

Mild obesity does not limit change in end-expiratory lung volume during cycling in young women

T. G. BABB, D. S. DeLOREY, B. L. WYRICK, AND P. P. GARDNER
*Institute for Exercise and Environmental Medicine, Presbyterian Hospital of Dallas
and The University of Texas Southwestern Medical Center, Dallas, Texas 77231*

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Babb, T. G., D. S. DeLorey, B. L. Wyrick, and P. P. Gardner. Mild obesity does not limit change in end-expiratory lung volume during cycling in young women. *J Appl Physiol* 92: 2483–2490, 2002; 10.1152/jappphysiol.00235.2001.—To investigate the effects of obesity on the regulation of end-expiratory lung volume (EELV) during exercise we studied nine obese ($41 \pm 6\%$ body fat and 35 ± 7 yr, mean \pm SD) and eight lean ($18 \pm 3\%$ body fat and 34 ± 4 yr) women. We hypothesized that the simple mass loading of obesity would constrain the decrease in EELV in the supine position and during exercise. All subjects underwent respiratory mechanics measurements in the supine and seated positions, and during graded cycle ergometry to exhaustion. Data were analyzed between groups by independent *t*-test in the supine and seated postures, and during exercise at ventilatory threshold and peak. Total lung capacity (TLC) was reduced in the obese women ($P < 0.05$). EELV was significantly lower in the obese subjects in the supine (37 ± 6 vs. $45 \pm 5\%$ TLC) and seated (45 ± 6 vs. $53 \pm 5\%$ TLC) positions and at ventilatory threshold (41 ± 4 vs. $49 \pm 5\%$ TLC) ($P < 0.01$). In conclusion, despite reduced resting lung volumes and alterations in respiratory mechanics during exercise, mild obesity in women does not appear to constrain EELV during cycling nor does it limit exercise capacity. Also, these data suggest that other nonmechanical factors also regulate the level of EELV during exercise.

ventilation; control of breathing; lung volumes; pulmonary function

OBESITY IS A MAJOR HEALTH CONCERN facing today's society. If the international standard of obesity [body mass index (BMI) of >30 kg/m²] is applied to the U.S. population, ~22% of Americans would be considered obese (17). Because exercise is a major component of weight loss and maintenance programs, documenting the effects of mild to moderate obesity could have a significant public health outcome.

Respiratory complications of morbid obesity ($>50\%$ body fat) are well known and include such mechanical constraints as decreased chest wall compliance, increased respiratory resistance, increased work of breathing, reduced lung volumes, sleep apnea, and hypoventilation syndrome (5, 24, 26–29). However, relatively little is known about the effects of mild to

moderate obesity on respiratory function at rest or during exercise (5, 6).

The earliest and most prominent change in pulmonary function with mild to moderate obesity is a reduction in end-expiratory lung volume (EELV) (29). Rather than functional residual capacity, which is specific to static respiratory mechanics, we use EELV, which is determined by both respiratory mechanics and respiratory muscle recruitment during exercise. EELV adopted during exercise is also influenced by expiratory flow limitation (4). The response of EELV during exercise is important, therefore, because it is a major component of the normal ventilatory response to exercise and reflects alterations in respiratory mechanics during exercise. EELV adopted during exercise has serious implications for tidal expiratory flow, respiratory muscle function, work of breathing, and/or shortness of breath (4). The determinants of EELV during exercise in mild to moderate obesity are unknown.

During treadmill walking, EELV remains low and unchanged in mildly obese women in contrast to that in lean women who decrease their EELV during exercise (5). This altered response of EELV during exercise in obesity means that the inspiratory muscles must assume all the respiratory work to increase tidal volume (V_T). Furthermore, if EELV were reduced further, the potential for expiratory flow limitation during exercise would be increased. The factors controlling the lower limits of EELV during exercise in mild to moderate obesity could be mechanical or compensatory in nature. We hypothesized that the simple mass loading of obesity constrains the decrease in EELV during exercise. The purpose of this study was to examine the effects of mild to moderate obesity on EELV and respiratory mechanics at rest, while supine and during exercise.

METHODS

Subjects. Two groups of women were recruited through local advertisements. Nine obese ($>30\%$ body fat) and eight lean ($<25\%$ body fat) subjects were included for study. In accordance with the institutional review board, all details of the study were discussed with the volunteers, and informed consent was obtained. All qualified participants were famil-

Address for reprint requests and other correspondence: T. G. Babb, Institute for Exercise and Environmental Medicine, 7232 Greenville Ave., Ste 435, Dallas, TX 75231 (E-mail: TonyBabb@TexasHealth.org).

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iarized to exercise on the cycle ergometer and instructed to avoid exercise, food, caffeine, and smoking for at least 2 h before exercise testing.

No subject had a history of asthma, cardiovascular disease, or musculoskeletal abnormalities that would preclude maximal exercise or had participated in regular vigorous exercise for the last 6 mo. Subjects not meeting these guidelines were excluded as well as individuals with respiratory symptoms.

Study protocol. Pulmonary function tests, resting electrocardiograms (ECG), and body composition tests were performed as an initial screening. If subjects met inclusion criteria for the study, they returned to the laboratory on two separate occasions for maximal exercise testing. The first exercise test served as a familiarization to the cycle ergometer and all testing procedures. At least 1 wk separated the two maximal exercise tests. After completion of the exercise tests, subjects returned to the laboratory once more for detailed pulmonary mechanics measurements.

