO_2 were assessed continuously during the cycle test. The Borg perceived exertion scale (0–20) was used to document perceived exertion during cycle exercise.

Training Intervention

The MST group performed an 8-wk training regime (24 training sessions) that was individually monitored in the laboratory. The strength-training sessions consisted of four sets of five repetitions with a focus on the rate of force development during the concentric contraction of the quadriceps from a 90° bend at the knees to straight legs. Instructional emphasis was placed on stopping the eccentric phase with a 90° bend in the knees, a pause, and then maximal mobilization of force in the concentric movement. The load corresponded to 85–90% of 1RM. Between repetitions, rest was determined by the patients, but it was limited to a second or two to regain composure. After each set, the subject rested for 2 min. When a patient was able to perform more than five repetitions in a set, the load was increased by 2.5-kg increments until only five repetitions could again be achieved. All strength training was performed on the same seated horizontal leg press apparatus used during the strength testing. Both the MST

TABLE 1. Characteristics of the patients with COPD.

	Maximal Strength-Training Group	Control Group
Age (yr)	62.8 ± 1.4	60.6 ± 3.0
Height (cm)	167.5 ± 3.4	172.4 ± 4.2
Body mass (kg)	73.7 ± 7.6	79.2 ± 5.7
[Hb] (g·dL^{-1})	15.3 ± 0.5	14.3 ± 0.4
Hct (%)	44.8 ± 1.3	42.4 ± 0.9
FEV_1 (L) (% predicted)	0.92 ± 0.13 (32.9 ± 3.3)	1.24 ± 0.17 (39.5 ± 6.4)
FVC (L) (% predicted)	1.99 ± 0.41 (53.4 ± 6.8)	2.80 ± 0.33 (69.1 ± 3.8)
FEV_1/FVC (%)	49.9 ± 4.6	45.2 ± 6.0

Data are presented as means ± SE. Hb, hemoglobin; Hct, hematocrit; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity.
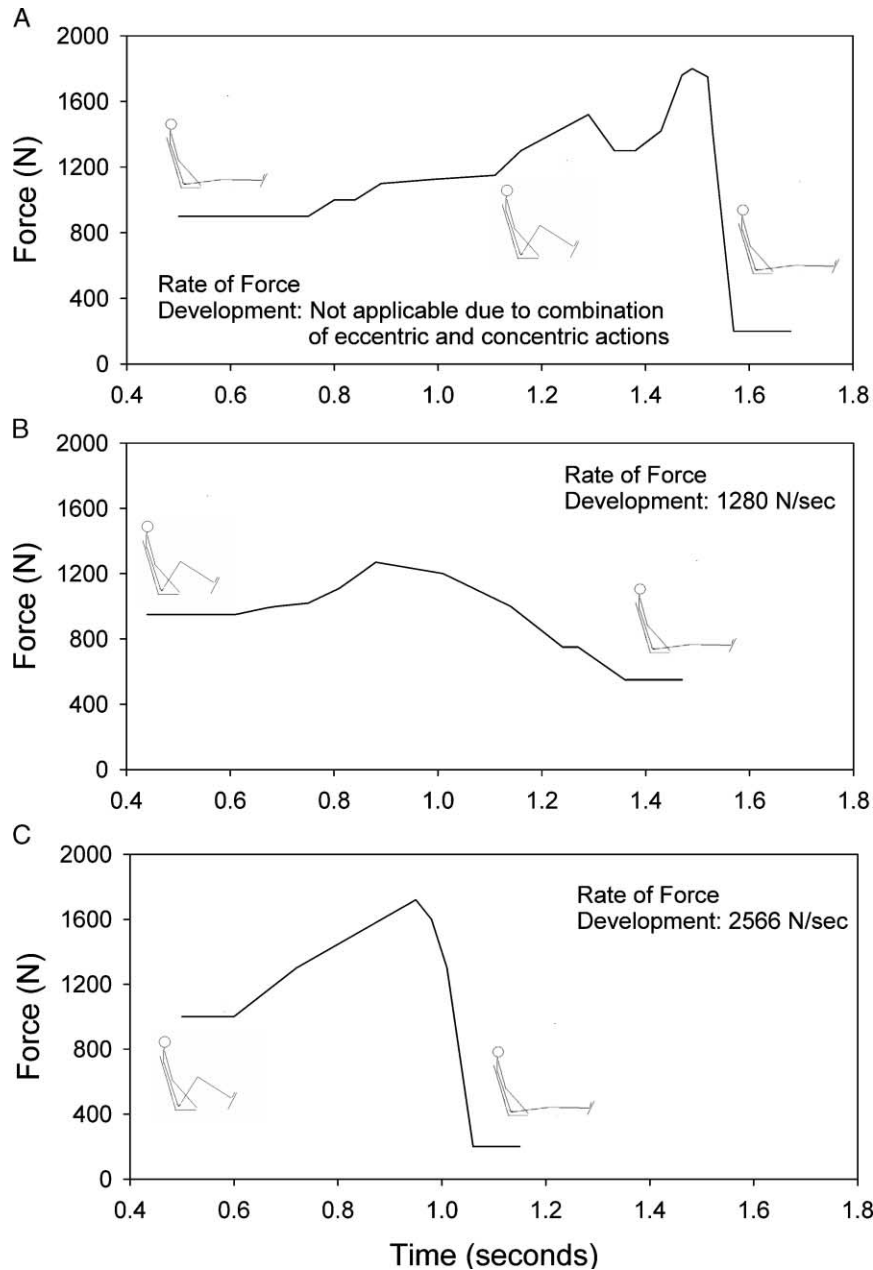
FIGURE 1—The force/time profiles generated by three varying approaches to strength training on a horizontal leg press device by a patient with COPD: A) eccentric movement before concentric movement; B) concentric start point, but with no directive regarding maximally mobilizing force; and C) the approach employed in the current study: with a concentric start point, the patient was instructed to create maximal force mobilization.

