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PAPER

Mild-to-moderate obesity: implications for respiratory mechanics at rest and during exercise in young men

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OBJECTIVE: To investigate the effect of mild-to-moderate obesity on respiratory mechanics at rest and during exercise in obese men. We hypothesized that the simple mass loading of obesity would alter both end-expiratory lung volume (EELV) and respiratory pressures (gastric, P_{ga} and transpulmonary, P_{TP}) in resting body positions and during graded cycle ergometry to exhaustion.
SUBJECTS: A total of 10 obese (38±5% body fat; mean±s.d.) and nine lean (18±4%) men were studied.
METHODS: Body composition (by body circumferences and hydrostatic weighing) and pulmonary function were measured at rest. Breathing mechanics were measured at rest in the upright-seated position, supine, and during cycling exercise. Data were analyzed by independent *t*-test.
RESULTS: EELV was significantly lower in the obese men in the supine (30±4 *vs* 37±6% total lung capacity (TLC)) and seated (39±6 *vs* 47±5%TLC) positions and at ventilatory threshold (35±5 *vs* 45±7%TLC) ($P<0.01$). In contrast, at peak exercise, EELV was not different between groups. Respiratory pressures (P_{ga} and P_{TP}) were elevated ($P<0.05$) during one or more phases of the breathing cycle at rest and during exercise in obese men.
CONCLUSION: These data demonstrate that mild-to-moderate obesity in young men results in reduced lung volumes and alterations in respiratory mechanics when supine, seated at rest, and during exercise. During moderate exercise, obesity does not appear to limit changes in EELV; however, the regulation of EELV during heavy exercise appears to be affected.
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Introduction

Obesity is an epidemic problem in the US and is among the most important health challenges of the 21st century (Surgeon General's Call to Action, USPHS 2001). More than 60% of Americans are classified as overweight and more than 30% are frankly obese (body mass index (BMI) $\geq 30\,\mathrm{kg/m^2}$).[1] These obese individuals are at an increased risk of morbidity and mortality from conditions such as diabetes, heart disease, hypertension, sleep-disordered breathing, and some forms of cancer. Medical costs related to obesity are estimated at $117 billion annually, with approximately 300 000 US deaths/year attributed to obesity.[2] Despite these important health concerns, the limitations imposed on respiratory function by obesity are rarely recognized and have received relatively little attention. We believe these obesity-related alterations in breathing are critical to pulmonary function at rest and during exercise in a large portion of the US population. This is especially true for patients with mild-to-moderate obesity who have yet to develop obesity-related comorbidities, but depend on exercise as a means to combat obesity.

We have shown that there are significant changes in respiratory function in women with mild-to-moderate obesity.[3,4] One of the most critical obesity-related changes in lung function is a reduction of end-expiratory lung volume (EELV) at rest and during exercise.[3,4] The EELV adopted during exercise is an essential component of the ventilatory response to exercise, and alterations in EELV reflect limitations in respiratory mechanics during exercise.[5–7] The EELV adopted during exercise has implications for breathing pattern, tidal expiratory flow, respiratory muscle function, and the work of breathing, and could result in shortness of breath on exertion.[5] Recently, in a study designed to examine the determinants of EELV in

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young mild-to-moderately obese women, we reported that EELV was constrained during heavy exercise in obese women as compared with leaner women.[4]

In otherwise healthy mild-to-moderately obese men, the effect of obesity on lung volumes at rest and during exercise has not been determined. The effects of simple mass loading of the chest wall in men could be markedly different from that observed in obese women since women have a tendency to deposit fat in the hips and buttocks and men have the tendency to deposit fat more centrally.[8,9] Central fat distribution (ie, abdominal fat including both subcutaneous and visceral fat) could have the greatest impact on diaphragm position and lung volume regulation.[10,11] Furthermore, both gender and fat distribution have been reported as important factors in obesity-related alterations in ventilatory function.[12,13,10,14] Thus, the EELV adopted during exercise could be altered in obese men and different from that previously observed in obese women. Therefore, the study of lung function at rest and during exercise in obese men is an extremely important issue.