Body composition. Standard measures of height and weight were made during initial screening of subjects. BMI and weight-to-height ratio were calculated from these measures. After enrollment in the study, waist, hip, bust, and chest circumferences were measured. Waist-to-hip ratio was calculated from the circumference data. Hydrostatic weighing was performed to determine percent body fat, lean body mass, and fat mass.

Pulmonary function. All subjects had standard spirometry, lung volume, and diffusing capacity determinations (model 6200 body plethysmograph, SensorMedics, Yorba Linda, CA). Pulmonary function was performed according to the guidelines of the American Thoracic Society (1). Predicted values were based on the norms of Knudson and colleagues (20, 21), Goldman and Becklake (18), and Burrows et al. (10). Maximal flow-volume loops were measured in a pressure-corrected volume-displacement body plethysmograph to eliminate the gas compression artifact (SensorMedics model 6200). Exercise tidal flow-volume loops were compared with this maximal flow-volume loop.

Gas exchange measurements. Measurements of oxygen uptake ($\dot{V}O_2$) and carbon dioxide production were made with the use of a computerized custom gas exchange system as described previously (2). System resistance was $<2 \text{ cmH}_2\text{O} \cdot \text{l}^{-1} \cdot \text{s}$ through 6 l/s for expiration. Ventilatory threshold (V_{Th}) was determined from the comparison of gas exchange indexes (11) and the V-slope method (32). V_{Th} was designated as the work rate most congruent among the different threshold determination methods.

Breathing mechanics. Expiratory and inspiratory flow were measured at rest and continuously during exercise as described previously (3). Inspiratory capacity (IC) was measured at rest and during exercise to determine placement of tidal flow-volume loops within the maximal flow-volume loop as previously described (2, 3). EELV was estimated from measurement of IC [EELV = total lung capacity (TLC) - IC] and reported as a percentage of TLC [(EELV/TLC) \times 100]. End-inspiratory lung volume (EILV) was calculated (EILV = EELV + V_T) and expressed as a percentage of TLC [EILV/TLC \times 100]. This assumes that TLC does not change significantly with body position (8) or exercise (7, 31, 35). Transpulmonary pressure (Ptp) was estimated as the differential pressure between oral and pleural pressure, which was measured with an esophageal balloon placed ~ 45 cm from the nares (Validyne pressure transducer, model MP45 \pm 100 cmH_2O , Northridge, CA). By convention, inspiratory efforts were negative in direction, and expiratory efforts were positive in direction. Validity of the balloon pressure was checked by having the subjects blow through a small orifice; if Ptp

remained constant while oral pressure increased, Ptp was considered appropriate. This check was done each time the subject changed body position. Gastric pressure (Pga) was measured with a balloon placed ~ 65 cm from the nares (Validyne, model MP45 \pm 340 cmH_2O). The pressures were displayed on a strip chart recorder (AstroMed, model MT 95000, Warwick, RI) and sampled in real time (100 Hz) on a computer (486Dx).

Exercise protocol. Testing began with the subjects seated on the cycle ergometer while baseline measurements were made. After 3 min of baseline measurements, subjects performed graded cycle ergometry on an electronically braked cycle ergometer (model CPE 2000, MedGraphics, St. Paul, MN). Initial work rate was 20 W, and work rate was increased each minute by 20 W. Test termination criteria included volitional exhaustion or a pedal rate of ≤ 50 rpm. Gas exchange measurements were made during each increment in work rate. IC was measured during the last 20 s of each exercise increment, and tidal flow-volume loops were measured continuously. ECG was monitored continuously through the use of a 12-lead ECG (model CS 100, Schiller, Baar, Switzerland), and blood pressure was monitored with the use of an automated system (Suntech 4240, Raleigh, NC). Maximal flow-volume loops were determined at rest, while subjects were seated on the cycle ergometer, just before baseline measurements and within 2 min after termination of exercise to determine whether exercise had induced bronchodilation or bronchoconstriction, which none of the subjects had.

Data analysis. V_T , breathing frequency (f), minute ventilation (\dot{V}_E), and exercise tidal flow-volume and pressure-volume loops were determined with the use of an interactive computer program as described previously (2, 3). Also calculated was expiratory airflow limitation, defined as the percentage of V_T (% V_T) where tidal expiratory flow impinged on maximal expiratory flow and where Ptp simultaneously exceeded the minimal critical pressure necessary to obtain maximal flow (2, 3). This traditionally definitive technique produces reliable estimates of expiratory flow limitation even compared with the newer negative expiratory pressure technique (25). Data were analyzed at rest, at V_{Th} , and during peak exercise.

The ventilatory response to exercise was determined on all points between rest and V_{Th} (below V_{Th}), and between V_{Th} and peak exercise (above V_{Th}) by least-squares regression ($\text{l} \cdot \text{min}^{-1} \cdot \text{W}^{-1}$) as previously described (2, 3). The fit of these data by least-squares regression was considered good on the basis of the average coefficient of determination (R^2), which below V_{Th} was 0.96 ± 0.04 and 0.98 ± 0.03 , and above V_{Th} was 0.97 ± 0.02 and 0.96 ± 0.03 for the lean and obese groups, respectively.

