

The Relationship Between Maximal Expiratory Flow and Increases of Maximal Exercise Capacity with Exercise Training

TONY G. BABB, KELLY A. LONG, and JOSEPH R. RODARTE

Institute for Exercise and Environmental Medicine, Presbyterian Hospital of Dallas; The University of Texas Southwestern Medical Center, Dallas; Department of Human Performance, Rice University, Houston; and Department of Medicine, Baylor College of Medicine and The Methodist Hospital, Houston, Texas

We previously reported that patients with mild to moderate airflow limitation have a lower exercise capacity than age-matched controls with normal lung function, but the mechanism of this reduction remains unclear (1). Although the reduced exercise capacity appeared consistent with deconditioning, the patients had altered breathing mechanics during exercise, which raised the possibility that the reduced exercise capacity and the altered breathing mechanics may have been causally related. Reversal of reduced exercise capacity by an adequate exercise training program is generally accepted as evidence of deconditioning as the cause of the reduced exercise capacity. We studied 11 asymptomatic volunteer subjects (58 ± 8 yr of age [mean \pm SD]) selected to have a range of lung function (FEV_1 from 61 to 114% predicted, with a mean of $90 \pm 18\%$ predicted). Only one subject had an FEV_1 of less than 70% predicted. Gas exchange and lung mechanics were measured during both steady-state and maximal exercise before and after training for 30 min/d on 3 d/wk for 10 wk, beginning at the steady-state workload previously determined to be the maximum steady-state exercise level that subjects could sustain for 30 min without exceeding 90% of their observed maximal heart rate (HR). The training workload was increased if the subject's HR decreased during the training period. After 10 wk, subjects performed another steady-state exercise test at the initial pretraining level, and another maximal exercise test. HR decreased significantly between the first and second steady-state exercise tests ($p < 0.05$), and maximal oxygen uptake ($\dot{V}O_{2max}$) and ventilation increased significantly ($p < 0.05$) during the incremental test, indicating a training effect. However, the training effect did not occur in all subjects. Relationships between exercise parameters and lung function were examined by regression against FEV_1 expressed as percent predicted. There was a significant positive correlation between $\dot{V}O_{2max}$ percent predicted and FEV_1 percent predicted ($p < 0.02$), and a negative correlation between FEV_1 and end-expiratory lung volume (EELV) at maximal exercise ($p < 0.03$). There was no significant correlation between FEV_1 and maximal HR achieved during exercise; moreover, all subjects achieved a maximal HR in excess of 80% predicted, suggesting a cardiovascular limitation to exercise. These data do not support the hypothesis that the lower initial $\dot{V}O_{2max}$ in the subjects with a reduced FEV_1 was due to deconditioning. Although increased EELV at maximal exercise, reduced $\dot{V}O_{2max}$, and a reduced $\dot{V}O_{2max}$ response with training are all statistically associated with a reduced FEV_1 , there is no direct evidence of causality. Babb TG, Long KA, Rodarte JR. The relationship between maximal expiratory flow and increases of maximal exercise capacity with exercise training.

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It is well documented that patients with severe chronic obstructive lung disease have a limited exercise capacity (2-8). It is believed that normal, young subjects have a large ventilatory reserve, and that maximal exercise capacity in this popu-

lation is due to cardiovascular limitation. The effects of mild airway obstruction are less well documented. Our laboratory previously demonstrated that compared with age-matched normal controls, patients with mild chronic obstructive pulmonary disease (COPD) achieved the same maximal heart rate and "ventilatory reserve," as judged by the ratio of maximal exercise ventilation to maximal voluntary ventilation (MVV), as age-matched normal controls, but that their maximal oxygen uptake ($\dot{V}O_{2max}$) was significantly reduced, roughly in proportion to the reduction in FEV_1 (1). Since the COPD patients met the usual criteria for cardiovascular limitation to exercise (9), their reduced exercise capacity, in the absence of any evidence of cardiac disease, may have been due to a habitual

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Correspondence and requests for reprints should be addressed to T. G. Babb, Ph.D., Institute for Exercise and Environmental Medicine, Presbyterian Hospital of Dallas, 7232 Greenville Avenue, Dallas, TX 75231.