and control group continued their normal daily living with modest regular activity, as recommended by their pulmonary physician.

Statistical Analysis

Pre- and posttest differences within each group were analyzed using the Wilcoxon signed rank test for paired groups (a Wilcoxon test with two related samples). Differences between groups in pre- and posttests were analyzed using a Mann–Whitney U test. Relationships between variables were assessed with linear regression analyses. Despite the small sample size, the significant effect size meant that these analyses were adequately powered ($\beta >$

0.8). The alpha level was set at $P < 0.05$. Data are presented as means ± SE.

RESULTS

Baseline Testing and Exercise Training Adherence

There were no significant differences in age, physical characteristics, blood O_2-carrying capacity, and baseline pulmonary function between those patients with COPD who performed MST and the normally active control group (Table 1). All subjects completed the study protocol without any adverse effects, and the MST group completed 100% of the planned training.

TABLE 2. Alterations in strength parameters in patients with COPD before and after 8 wk of maximal strength training and normal activity in the control group.

	Maximal Strength-Training Group		Control Group		
	Pre	Post	Pre	Post	Overall Change in Means (95% CI)
RFD dynamic 90° (N·s^{-1})	2262 ± 381	4146 ± 528*,#	3389 ± 595	3317 ± 507	1957 (1276–2637)
1RM dynamic 90° (kg)	118 ± 17	150 ± 21*,#	143 ± 24	139 ± 23	36 (21–50)
Peak force, static (N)	1179 ± 208	1509 ± 176*,#	1329 ± 196	1328 ± 172	332 (114–549)
Peak force, dynamic (N)	1022 ± 189	1159 ± 146	1336 ± 227	1382 ± 193	124 (42–205)
Body mass (kg)	73.7 ± 7.6	75.6 ± 7.2	79.0 ± 5.8	79.0 ± 5.6	2 (1–3)

Data are presented as means ± SE. Overall change in means is the change (post–pre means) for the training group minus the change (post–pre means for the control group). CI, confidence interval; RFD dynamic, dynamic rate of force development; 1RM dynamic, dynamic one-repetition maximum. * Significant difference from pre- to posttest ($P < 0.05$); # significant difference between groups ($P < 0.05$).

Strength Parameters

The control group revealed no significant change in strength-related variables, whereas the MST group significantly increased in every measured variable except for peak force measured dynamically. Neither group experienced a change in body mass during the 8-wk period (Fig. 2, Table 2).

Submaximal Cycle Exercise

The MST group improved their mechanical efficiency of cycling at 40 W by 31.3 ± 6.8%, with no other significant change in the measured variables. Perceived exertion in these patients while cycling at 40 W was also significantly reduced after MST, whereas this and all other physiological responses were unchanged by the 8-wk period in the controls (Fig. 2, Table 3). The O_2 or CO_2 ventilatory equivalents were not altered by the MST.

Pulmonary Function

There were no significant changes in the control group. The MST group experienced a significant improvement in FEV_1 from 0.92 ± 0.13 to 1.09 ± 0.13 L (~21.5%) (overall change in means = 0.23, CI = 0.1 to 0.4 L), which, in turn, improved FEV_1 percentage predicted from 33 ± 3 to 40 ± 3% predicted (~21.2%). FVC also significantly increased from 1.99 ± 0.4 to 2.19 ± 0.3 L (~10%) (overall change in means = 0.44, CI = 0.12 to 0.76 L). Because both FEV_1 and FVC increased after MST, the ratio of FEV_1/FVC increased insignificantly from 49.9 ± to 5 to 52 ± 4% (overall change in means = 0.02, CI = −0.04 to 0.07%).