While knowledge of the lung volume response to exercise is important for understanding respiratory mechanics in obese men, it may also be clinically relevant. Exercise is commonly prescribed in the treatment of obesity;[15] however, compliance with these exercise programs has been relatively poor.[16] The adherence of obese individuals to exercise treatment programs is influenced by breathing discomfort or dyspnea, which many obese individuals report during exertion. As stated above, the EELV adopted during exercise could contribute to shortness of breath on exertion.[17,18] Therefore, further study of respiratory mechanics during exercise in obese patients is necessary.

The purpose of this study was to examine the effects of mild-to-moderate obesity on EELV and respiratory mechanics while supine, seated at rest, and during exercise in a group of mild-to-moderately obese men. We hypothesized that the simple mass loading of obesity in men would alter the normal decrease in EELV during exercise as compared with leaner men.

Methods

Subjects

Two groups of men were recruited through local advertisements. A total of 10 obese ($>30\%$ body fat) and nine lean ($<25\%$ body fat) men 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 familiarized to exercise on the cycle ergometer and instructed to avoid exercise, food, and caffeine for at least 2 h prior to 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 months. Subjects not meeting these guidelines were excluded as well as individuals with respiratory symptoms.

Study design

Pulmonary function tests, a resting electrocardiogram (ECG), and body composition measurements were performed as an initial screening. Afterwards, the participants returned to the laboratory on two separate occasions, once for maximal exercise testing and once for detailed pulmonary mechanics measurements. Before the maximal exercise test, each subject was studied at rest in the supine position and while seated upright.

Body composition

Standard measures of height and weight were made upon initial screening of subjects. BMI and weight-to-height ratio were calculated from these measures. Waist, hip, and chest circumferences were also 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, USA). Pulmonary function testing was performed according to the guidelines of the American Thoracic Society.[19] Predicted values for spirometry, lung volumes, and diffusing capacity were based on the norms of Knudson *et al*,[20,21] Goldman and Becklake,[22] and Burrows *et al*,[23] respectively. Maximal flow-volume loops were measured in a pressure-corrected volume-displacement body plethysmograph to minimize the gas compression artefact (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 ($\dot{V}CO_2$) were made with the use of a computerized custom gas exchange system as described previously.[24] System resistance was <2 cm H_2O per l/s through 6 l/s for expiration. Ventilatory threshold (VTh) was determined from the comparison of gas exchange indices[25] and the V-slope method.[26]

Breathing mechanics

Expiratory and inspiratory flow were measured at rest and continuously during exercise as described previously.[27]

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.[24,27] EELV was estimated from measurement of IC and reported as a percentage of total lung capacity (TLC). End-inspiratory lung volume was calculated (EILV = EELV + tidal volume, V_T) and expressed as a percentage of TLC. This assumes that TLC does not change significantly with body position[28] or exercise.[29–31]

Transpulmonary pressure (P_{TP}) was estimated as the differential pressure between oral and pleural pressure, which was measured with an esophageal balloon placed approximately 45 cm from the nare (Validyne pressure transducer, model MP45 ± 100 cmH$_2$O, Northridge, CA, USA). Validity of the balloon pressure was checked by having the subjects blow through a small orifice; if P_{TP} remained constant while oral pressure increased, P_{TP} was considered appropriate. This check was done each time the subject changed body position. Gastric pressure (P_{ga}) was measured with a balloon placed approximately 65 cm from the nare (Validyne, Northridge, CA, USA, model MP45 ± 340 cmH$_2$O). The pressures were displayed on a strip chart recorder (AstroMed, Model MT 95000, Warwick, RI, USA) and sampled real time on a computer. No pressures were reported when supine due to the potential for artefact in the measurement of P_{TP} and P_{ga} when in the supine body position.

Exercise protocol

Testing began with the subjects seated on the cycle ergometer while baseline measurements were made. The subjects breathed through a mouthpiece while wearing a noseclip. After 3 min of baseline measurements, the subjects performed graded cycle ergometry on an electronically braked cycle ergometer (model CPE 2000; MedGraphics, St Paul, MN, USA). Initial work rate was 30 W, and the work rate was increased each minute by 30 W until volitional exhaustion or pedal rate ≤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, USA). Ratings of perceived exertion (Borg 20 point scale) and breathlessness (Borg 10 point scale) were recorded at each work rate. Maximal flow-volume loops were determined at rest, while the subjects were seated on the cycle ergometer just before baseline measurements, and within 2 min following termination of exercise to determine if exercise had induced bronchodilitation or bronchoconstriction, which none of the subjects experienced.