Differences between groups were determined by an independent *t*-test. Relationships among variables were determined with Pearson correlation coefficients. A *P* value of <0.05 was considered significant.

RESULTS

Subjects. Subject characteristics are shown in Table 1. Nine healthy mild to moderately obese women and eight lean women were studied. Weights, percent body fat, BMI, waist and hip circumferences, waist-to-hip ratios ($P < 0.05$), and height-to-weight ratios were all significantly greater ($P < 0.0001$) in the obese group. No differences were noted for age and height. All subjects in the lean group were nonsmokers, whereas two

Table 1. *Subject characteristics*

	Age, yr	Ht, cm	Wt, kg	BF, %	Waist, in.	Hip, in.	Waist/Hip	Wt/Ht	BMI, kg/m ²
Lean (<i>n</i> = 8)	34 ± 4	161 ± 6	57 ± 7	18 ± 3	26 ± 2	35 ± 2	0.75 ± 0.03	0.35 ± 0.04	22 ± 3
Obese (<i>n</i> = 9)	35 ± 7	168 ± 8	98 ± 16	41 ± 6	40 ± 5	48 ± 4	0.83 ± 0.08	0.58 ± 0.08	34 ± 4
<i>P</i> value	ns	ns	0.001	0.001	0.001	0.001	0.05	0.001	0.001

Values are means ± SD. Ht, height; wt, weight; BF, percent body fat; waist, circumference at waist; hip, circumference at hip; waist/hip, ratio of waist to hip circumferences; wt/ht, ratio of weight to height (kg/cm); BMI, body mass index; ns, nonsignificant.

subjects in the obese group were currently smoking (1.4 ± 1.5 pack·yr for $n = 8$; individually 4.5 and 5 pack·yr).

Pulmonary function. Pulmonary function data are presented in Table 2. All subjects had normal spirometry on the basis of predicted values. Relative to the lean subjects, forced expiratory volume in 1 s (FEV₁) as a percentage of predicted volume was also significantly lower ($P < 0.05$) in the obese group. Additionally, TLC and residual volume as a percentage of predicted volume were significantly lower ($P < 0.05$) in the obese group (Fig. 1). The reduction observed in FEV₁ in the obese women is likely related to their reduced TLC. In absolute terms, functional residual capacity, thoracic gas volume, and expiratory reserve volume were significantly lower ($P < 0.05$) in the obese group. Diffusing capacity of the lung for carbon monoxide (DL_{CO}) as a percentage of that predicted was also significantly ($P < 0.001$) reduced in the obese subjects. Correcting DL_{CO} for alveolar volume resulted in DL_{CO}/alveolar volume as the percentage of that predicted being significantly higher ($P < 0.05$) in the obese subjects. TLC (%predicted) was significantly correlated ($P \leq 0.05$) with only body weight ($r = -0.48$) and waist circumference ($r = -0.49$) but not with percent body fat or BMI, suggesting that these measures are not sensitive enough to predict the change in lung function with mild obesity.

Exercise capacity. Peak values obtained during exercise testing are shown in Table 3. Comparison with predicted values for absolute $\dot{V}O_2$, heart rate, and the respiratory exchange ratio demonstrated maximal effort during exercise testing. Work rate, exercise time, and heart rate were not significantly different between groups at peak exercise. Surprisingly, cardiopulmonary capacity was not decreased in the obese women despite their decreased lung volumes. Also, ratings of perceived exertion and breathlessness were also similar between groups at peak exercise. However, American Heart Association norms for maximal $\dot{V}O_2$ (ml·kg⁻¹·min⁻¹) demonstrated average cardiovascular fitness for the lean subjects and low cardiovascular

fitness for the obese subjects, which was significantly less than in the lean women ($P < 0.001$). Values obtained at VTh are shown in Table 4. Relative $\dot{V}O_2$ (ml·kg⁻¹·min⁻¹) was significantly lower in the obese group at VTh, although other variables were similar between the obese and lean women.

Ventilation and ventilatory response to exercise. Ventilation during exercise is shown in Fig. 2. \dot{V}_E was not significantly different between groups at rest or peak exercise; however, \dot{V}_E was significantly greater ($P < 0.05$) in the obese women at VTh. This was due to an increase in *f* ($P < 0.05$). Because TLC was reduced in the obese women, correlation analysis was performed on breathing pattern and lung function. TLC (percent predicted) was significantly correlated with *f* ($r = -0.76$, $P < 0.001$) and *V_T* ($r = 0.64$, $P < 0.01$) during exercise at VTh and during maximal exercise ($r = 0.82$, $P < 0.001$; and $r = -0.60$, $P < 0.05$, respectively). This association suggests that *f* was highest and *V_T* was lowest in the obese women with lower lung volumes and demonstrates a lung volume-related constraint on breathing pattern.

To further examine the ventilatory response to exercise, the slope of \dot{V}_E vs. work rate was calculated below and above VTh. The ventilatory response was significantly greater ($P < 0.05$) below VTh in the obese subjects (0.34 ± 0.08 vs. 0.22 ± 0.09), whereas the ventilatory response to exercise above VTh was not significantly lower in the obese women (0.57 ± 0.13 vs. 0.66 ± 0.13). An elevated ventilatory response to exercise below VTh is usually accompanied by an increased metabolic demand during submaximal exercise (14). To test for an increased metabolic demand, $\dot{V}O_2$ was compared between groups at submaximal work rates of 20, 40, and 60 W. As suspected $\dot{V}O_2$ (l/min) was significantly greater ($P < 0.01$) in the obese group at these work rates. Also, the slope of $\dot{V}O_2$ vs. work rate was higher in the obese women (11.7 ± 2.1) compared with the lean women (9.2 ± 2.7 ml/W), although this difference failed to reach significance ($P = 0.0588$).