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lower level of activity. Since there was no obvious mechanism by which the amount of airflow obstruction and abnormal breathing mechanics in the COPD subjects should affect cardiac function, a plausible explanation for these results was that the abnormal respiratory mechanics produced subliminal symptoms, which caused the patients to reduce their activities of daily living (ADL) as compared with the control group. We therefore conducted the present study to confirm the previous findings of reduced exercise capacity in subjects with very mild reductions of maximal expiratory flow (1) and to examine the effect of an exercise training program on exercise capacity. Since in the previous study FEV_1 and $\dot{V}_{O_{2max}}$ were also inversely correlated with end-expiratory lung volume (EELV) at maximum exercise (1), we also determined EELV before and after training.

METHODS

Subjects

The 11 subjects (58 ± 8 yr of age [mean \pm SD]) in this study were selected from volunteers who considered themselves well and were recruited through local advertisements, or from subjects receiving routine occupational medicine examinations. All subjects had normal electrocardiograms (ECGs) and no history of asthma, cardiovascular disease, or musculoskeletal abnormalities that would preclude maximal exercise. Subjects were selected whose pulmonary function ranged from mild chronic airflow limitation to above normal, according to American Thoracic Society (ATS) guidelines and whose TLC was $\geq 90\%$ predicted. None of the subjects had significant increases in FEV_1 with inhaled bronchodilator ($> 15\%$, ATS guidelines). One subject had an FEV_1 of 61% predicted; all others had FEV_1 values that ranged from 70% to 114% predicted, with a mean of 90% predicted and an SD of 18% predicted. Six of the 11 subjects had an FEV_1 above 80% predicted. This group contained one of the two women in the study, and did not differ significantly from the subjects with lower FEV_1 values with regard to age, height, weight, FVC percent predicted, or TLC. The subjects with $FEV_1 > 80\%$ predicted had a smoking history of 41 ± 27 pack-yr, whereas the subjects with $FEV_1 < 80\%$ predicted had a smoking history of 51 ± 23 pack-yr. Two current and three prior smokers had an FEV_1 greater than 80% predicted, and three current cigarette smokers, one pipe smoker, and one ex-smoker had FEV_1 values of less than 80% predicted. None of the subjects had participated in regular vigorous exercise for the 6 mo preceding the study. Subject characteristics and pulmonary function values are shown in Table 1.

Maximal Exercise

Graded cycle ergometry was performed on an electronically braked cycle ergometer (Medical Graphics, St. Paul, MN), using 1-min, 20-W or 30-W increments in work rate. The subjects pedaled at a rate of 60 to 80 rpm. Subjects were encouraged to exercise to exhaustion. Measurements of minute ventilation (\dot{V}_E), V_T , breathing frequency (f_b), \dot{V}_{O_2} , and carbon dioxide production (\dot{V}_{CO_2}) were made with the use of a computerized, breath-by-breath system (Medical Graphics 2001). Gas-exchange measurements were made during each work increment. Calibration of the analyzer was done with reference gases before each test. For each patient, the ECG was monitored continuously and blood pressure was monitored at each work rate during the exercise test. Maximal exercise tests were performed before and after 10 wk of supervised exercise training.

Ventilatory threshold (VTh) was determined from gas exchange measurements, using a value consistent with both the method described by Caiozzo and others (10) and the V-slope method as described by Wassermann and others (9).

To measure flow, volume, and transpulmonary pressure (P_{TP}) continuously during the maximal exercise test, a Hans Rudolph valve (Model 2700) was connected to separate inspiratory and expiratory pneumotachographs (No. 3 Fleisch pneumotachographs, Switzerland, and Celesco transducers, Canoga Park, CA, ± 2 cm H_2O). Expired gas was directed to the exercise gas analysis system. The pneumotachograph in the expiratory line does not affect the accuracy of the gas exchange measurements (1). The separate expiratory and inspiratory flow signals were electronically summed to give a single bidirectional flow signal, and volume was determined from the integration of the single flow signal. The flow resistances of the inspiratory and expiratory circuits were less than 1.0 cm H_2O per $L \cdot s^{-1}$ for flows of $\pm 10 L \cdot s^{-1}$. The pneumotachographs were checked for linearity before the study began, using known flow rates, and were calibrated before each test with a calibrated syringe. An esophageal balloon was placed 45 cm from the nares and connected to a Celesco (± 100 cm H_2O) pressure transducer, the negative port of which was connected to a pressure tap on the mouthpiece, to measure P_{TP} . Balloon volume and placement were checked by having the subject make respiratory efforts with the airway occluded to confirm equal changes in airway opening and esophageal pressure (P_{es}). Flow, volume, and P_{TP} were displayed on a strip chart recorder (HP-7414A; Hewlett-Packard, Inc., Palo Alto, CA), and were sampled in real time (100 Hz) on a computer (DEC 11/73; Digital, Dallas, TX). A noseclip was worn during rest and exercise data collections.