Maximal Exercise Cycle Tests

In agreement with the finding of improved mechanical efficiency in the MST group while cycling at 40 W, this group achieved a significantly greater maximal cycle work rate (~10%) with an unchanged $\dot{V}O_{2peak}$. This was not the case for the control group, whose only change between pre- and posttesting was an unexpected reduction in ventilation at WR_{max} (Fig. 2, Table 4). The O_2 or CO_2 ventilatory equivalents were not altered by the MST.

Correlational Analyses

The improvement in 1RM correlated positively with the improvement in the rate of force development ($r = 0.79$, CI −0.06 to 0.98), mechanical efficiency ($r = 0.65$, CI −0.34 to 0.96), and FEV_1 ($r^2 = 0.67$, CI −0.31 to 0.96). The improvement in the rate of force development correlated positively with the improvement in mechanical efficiency ($r = 0.69$, CI −0.28 to 0.96) and FEV_1 ($r = 0.76$, CI −0.13 to 0.97). All were statistically significant correlations.

DISCUSSION

The major novel finding of this study is that MST significantly improved strength and rate of force development in patients with COPD, resulted in an approximately 32% increase in mechanical efficiency and a fall in perceived exertion during submaximal work. This improvement returned these patients to within the accepted normal range for cycling mechanical efficiency and is likely to translate into improved performance of daily activities and improved quality of life. In addition, MST significantly improved pulmonary function, as indicated by

TABLE 3. Physiological responses to a 40-W work rate in patients with COPD before and after 8 wk of maximal strength training and normal activity in the control group.

	Maximal Strength-Training Group		Control Group		
	Pre	Post	Pre	Post	Overall Change in Means (95% CI)
VO$_2$ (mL·kg^{-1}·min^{-1})	14.0 ± 1.7	11.0 ± 0.9*	11.8 ± 1.43	11.3 ± 1.1	−3 (−4 to −1)
VO$_2$ (L·min^{-1})	0.98 ± 0.06	0.81 ± 0.05*,#	0.91 ± 0.02	0.88 ± 0.06	−0.14 (−0.25 to −0.03)
Mechanical efficiency (%)	16 ± 2	21 ± 2*,#	17 ± 2	18 ± 2	3.4 (−0.01 to 6.8)
HR (bpm)	125 ± 4.5	115 ± 7.5	117 ± 10	110 ± 8.5	−2.4 (−14.4 to 9.6)
V̇E (L·min^{-1})	30.9 ± 2.2	28.0 ± 1.8	27.8 ± 2.0	26.0 ± 1.9	−1.2 (−3.5 to 1.2)
[La$^-$] (mmol·L^{-1})	2.35 ± 0.34	2.42 ± 0.32	2.61 ± 0.38	1.98 ± 0.28	0.7 (0.2 to 1.2)
RER	0.89 ± 0.01	0.90 ± 0.01	0.89 ± 0.02	0.88 ± 0.02	0.03 (−0.02 to 0.07)
SaO$_2$ (%)	93 ± 1.2	92 ± 0.4	93 ± 1.3	92 ± 0.4	0.23 (−2.95 to 3.42)
Perceived exertion (0–20)	13.7 ± 1.1	11.8 ± 0.53*	12.4 ± 1.3	12.0 ± 1.1	−1.43 (−3.13 to 0.27)

Data are presented as means ± SE. Overall change in means is the change (post–pre means) for the training group minus the change (post–pre means for the control group). CI, confidence interval; VO$_2$, oxygen uptake; HR, heart rate; V̇E, minute ventilation; La$^-$, lactate; RER, respiratory exchange ratio; SaO$_2$, arterial oxygen saturation. * Significant difference from pre- to posttest ($P < 0.05$); # significant difference between groups ($P < 0.05$).
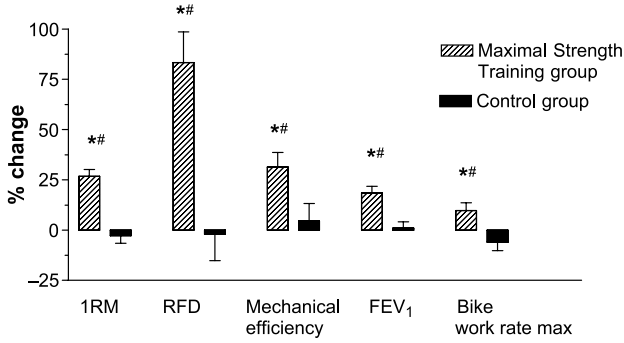
FIGURE 2—The percent change in maximal strength (1RM), rate of force development, mechanical efficiency during cycling and cycling work rate maximum (WR_{max}), forced expiratory volume in 1 s (FEV_1), after 8 wk of maximal mobilization of force in the concentric-phase strength training (MST) and the control group. * Significant difference between pre- and posttest ($P < 0.05$); # significant difference between groups ($P < 0.05$).