Table 2. *Pulmonary function*

	FVC, %pred	FEV ₁ , %pred	FEV ₁ /FVC, %	PEF, %pred	MVV, %pred	DL _{CO} , %pred	DL _{CO} /VA, %pred
Lean (<i>n</i> = 8)	116 ± 13	110 ± 8	82 ± 7	121 ± 14	107 ± 13	101 ± 12	99 ± 7
Obese (<i>n</i> = 9)	107 ± 13	101 ± 9	80 ± 4	115 ± 12	103 ± 8	79 ± 8	113 ± 14
<i>P</i> value	ns	0.05	ns	ns	ns	0.001	0.05

Values are means ± SD. FVC, forced vital capacity; FEV₁, forced expiratory volume in 1 s; PEF, peak expiratory flow; MVV, measured maximal voluntary ventilation; DL_{CO}, diffusing capacity of the lung; VA, alveolar volume; pred, predicted.

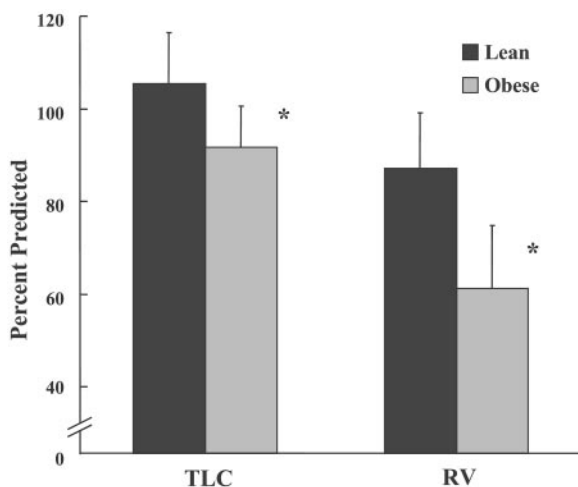


Fig. 1. Resting lung volumes plotted as a percentage of predicted values. Values are means \pm SD. TLC, total lung capacity; RV, residual volume. * $P < 0.05$.

Furthermore, there were significant correlations between the ventilatory slope below VTh and most of the indicators of body size (i.e., waist circumference, BMI, height-to-weight ratio, hip circumference, weight-to-height ratio, and percent body fat). The ventilatory response slope above VTh was not significantly correlated to any of the indicators of body size. This suggests that body size had little influence on the ventilatory response to exercise during heavy to maximal exercise.

Breathing mechanics. EELV at rest and during exercise is shown in Fig. 3. At rest, EELV was measured in the supine and seated positions. In the supine posture, all static mechanical forces of the rib cage and abdomen are expiratory in nature and push EELV to its lowest passive point (34). Thus EELV determined in this posture represents the static mechanical limit or lowest possible EELV attainable without expiratory muscle recruitment. EELV was significantly lower ($P < 0.01$) in the obese subjects in the supine position, at rest, and during exercise at VTh, but not during peak exercise. Both groups increased EELV during exercise above VTh, with the obese subjects actually hyperinflating above their resting EELV at peak exercise. Neither group of subjects decreased its EELV during exercise to the mechanical limit obtained in the supine position.

When both groups were combined, resting EELV was significantly correlated with body weight ($r = -0.55$, $P = 0.02$), percent body fat ($r = -0.54$, $P = 0.03$), waist

circumference ($r = -0.70$, $P = 0.002$), bust circumference ($r = -0.70$, $P = 0.003$), hip circumference ($r = -0.62$, $P = 0.007$), weight-to-height ratio ($r = -0.62$, $P = 0.008$), BMI ($r = -0.67$, $P = 0.03$), and waist-to-hip ratio ($r = -0.55$, $P = 0.02$). These results confirm the relationship between obesity and lower EELV. However, many of the usual indicators of body weight or body size are rather low predictors of the decrease in EELV, which suggests that indicators of fat distribution may be better predictors of the reduced EELV (e.g., percent body fat has lower correlation than waist circumference). All these correlations increased when correlated with EELV during exercise at VTh (e.g., percent body fat, $r = 0.67$, $P = 0.003$) but not during peak exercise when only body weight, height-to-weight ratio, and BMI were significantly correlated with EELV (range of r values: 0.49–0.51). Supine values were similar to those during exercise at VTh.

EILV as a percentage of TLC exhibited a similar pattern to EELV (Fig. 4). EILV was significantly lower ($P < 0.05$) in the obese subjects at rest and VTh but not during peak exercise. Both groups approached their TLC during maximal exercise, which is normal for maximal exercise. In the supine position, EILV was lower in the obese women but failed to reach significance. These lung volume data also demonstrate that V_T, in absolute liters, was similar between the two groups throughout exercise, which is not the usual expectation in obesity.