EELV was estimated at rest and during maximal exercise from measurement of inspiratory capacity (IC). IC was measured during the last 10 s of each exercise increment. Measurement of IC was performed by having subjects inhale maximally to TLC (on cue from the investigator). A maximal inspiratory effort was confirmed by comparing maximal P_{TP} during the IC maneuver with maximal static recoil pressure determined at baseline. We assumed that TLC did not change significantly during exercise in the control subjects or patients (8, 11, 12). All subjects were able to perform the procedure without difficulty.

Maximal and tidal flow-volume and pressure-volume loops were determined at rest, while the subjects were seated on the cycle ergometer just before the baseline measurements, and within 1 min after terminating exercise to determine if exercise had induced bronchodilation. Tidal flow-volume and pressure-volume loops were measured at each work increment. A typical tidal flow-volume and corresponding pressure-volume loop was chosen from the breaths preceding the maximal inspiration, and was positioned within the maximal flow-volume loop, using the measured IC. A breath was considered typical if it had similar volume and flow characteristics to the other breaths prior to the IC maneuver. All measurements were made before and after 10 wk of exercise training.

Submaximal Exercise

On separate days, the subjects were exercised for 30 min at a constant work rate, to determine the highest exercise level the subject could sustain for 30 min without the subject's heart rate (HR) exceeding 90% of that achieved during the incremental maximal exercise test. We had previously determined that this is very close to the maximum work rate subjects can sustain. During the first practice session, the work rate was equal to or slightly greater than the workload of VTh as determined during the maximal graded exercise test. If HR exceeded 90% of the subject's observed maximal HR, or if the subject could not tolerate the exercise for 30 min, then a lower workload was tried on

TABLE 1

PHYSICAL CHARACTERISTICS AND PULMONARY FUNCTION OF SUBJECTS*

Subjects	Age (yr)	Height (cm)	Weight (kg)	FVC (% pred)	FEV_1 (% pred)	FEV_1/FVC (%)	MVV (% pred)	TLC (% pred)
(9M, 2W)	58 ± 8	176 ± 9	91 ± 17	103 ± 10	90 ± 18	73 ± 15	103 ± 17	111 ± 11

Definition of abbreviation: MVV = maximal voluntary ventilation.

* Values are mean \pm SD. There were no significant changes in pulmonary function after training.

another practice day. If HR did not approach 90% of maximal HR, then the workload was increased at the next exercise practice session. All subjects had at least two practice sessions before the submaximal testing session.

Both at rest and during exercise the subjects breathed through the same apparatus as used in the maximal exercise test, and gas exchange measurements were made in the same manner as used in the maximal exercise test. The ECG was monitored continuously and recorded every 5 min, as were gas exchange measurements. Blood pressure was taken every 5 min. A pedal rate of approximately 60 rpm was maintained.

An esophageal balloon was placed as during maximal testing, and flow, volume, and PTP were monitored continuously during the submaximal exercise. Maximal and tidal flow-volume and pressure-volume loops were determined at rest; tidal flow-volume and pressure-volume loops were monitored continuously during the submaximal exercise, processed as outlined for maximal exercise, and recorded every 5 min for 30 min of submaximal exercise. EELV was estimated at rest and during the submaximal exercise from measurement of IC.

The testing procedure began with the subjects seated on the cycle ergometer while baseline measurements were made. After 2 min of baseline measurements, the subjects performed constant-load cycle ergometry. Exercise began with 3 min of warm-up exercise at 20 or 30 W; thereafter, the work rate was increased over 1 min to the established work rate and the subjects exercised for 30 min. Submaximal tests were performed before and after 10 wk of exercise training.