an approximately 22% increase in FEV_1. On the basis of the clinical definition of severe COPD (i.e., $FEV_1 < 1.0$ L), the improvement in FEV_1 attained by these patients (0.92 ± 0.32 to 1.09 ± 0.34 L) returned them from a severe to a moderate diagnosis. This positive effect on pulmonary function, coupled with the increase in mechanical efficiency, supports a therapeutic role for MST as a useful treatment for patients with COPD.

MST and Mechanical Efficiency

There is now considerable evidence that COPD may be associated with attenuated mechanical efficiency (2,3,21,24,25). Most recently, the direct assessment of an isolated skeletal muscle model in patients with COPD revealed a concomitant reduction in the mechanical efficiency of and the number of type I fibers within this muscle group (24). Indeed, in contrast to healthy aging (16), older patients with moderate to severe COPD consistently demonstrate an increase in the proportion of type II fibers, assessed either histochemically (13) or by the expression of myosin heavy chain isoforms (18). As exercise intensity and or rate of force development increases, there is a growing reliance on type II muscle fibers that has been proposed to lead to less efficient muscular work (5). There are convincing data, both from in

vitro (6) and in vivo (5,12) studies, that the energetic cost of force production is fiber-type specific. Although the current study did not directly assess the muscle structure of these patients with COPD, fiber-type differences may explain the documented attenuation in mechanical efficiency during cycling in this study (~15% vs normal (~23%)) (24,25).

The MST performed three times a week for 8 wk resulted in a $31.3 \pm 6.8\%$ ($\pm$ SE) improvement in mechanical efficiency during submaximal cycle exercise, with no such changes apparent during the same time frame in the control group. The improvement in mechanical efficiency induced by MST correlated significantly with the increased rate of force development ($r = 0.69$) and peak force ($r = 0.65$). An increased rate of force development leads to longer atonic periods between contractions and enhanced muscle perfusion, whereas an increased peak force results in a reduction in the relative load placed on the muscle during submaximal efforts (19). Additionally, it was previously documented in humans that within 2–4 wk of an 8-wk strength-training program, there was a significant reduction in the percentage of type IIx fibers, with a concomitant trend toward an increase in the more fatigue-resistant type IIa fibers (28). Therefore, the consequences of MST, coupled with an increased reliance on type II fibers in patients with COPD, may explain some of the improvements observed in mechanical efficiency in these patients.

In the current study, a possible limitation in terms of cycling efficiency was the regular exposure to cycling as a warm-up in the strength-training group, but not in the controls. However, the minimal load and duration of this cycling exposure, combined with our inability to improve mechanical efficiency with cycling-specific training in other COPD patients (unpublished observations), refutes this as a major limitation of the present study.

MST and Pulmonary Function

The MST group revealed a clear improvement in both FEV_1 and FVC. Although MST did not involve any specific expiratory muscle training, it is well accepted that the biomechanics of leg press exercise demands an integral involvement and, therefore, training adaptation of the abdominal muscles (15,29). Acknowledging the negative

TABLE 4. Physiological responses at maximal cycle exercise in patients with COPD before and after 8 wk of maximal strength training and normal activity in the control group.

	Maximal Strength-Training Group		Control Group		
	Pre	Post	Pre	Post	Overall Change in Means (95% CI)
$\dot{V}O_{2peak\ I}$ (mL·kg^{-1}·min^{-1})	20.6 ± 2.3	19.3 ± 2.1	20.9 ± 2.4	19.9 ± 2.2	-0.23 (-1.81 to 1.34)
$\dot{V}O_{2peak}$ (L·min^{-1})	1.45 ± 0.11	1.41 ± 0.13	1.66 ± 0.24	1.57 ± 0.19	0.05 (-0.06 to 0.16)
Maximal workload (W)	72 ± 9.8	79 ± 8.6*	100 ± 16	94 ± 14	11 (-0.72 to 22.72)
HR (bpm)	139 ± 3	137 ± 5	148 ± 15	141 ± 13	5 (-2 to 12)
$\dot{V}E$ (L·min^{-1})	46 ± 4	45 ± 5	52 ± 10	45 ± 7*	5 (-0.02 to 10)
[La$^-$] (mmol·L^{-1})	3.4 ± 0.4	3.5 ± 0.4	3.5 ± 0.7	2.85 ± 0.42	0.7 (-0.3 to 1.43)
RER	0.94 ± 0.02	0.94 ± 0.03	0.95 ± 0.03	0.92 ± 0.02	0.3 (0 to 0.06)
SaO$_2$ (%)	90 ± 1	89 ± 1	92 ± 1	90 ± 1	0.23 (-2.04 to 2.51)
Perceived exertion (0–20)	17 ± 1	18 ± 1	18 ± 1	17 ± 1	1.83 (0.53 to 3.14)