Expiratory airflow limitation was calculated at rest, VTh, and peak exercise. Expiratory airflow limitation was absent at rest in both groups. At VTh, three obese women experienced expiratory airflow limitation ($5 \pm 8\%$ V_T for $n = 9$; individually 12, 13, and 20% V_T), whereas flow limitation was not detected in any of the lean subjects ($P = 0.09$). These same three obese women plus one other obese woman had flow limitation ($12 \pm 17\%$ V_T for $n = 9$; 18, 20, 24, and 50% V_T individually) during peak exercise, whereas only one lean woman experienced flow limitation ($1 \pm 4\%$ V_T for $n = 8$ lean women; 10% V_T individually) during peak exercise ($P = 0.09$). When supine, two lean ($7 \pm 15\%$ V_T for $n = 8$; 16 and 43% V_T individually) and four obese women ($24 \pm 26\%$ V_T for $n = 9$; 41, 43, 45, 63% V_T individually) were flow limited ($P = 0.15$). Expiratory flow limitation in the supine position could be influenced by changes in maximal expiratory flow and critical pressure, although these changes are relatively small (12). However, in obese women, it is not uncommon to find expiratory flow limitation in the supine

Table 3. Peak exercise

	Time, min	Workload, W	\dot{V}_{O_2} , l/min	\dot{V}_{O_2} , %pred	\dot{V}_{O_2} , ml·kg ⁻¹ ·min ⁻¹	RER	\dot{V}_E /MVV, l/min	HR, %pred	RPE (0–20)	RPB (0–10)
Lean ($n=8$)	6.84 \pm 1.12	142 \pm 25	1.66 \pm 0.23	87 \pm 11	29 \pm 4	1.33 \pm 0.06	61 \pm 9	95 \pm 5	18 \pm 1	8 \pm 1
Obese ($n=9$)	6.30 \pm 1.05	138 \pm 18	1.84 \pm 0.31*	89 \pm 11*	19 \pm 3*	1.25 \pm 0.10	68 \pm 13	91 \pm 8	18 \pm 2	8 \pm 3
<i>P</i> value	ns	ns	ns	ns	0.001	ns	ns	ns	ns	ns

Values are means \pm SD; n = no. of subjects (indicated in parentheses or * $n = 8$). \dot{V}_{O_2} , oxygen uptake; RER, respiratory exchange ratio; \dot{V}_E , minute ventilation; MVV, measured maximal voluntary ventilation; HR, heart rate; RPE, ratings of perceived exertion; RPB, ratings of perceived breathlessness.

Table 4. *Submaximal exercise at ventilatory threshold*

	Time, min	Workload, W	\dot{V}_{O_2} , l/min	\dot{V}_{O_2} , %pred	\dot{V}_{O_2} , ml·kg ⁻¹ ·min ⁻¹	HR, %pred	RPE (0–20)	RPE (0–10)
Lean (<i>n</i> = 8)	3.62 ± 1.06	72 ± 21	1.09 ± 0.19	57 ± 9	19 ± 3	71 ± 8	11 ± 2	2 ± 1
Obese (<i>n</i> = 9)	3.11 ± 0.78	62 ± 16	1.18 ± 0.13*	58 ± 8*	12 ± 1*	66 ± 8	11 ± 2	2 ± 1
<i>P</i> value	ns	ns	ns	ns	0.001	ns	ns	ns

Values are means ± SD; *n* = no. of subjects (indicated in parentheses or **n* = 8).

position. Up to 61% of women with a BMI of 51 ± 9 have been found to have expiratory flow limitation in the supine position (16). This was with the expiratory flow limitation determined by the negative expiratory pressure device, which can detect expiratory flow limitation independent of changes in maximal expiratory flow and volume-time histories that might occur in the supine position. Three of the obese women with expiratory flow limitation at rest also had flow limitation during peak exercise.

Respiratory pressures, resistance, and mechanical work of breathing. The total mechanical work of breathing against the lung was not significantly different between the two groups at rest or during exercise, nor was total elastic work (Fig. 5). The difference in total resistive work against the lung only approached significance at VTh (*P* = 0.06) and peak exercise (*P* = 0.15), although it was higher in the obese women. The higher resistive work of breathing was primarily due to the expiratory airflow limitation in the obese women. Without the airflow-limited women (*n* = 5), resistive work of breathing was much lower in the obese women (65 ± 27 J/min at a \dot{V}_E of 73 ± 7 l/min and a work rate of 132 ± 11 W) and similar to that in the lean women.

Respiratory pressures were measured continuously throughout exercise and were analyzed at end inspiration, peak expiratory pressure, and end expiration of the breathing cycle. Pga during the different phases of breathing is shown in Fig. 6. Although not significant, end-expiratory Pga was higher in the obese women at rest. Pga was significantly increased during all phases of breathing at VTh and peak exercise. At end inspiration, the diaphragm has to displace the abdominal contents, which appears to also increase Pga. At peak expiration during exercise, recruitment of expiratory

muscles must displace abdominal contents and push the diaphragm upward during expiration. Thus Pga remained increased in the obese women. While supine, the obese women again had a higher Pga during all phases of the breathing cycle, although differences failed to reach significance.

A similar plot is shown in Fig. 7 for Ptp. Although not significant at rest, Ptp was lower in the obese women, as would be expected from an elevated Pga. Peak expiratory pressure was increased significantly in the obese women during exercise, which probably reflects a slightly higher resistance. Pulmonary resistance was higher due to the presence of tidal expiratory airflow limitation in four of the obese women (data not shown).