Training

All subjects participated in a supervised exercise training program for 10 wk. They cycled 30 min/d on 3 d/wk at the target HR selected as described earlier. Each exercise session was monitored by an exercise physiologist to assure that each subject maintained his or her assigned workload for the entire exercise period. The HR was measured every 5 min, or six times over the 30-min session, and the workload was adjusted if needed. The mean HR for each of the 30 sessions was averaged as an index of the intensity of training. For those subjects whose exercise HR fell during the 10-wk program, the power output was increased to maintain HR within the target range. Six subjects completed all 30 sessions in 10 wk. Three of the subjects with a high FEV₁ completed 29, 29, and 28 sessions, respectively, and two of the subjects with a low FEV₁ completed 28 sessions each. There was no relationship between the number of training sessions completed and the change in $\dot{V}_{O_{2max}}$ with training. Nor was there a significant difference in the number of training sessions completed by subjects with a high FEV₁ and subjects with a low FEV₁. Overall, the subjects completed 29.3 ± 0.9 training sessions. The mean HR for all subjects averaged $80 \pm 6\%$ of that observed during the maximal exercise test. Because the HR gradually increased during the 30 min of exercise, the mean HR was less than the goal of an HR of 90% at the end of exercise. The mean HR during the training sessions did not correlate with the percent predicted FEV₁, indicating that all subjects trained at equal intensities.

Data Analysis

V_T , f_b , \dot{V}_E , inspiratory time (T_I), and expiratory time (T_E) were calculated from the volume signal by an interactive computer program developed in our laboratory. An investigator using the interactive computer program screened the computer-stored data and played the data back on a graphics terminal to generate exercise flow-volume loops and pressure-volume loops. Within-subject differences before and after training were examined with paired *t* tests. Relationships between variables across subjects were examined with linear regression analysis.

RESULTS

In accord with our previous results, maximal exercise capacity as evidenced by $\dot{V}_{O_{2max}}$ percent predicted and maximal exercise ventilation were significantly correlated with FEV₁ percent predicted ($p < 0.02$). The relationship between $\dot{V}_{O_{2max}}$ and FEV₁ is shown in Figure 1. There was no significant relationship between FEV₁ percent predicted and percent predicted maximal HR achieved during exercise. There was a sta-

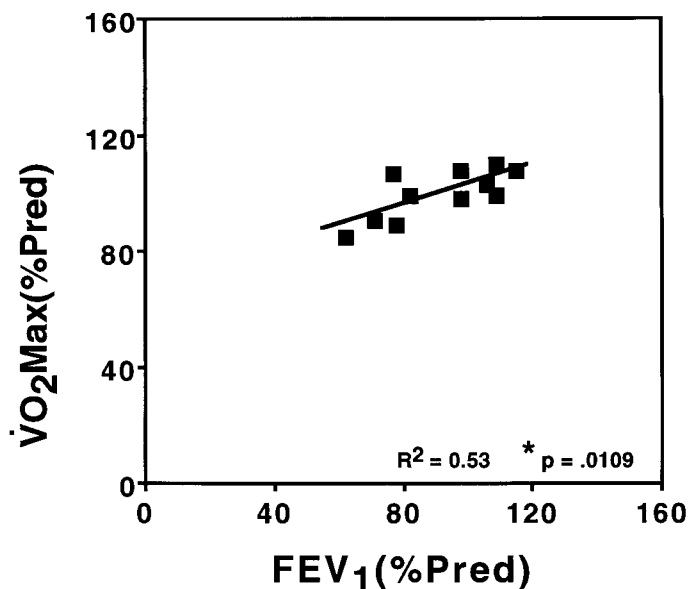


Figure 1. Relationship between exercise capacity and pulmonary function. $\dot{V}_{O_{2max}}$ during the incremental test prior to exercise training expressed as percent predicted is plotted against FEV₁ as percent predicted. There is a significant positive correlation between the two variables, indicated by the regression line plotted on the figure: $y = 69.7 + 34x$; $R^2 = 0.53$, $p = 0.01$. This relationship predicts a $\dot{V}_{O_{2max}}$ of 100% predicted at an FEV₁ of 89% predicted.

tistically significant inverse relationship between FEV₁ percent predicted and EELV expressed as a percent of TLC, in accord with previous observations (1) (Figure 2; $p < 0.05$).