Data are presented as means $\pm$ SE. Overall change in means is the change (post–pre means) for the training group minus the change (post–pre means for the control group; CI, confidence interval; $\dot{V}O_{2peak}$, maximal oxygen uptake; HR, cardiac frequency, $\dot{V}E$, minute ventilation, La$^-$, lactate; RER, respiratory exchange ratio; SaO$_2$, arterial oxygen saturation.
* Significant difference from pre- to posttest ($P < 0.05$).

impact of COPD on lung elastic recoil and airway resistance, but also recognizing the concomitant weakening of the respiratory muscles, it is possible that functional improvements of the expiratory muscles (abdominal wall, internal intercostals) may have led to improved pulmonary function, as measured by forced expiration. Indeed, several indications in the literature suggest that a relationship between peripheral strength-training responses and pulmonary function in COPD may exist (27,30). Most recently, Kongsgaard et al. (14) added to this trend by recognizing that their control group of patients with COPD who only performed breathing exercises had a fall in FEV_1 during a 12-wk period, whereas the strength-trained patients maintained their prestudy levels. In the current study, with only an 8-wk duration, the FEV_1 of the control subjects remained constant, but the MST group revealed an approximately 20% increase in FEV_1, which correlated well with their improvements in the rate of force development ($r^2 = 0.58$) and 1RM ($r^2 = 0.45$). Although the improvements in FEV_1 and FVC are in a positive direction, and FEV_1 correlates well with many indices of COPD disease severity, the clinical impact of these improvements in pulmonary function on patient health still need to be determined.

MST, Strength, and Rate of Force Development

There is clear evidence that COPD is associated with attenuated muscle strength (9). In fact, up to 70% of patients demonstrate lower quadriceps-muscle strength than their healthy age-matched counterparts, and disease severity, as assessed by symptom intensity and pulmonary function, is well correlated with this loss of strength (4). The muscle-mass wasting that accompanies COPD, perhaps as a simple consequence of inactivity, seems to explain these findings in that the ratio of quadriceps strength/muscle CSA is preserved (4,7). Although not the major goal of this study, the improvements in muscle strength attained by the current patients with COPD (Fig. 2, Table 4) are in agreement with the findings of Simpson et al. (27), who revealed that more traditional strength training could improve muscle strength in such patients and lead to higher exercise capacities in terms of time to exhaustion at submaximal workload. It should be

noted that in these studies and in the current research, there are always considerable, unavoidable learning effects that are probably as important, in terms of the strength changes, as the physiological adaptations in the muscle itself. Additionally, test–retest data were not collected in the present study, which may also limit the interpretation of the recorded strength changes. However, in our hands, these tests have revealed excellent test–retest data.

Clinical Implications

COPD is the most common chronic pulmonary disease, with more than 14 million North Americans living with this diagnosis (8). It is universally accepted that a substantially reduced exercise capacity accompanies COPD, with a subsequent attenuation in maximal work rate. However, it is also becoming increasingly apparent that, in addition to the degradation of pulmonary function, a reduction in skeletal muscle mechanical efficiency may also contribute to the challenge of physical work performed by these patients (2,3,24,25). These multiple factors combine, leading to inactivity, muscle disuse, and a downward spiral toward even more exaggerated ill health.

The current findings reveal that because MST is minimally taxing to the ventilatory and cardiac systems, it is well suited to patients with COPD, not resulting in the normal dyspnea-associated discomfort experienced by this population during conventional exercise such as walking. This is highlighted in the current patients by the 100% compliance and completion of training in the MST group. The inclusion of MST in a cardiopulmonary rehabilitation program could, according to the current data, result in an approximately 32% increase in mechanical efficiency. In the real world, this translates to either having the potential to perform significantly more work or to perform the same work with a reduced effort, which is likely an important result with practical implications for patients with COPD.

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