DISCUSSION

There were five major new findings of this study regarding mild to moderately obese women. 1) EELV is reduced while at rest and during exercise at least partially due to expiratory forces on the diaphragm as evidenced by an increase in Pga. 2) TLC is also significantly reduced in these low levels of obesity. 3) In contrast to that observed previously in mildly obese women during treadmill walking (5), EELV was decreased during moderate incremental cycling. 4) The reduced EELV induced in the supine body position, when all rib cage and abdominal forces are expiratory in nature, is lower than the EELV adopted during exercise. This suggests that factors other than mechanical limits on lung volume influence the EELV adopted during cycling. 5) On average, the work of breathing in mild to moderate obesity remains within normal limits

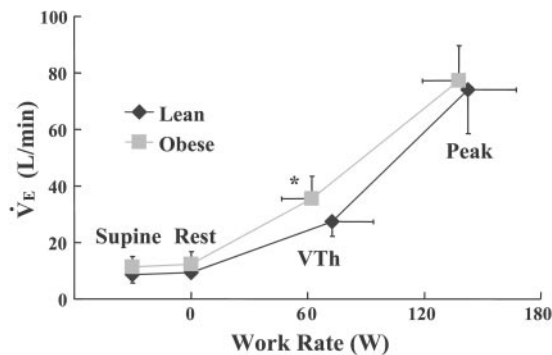


Fig. 2. Minute ventilation (\dot{V}_E) plotted against work rate for supine and seated (rest) postures, and during exercise at ventilatory threshold (VTh) and peak. Values are means ± SD. **P* < 0.05.

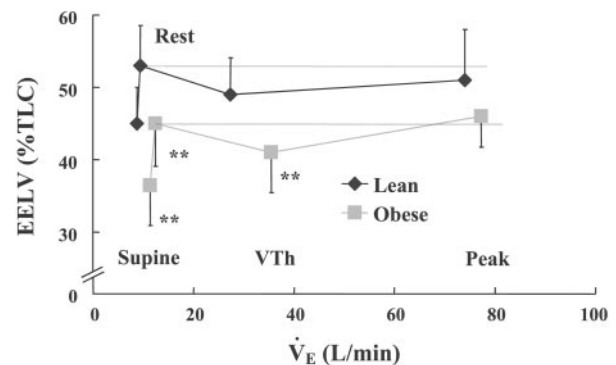


Fig. 3. End-expiratory lung volume (EELV) expressed as percent TLC (%TLC) plotted against \dot{V}_E for supine and rest postures and during exercise at VTh and peak. Thin dashed line indicates resting EELV level. Values are means ± SD. ***P* < 0.01.

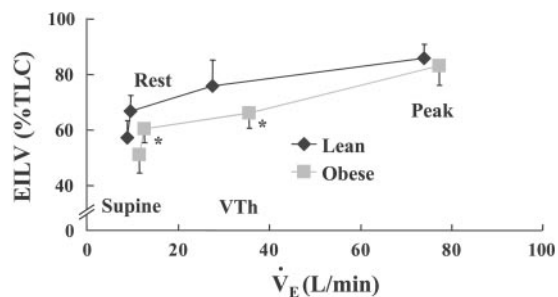


Fig. 4. End-inspiratory lung volume (EILV) expressed as %TLC plotted against \dot{V}_E for supine and rest postures, and during exercise at VTh and peak. Values are means \pm SD. * $P < 0.05$.

during exercise so that exercise tolerance and breathlessness are not altered. However, the resistive work of breathing tends to be higher in some obese women due to the presence of expiratory flow limitation during moderate to maximal exercise.

The lower EELV at rest, exercise, and supine is typical of that reported before (5, 9, 29). Although the

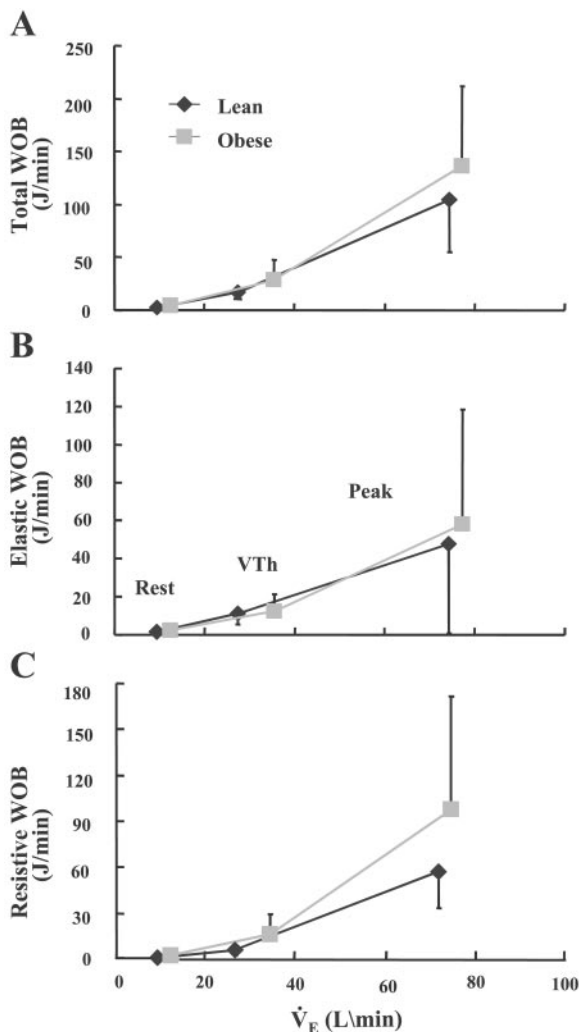


Fig. 5. Total (A), elastic (B), and resistive (C) work of breathing (WOB) plotted against \dot{V}_E at rest and during exercise at VTh and peak. Values are means \pm SD.