The subjects found it very difficult to sustain the exercise intensity for the entire 30-min exercise period. The HR during training, averaged over the 30-min session and over the 30 sessions, was $80 \pm 6\%$ of the maximum HR observed during the

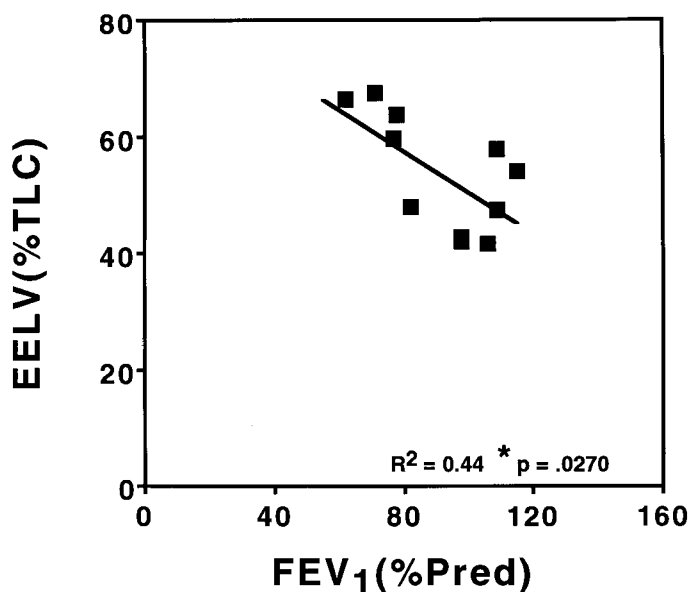


Figure 2. The relationship between end-expiratory lung volume (EELV) and pulmonary function. EELV during maximal exercise before training is plotted against FEV₁ as percent predicted as a significant inverse correlation, demonstrated by the regression line plotted in the figure: $y = 87 - 0.36x$; $R^2 = 0.44$, $p < 0.03$.

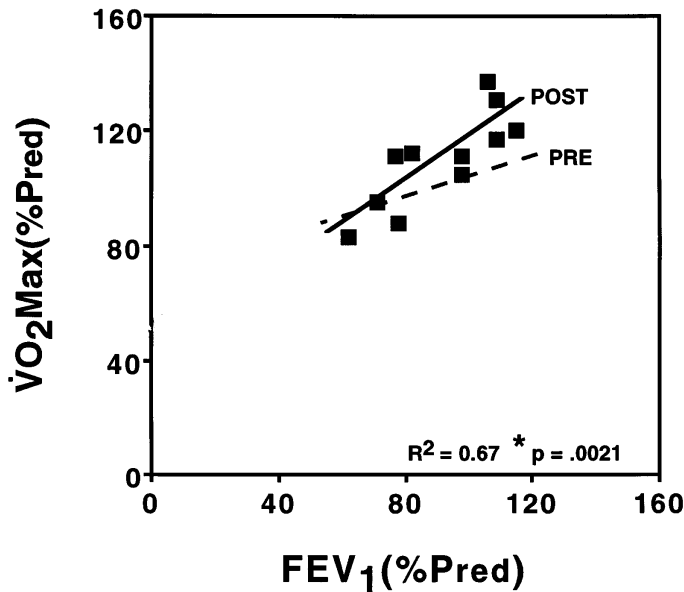


Figure 3. The relationship between maximal exercise capacity after training and pulmonary function. $\dot{V}O_{2max}$ during a 1-min incremental exercise test after the training program is plotted against FEV_1 as percent predicted. Because there was no significant change in FEV_1 with training, the pretraining values are used to facilitate the comparison with Figure 1. The pretraining regression equation from Figure 1 is indicated by the *dashed line* and the posttraining regression equation is indicated by the *solid line*: $y = 42 + 0.76x$; $R^2 = 0.67$, $p = 0.002$. Regression equations intersect at FEV_1 of 66% predicted, indicating that those subjects with higher FEV_1 increased their $\dot{V}O_{2max}$ with training.

incremental exercise test. The initial training level was at a workload that was $118 \pm 14\%$ of the VTh. During the pretraining 30-min steady-state exercise test, there was a 3.4 ± 8 L/min drift in \dot{V}_E and a 16.5 ± 7 beat/min drift in HR between Min 5 and Min 30. Neither the HR as a percent predicted or observed maximum value, nor the workload relative to VTh, or the ventilatory or HR drift was correlated with FEV_1 , suggesting that the subjects trained at the same relative intensity, as judged by these criteria.

The training program resulted in an increase in $\dot{V}O_{2max}$ percent predicted from $100.7 \pm 8.5\%$ to $111.0 \pm 16.7\%$ predicted ($p = 0.01$). \dot{V}_E (82 to 94 L/min) and work rate (173 to 204 W) during maximal exercise were also increased after training ($p < 0.05$). The training program had no effect on any parameters of lung mechanics (FEV_1 , VC, FVC, TLC). After training, the relationship between FEV_1 percent predicted and $\dot{V}O_{2max}$ was stronger (Figure 3; $p < 0.01$). R^2 increased from 0.53 to 0.67, and the slope was significantly steeper, indicating that the increase in $\dot{V}O_{2max}$ was a function of the FEV_1 (Figure 4). The intersection of the two regression lines for $\dot{V}O_{2max}$ before and after training in Figure 3, and the zero value for the increase in $\dot{V}O_{2max}$ in Figure 4, occurred at an FEV_1 of 66% predicted. After exercise training, the correlation between FEV_1 percent predicted and EELV at maximal exercise was stronger ($R^2 = 0.44$ to 0.71), but the inverse relationship was less steep (Figure 5). The difference in EELV at maximal exercise between the pretraining and posttraining tests (an increase from 54% to 59% TLC, $p < 0.05$) was significantly correlated with FEV_1 percent predicted ($p < 0.03$; data not shown). There was an increase in VTh of $9.4 \pm 6\%$ of $\dot{V}O_{2max}$ after training

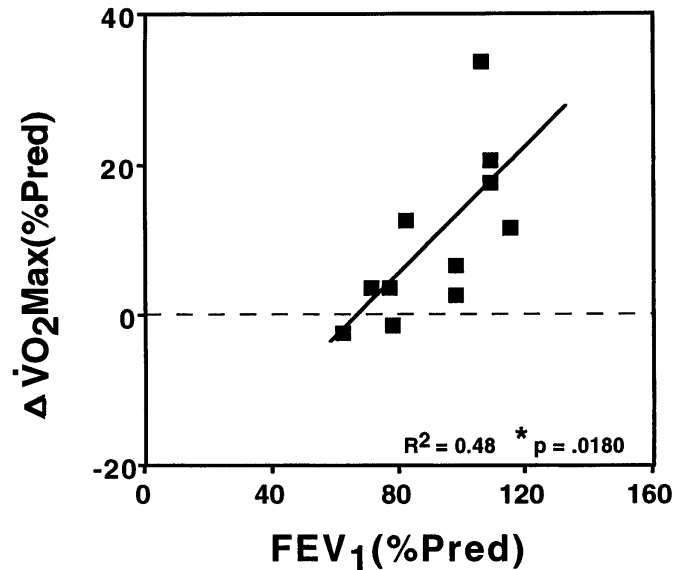


Figure 4. Relationship between the effect of exercise training on exercise capacity and pulmonary function. The difference in $\dot{V}O_{2max}$ during an incremental test before and after a conditioning program is plotted against FEV_1 as percent predicted. There is a highly significant relationship between $\Delta \dot{V}O_{2max}$ and FEV_1 , as shown by the plotted regression equation: $y = -27 + 0.42x$; $R^2 = 0.48$, $p < 0.02$. This positive relationship is not consistent with there being a constant fractional increase in $\dot{V}O_{2max}$, since subjects with the lowest FEV_1 values and lowest initial $\dot{V}O_{2max}$ had no increase or even a slight decrease in $\dot{V}O_{2max}$ with training.

($p < 0.001$). The correlation between the increase in VTh and FEV_1 percent predicted was not significant.