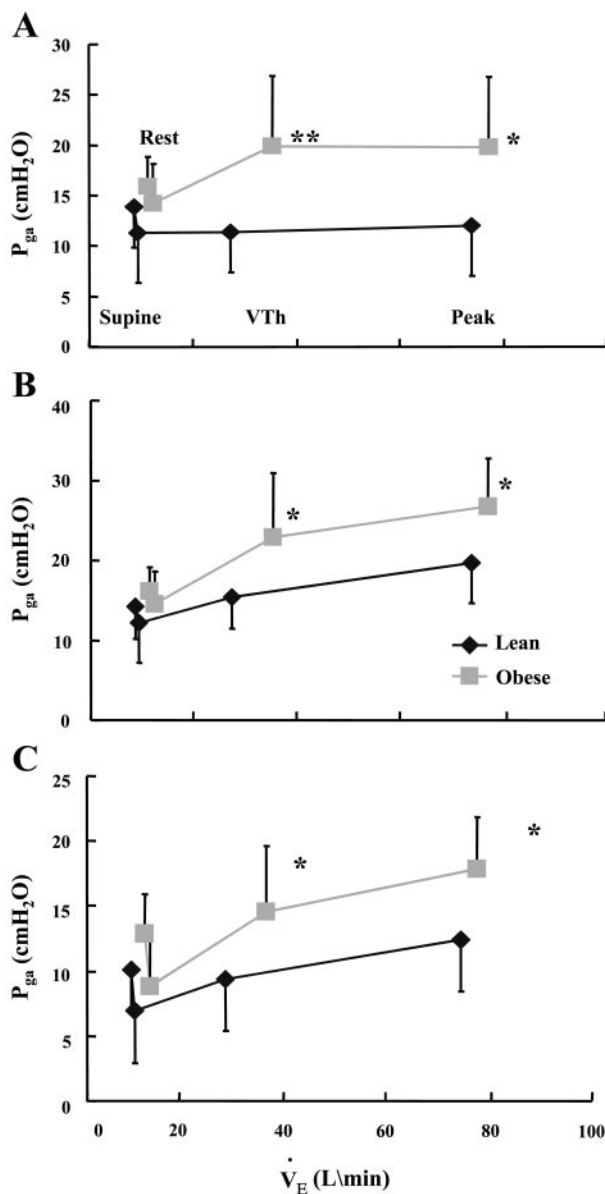


Fig. 6. Gastric pressure (P_{ga}) at end inspiration (A), peak expiratory pressure (B), and end expiration (C) plotted against \dot{V}_E for supine and rest postures and during exercise at VTh and peak. Values are means \pm SD. * $P < 0.05$; ** $P < 0.01$.

reason for the reduction in resting EELV has been speculated to be due to expiratory abdominal forces on the diaphragm, in contrast to inspiratory abdominal forces found in leaner individuals, this has not been shown before. In these obese subjects, P_{ga} was 2 cmH_2O higher at rest. This is probably due to an increased abdominal load, which displaces the diaphragm upward. An increase of $\sim 2 \text{ cmH}_2\text{O}$ as shown in Fig. 6, given the static compliance of the lung of $0.20 \text{ l/cmH}_2\text{O}$ in the obese women, could decrease EELV by $\sim 400 \text{ ml}$, which for these obese women would be a decrease of $\sim 8\%$ of TLC. This is approximately the difference in EELV between the obese and lean women at rest (Fig. 3). Thus the increased P_{ga} could account for almost all of the decrease in EELV. Supposedly, the

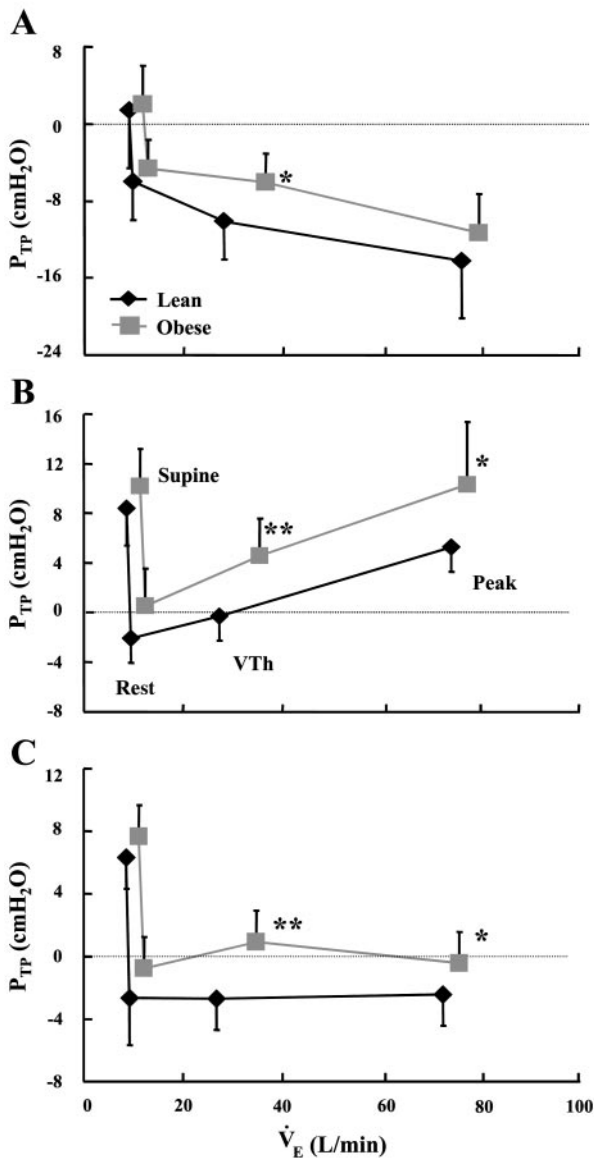


Fig. 7. Transpulmonary pressure (P_{TP}) at end inspiration (A), peak expiratory pressure (B), and end expiration (C) plotted against \dot{V}_E for supine and rest postures and during exercise at VTh and peak. Values are means \pm SD. * $P < 0.05$; ** $P < 0.01$.

increased pressure could be related to increased abdominal fat distribution. However, it is unknown whether this increased pressure is dependent on abdominal subcutaneous fat or visceral fat distributions (13, 22, 23), but the importance of fat distribution on lung function is important to determine in future studies.

The reduction in TLC is contrary to the findings of Ray et al. (29), who reported that TLC is usually not reduced until a weight-to-height ratio of 1.10 (kg/cm). Our obese subjects had a weight-to-height ratio of 0.58. Our findings suggest that TLC can be reduced even in mildly obese women. This finding has important implications for clinical reasons because it is often difficult to determine whether a reduction in lung volume is the effect of obesity or due to underlying pulmonary dys-

function. In these obese women, the average predicted TLC was 5.54 liters, and they had an average TLC of 5.07 liters. It is unclear how much rib cage loading contributes to the decreased TLC vs. abdominal impedance to maximal inspiration. There was no indication that inspiratory muscle weakness was a factor, but this was not a focus of this study. It also appears that the reduction in DL_{CO} may be related to this reduction in TLC. DL_{CO} is normal when corrected by alveolar volume.

In contrast to the expected EELV, the obese subjects were able to decrease their EELV during exercise, although they had to increase EELV during peak exercise, probably to avoid approaching expiratory flow limitation or to avoid extensive tidal expiratory airflow limitation. It is unclear why these obese women decreased their EELV during submaximal cycling whereas previous findings found EELV during treadmill walking not to be decreased (5). This could be related to differences between walking and cycling, which have recently been shown to evoke different ventilatory responses (19, 30). Otherwise, these obese women were very similar to those studied earlier. However, it is unknown whether the proportion of abdominal fat may have been different between these two studies. This could play an important role in the level of EELV as well as in observed differences in pulmonary function (13, 22, 23). The fact that there were many significant correlations between body size and EELV, both at rest and during exercise, suggests that many aspects of body composition may be important here. Given these findings, the difference between responses to cycling and walking could be important in the selection of an exercise mode for the treatment of obesity and deserves further study.

To subjectively investigate why these subjects did not decrease their EELV during exercise to its lowest possible static mechanical level, we placed their tidal flow-volume loop from peak exercise at their supine EELV. These loops were then placed within a subject's maximal flow-volume loop where tidal expiratory flow was compared with maximal expiratory flow (data not shown). This examination revealed that although a lower lung volume may be reached in the supine position, expiratory airflow limitation would be much greater during exercise (i.e., more subjects with flow limitation as well as more extensive flow limitation). Thus this would make breathing at this lower lung volume a poor strategy for breathing during exercise in these obese women. Furthermore, the work (i.e., expiratory pressure) necessary to further displace the diaphragm upward would be much higher, thus making this position less efficient regarding the work of breathing. These findings suggest that EELV is not determined by mechanical limits alone but is under the regulation of other nonmechanical factors as well (i.e., respiratory muscle effort or expiratory airway mechanics such as dynamic airway compression).

The mechanical work of breathing was not significantly increased, although it approached significance at peak exercise. However, if the ventilatory response to

exercise above $V_{T\text{H}}$ in the obese women was higher like it was below $V_{T\text{H}}$, \dot{V}_E would have been higher and the work of breathing would have been much greater in the obese women. Also, at the increased ventilatory rate, the current level of work of breathing would have been obtained at a much lower work rate. Thus we believe that two important components help to preserve a normal ventilatory capacity in these mildly obese women: 1) the ventilatory response to heavy exercise appears to be slightly attenuated in the obese women, and 2) the work of breathing is not out of proportion to that compared with normal subjects. This in essence maintains exercise respiratory drive in proportion to exercise intensity (15, 33). This is supported by the fact that ratings of shortness of breath were similar between the obese and lean women during all levels of exercise.

In conclusion, mild to moderate obesity in women does not appear to constrain EELV during cycling nor does it constrain exercise capacity, despite reduced resting lung volumes and alterations in respiratory mechanics during exercise. Also, the level of EELV adopted during exercise is higher than that imposed when supine, which suggests that other nonmechanical factors also contribute to the level of EELV adopted during exercise.

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