During the submaximal exercise session before training, mean HR was $71 \pm 7\%$ of the predicted maximum HR, and $\dot{V}O_2$ at the end of exercise was $73 \pm 6\%$ of the observed $\dot{V}O_{2max}$. Neither correlated with FEV_1 . At the end of the training session, when tested at the same work rate, mean HR decreased to $65 \pm 8\%$ of the predicted maximum ($p < 0.02$). There was no significant change in the ventilatory drift during steady-state exercise, but there was a 5.0 ± 6.5 beat/min decrease in HR drift ($p < 0.03$) between 5 and 30 min of steady exercise. The decrease in HR drift was significantly correlated with FEV_1 percent predicted ($p < 0.01$), but the decrease in HR was not significant ($r = -0.44$).

DISCUSSION

In this study of individuals who considered themselves well, with airflows ranging from mild airway obstruction to greater than the predicted normal value, we confirmed our previous results that FEV_1 percent predicted is strongly associated with $\dot{V}O_{2max}$ percent predicted, and is inversely correlated with EELV as percent TLC during maximal exercise (1). The ratio of maximal exercise ventilation to MVV in subjects with above-average and reduced flows is similar. The ratio of ventilation to oxygen consumption is normal and $\dot{V}O_{2max}$ is lower in subjects with reduced FEV_1 than in individuals with higher flows. In this study, the association between FEV_1 percent predicted and $\dot{V}O_{2max}$ percent predicted was demonstrated over a smaller range of flows than in previous studies (coefficient of variation [CV] of FEV_1 percent predicted of 20%). FEV_1 percent predicted is significantly correlated with both $\dot{V}O_{2max}$ and EELV during maximum exercise before and after

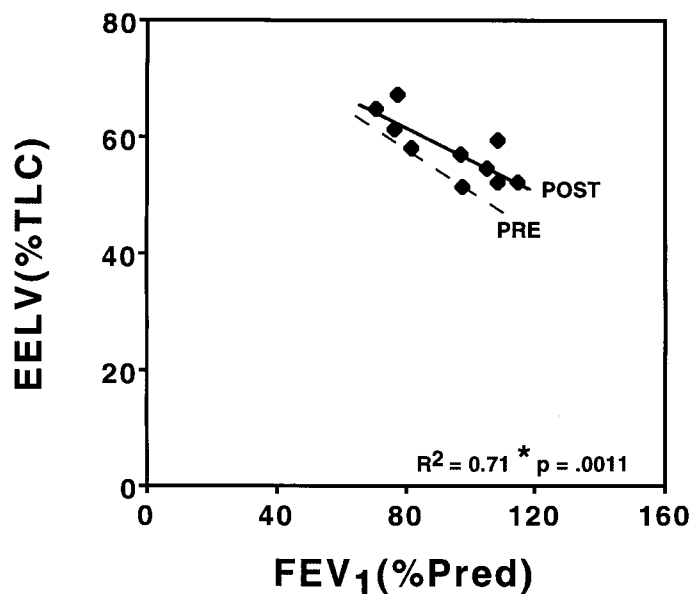


Figure 5. The relationship between end-expiratory lung volume (EELV) during maximal exercise after training and pulmonary function. EELV as percent predicted TLC after an exercise training program was plotted against FEV₁ as percent predicted. There is a strong inverse relationship shown by the *solid regression line*: $y = 84 - 0.27x$; $R^2 = 0.71$, $p = 0.001$. The relationship before exercise training is shown by the *dashed line*. Although the variance in EELV is reduced after exercise training, the variances better fit FEV₁, as evidenced by the increase in R^2 from 0.44 to 0.71.

exercise training. These associations are stronger after training, as judged by an increased R^2 . However, such correlations do not prove cause and effect.

The association between FEV₁ and $\dot{V}O_{2max}$ was strengthened rather than reduced by a conditioning program. The relative intensity of the exercise training was constant in all subjects as judged by average HR during training, workload relative to V_{Th} measured by an incremental exercise test, and the drift of $\dot{V}E$ and HR during the pretraining steady-state exercise test. There was no correlation between any of these parameters and percent predicted FEV₁, suggesting that subjects with different FEV₁ values began training at equal intensities. Our exercise program was consistent with conventional standards for prescribing exercise according to the guidelines of the American College of Sports Medicine (13). All subjects were closely monitored to insure that they continuously exercised at the prescribed level, which was above the V_{th} identified on the 1-min incremental maximal exercise test. Most subjects found it quite difficult to maintain the prescribed level of exercise for the entire 30 min. The subjects with the lower percent predicted FEV₁ values also had a lower percent predicted $\dot{V}O_{2max}$ and trained at a lower absolute exercise intensity, but at the same relative intensity as the individuals with higher flows. If the lower level of fitness was due only to a reduced level of daily activity, one would expect such individuals to benefit at least as much from a conditioning program as the individuals with higher flows, if not more so.

Overall, our subjects showed significant conditioning with training, as judged by an increase in $\dot{V}O_{2max}$ without a change in maximal HR, an increase in V_{Th} during an incremental exercise test, and a decrease in average HR and HR drift during steady-state exercise. The increase in $\dot{V}O_{2max}$ and the decrease in HR drift were correlated with percent predicted FEV₁ ($p < 0.02$).

For two subjects with a lower FEV₁ percent predicted who demonstrated little or no effect of training, we obtained the results of Doppler flow studies of major arteries serving the legs, and stress ultrasound examinations of the heart, which revealed normal function of both the left and right ventricles. One of these individuals had an additional 5 wk of training in 30-min sessions for 3 d/wk at the highest intensity he could sustain. His mean HR during the additional 5 wk was less than 10% greater than during the initial 10 wk, indicating that the exercise during the initial period was very near the highest intensity he could sustain for 30 min. Repeat maximal exercise testing at the end of the 15th week was not different from the initial or the 10-wk tests.

In contrast to what would be expected if the lower $\dot{V}O_{2max}$ of the subjects with lower FEV₁ values were due to a reduced level of daily activity, a supervised, strenuous 10-wk conditioning program increased rather than reversed the correlation between $\dot{V}O_{2max}$ and FEV₁. Those individuals with lower percent predicted FEV₁ values had little or no increase in $\dot{V}O_{2max}$ with an exercise program of high relative intensity. Is the lower $\dot{V}O_{2max}$ and $\dot{V}E$ during maximal exercise in patients with lower FEV₁ values due to some ventilatory limitation? Our indices of a conditioning effect during submaximal exercise are more variable than $\dot{V}O_{2max}$ and less strongly associated with FEV₁. Casaburi and colleagues (14), in a study of patients with severe airway obstruction, reported training effects during submaximal exercise, but not an increased $\dot{V}O_{2max}$ (14). Sridhar and associates (15), in a study of patients with moderate airflow obstruction, reported that during incremental exercise, added dead space decreased maximal exercise capacity rather than increasing maximal $\dot{V}E$. Brown and coworkers (16) reported that in COPD patients, although dead space increased maximal $\dot{V}E$, exercise capacity was decreased and Pa_{CO₂} was increased. In contrast, normal subjects studied by McParland and associates (17) and Johnson and coworkers (18–20) demonstrated increased maximal $\dot{V}E$ with inspiration of CO₂ during exercise. Ventilatory limitation is a complex phenomenon. Patients with resting CO₂ retention can voluntarily increase their $\dot{V}E$, reducing their CO₂ for brief periods. Consequently, ventilatory limitation is not as simple as an absolute inability to increase ventilation.

In the current study, subjects who had a decrease in HR during the 10-wk training program increased their steady-state workload so that although relative work intensity as judged by HR was held constant, absolute work intensity was not. This occurred in subjects with higher FEV₁ values, who increased their $\dot{V}O_{2max}$, so that the slope of the relationship between FEV₁ percent predicted and $\dot{V}O_{2max}$ became steeper. Dynamic hyperinflation occurs during exercise in older normal subjects and patients with airflow obstruction (1). After training, the subjects in our study with a higher FEV₁ who increased their $\dot{V}O_{2max}$ and maximal exercise $\dot{V}E$ also increased their EELV, so that the relationship between EELV and FEV₁, although more highly correlated at a lower slope, suggested that their exercise $\dot{V}E$ relative to their "ventilatory capacity" became more similar. Although far from conclusive, this study suggests that in older individuals, $\dot{V}E$ plays more of a role in maximal exercise capacity than has been previously appreciated.

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