Data analysis

V_T, breathing frequency (f_b), 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.[24,27] The mechanical work of breathing against the lung was estimated per breath from the area enclosed by the dynamic tidal pressure-volume loop (ie, using P_{TP}) with the addition of that portion of a triangle describing work that fell outside the tidal pressure-volume loop (ie, part of inspiratory elastic work) and then averaged.[32] 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 P_{TP} simultaneously exceeded the minimal critical pressure necessary to obtain maximal flow (P_{crit}).[24,27] Isovolume pressure flow curves were constructed from data collected in the body box and subsequently used to determine P_{crit}. These P_{crit} values were used solely to confirm expiratory flow limitation during exercise. Data were analyzed at rest, VTh, and during peak exercise.

The relationship between $\dot{V}_E$ and work rate was used to describe the overall ventilatory response to exercise. This method has been described previously.[24,27,33,4] Briefly, $\dot{V}_E$ was plotted against work rate and slopes were calculated for each individual. The ventilatory response to exercise was determined on all points between rest and VTh (below VTh), and between VTh and peak exercise (above VTh) by least-squares regression (l/min/W). The fit of these data by least-square regression was considered good based upon the average coefficient of determination (R^2), which below VTh was 0.92 ± 0.04 and 0.94 ± 0.09. Above VTh, the average was 0.96 ± 0.04 and 0.98 ± 0.03 for the lean and obese men, respectively.

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

Results

Subject characteristics and body composition

Subject characteristics are shown in Table 1. Weight, percent body fat, fat weight, lean body mass, BMI, waist and hip circumferences, height-to-weight ratio, and waist-to-hip ratio were all significantly greater ($P<0.001$) in the obese group. Based on NHLBI clinical guidelines and BMI, the obese subjects in this study were classified as either mild (Class I, $n=6$) or moderately obese (Class II, $n=4$). No differences were noted for age and height. Based on the waist-to-hip ratio, the obese men had more centrally located fat weight than the lean men. Two subjects in the lean group were former smokers with a 2 ± 1 pack year history, while six subjects in the obese group were former smokers with a 7 ± 7 pack year history.

Pulmonary function

Resting pulmonary function data are presented in Table 2. All subjects had normal spirometry based on predicted

Table 1 Subject characteristics

	Age (y)	*Ht (cm)*	*Wt (kg)*	*BF (%)*	*Waist (in)*	*Hip (in)*	*Waist/hip*	*Wt/Ht*	*BMI (kg/m^2)*	*Fatwt (kg)*	*LBM (kg)*
Lean ($n=9$)	30±7	176±6	73±6	18±4	33±2	38±1	0.86±0.04	0.41±0.03	23±3	13±4	60±5
Obese ($n=10$)	36±6	180±4	113±13	38±4	46±4	47±3	0.97±0.06	0.63±0.07	35±4	43±9	70±7
P-value	NS	NS	<0.0001	<0.0001	<0.0001	<0.0001	<0.0002	<0.0001	<0.0001	<0.0001	<0.0015

Values are means±s.d. Ht = height; wt = weight; BF = percent body fat; waist = circumference at waist; hip = circumference at hip; waist/hip = ratio of waist circumference to hip circumference; Wt/Ht = ratio of weight to height (kg/cm); BMI = body mass index; Fatwt = fat weight; LBM = lean body mass; NS = nonsignificant.

Table 2 Pulmonary function

	FVC (% pred)	*FEV$_1$ (% pred)*	*FEV$_1$/FVC (%)*	*PEF (% pred)*	*MVV (% pred)*	D$_{Lco}$ *(% pred)*	D$_{Lco}$*/VA (% pred)*	*TLC (%pred)*	*RV (%pred)*	*RV/TLC (%)*
Lean ($n=9$)	105±10	100±5	81±6	107±11	98±8	106±8	111±10	95±7	73±10	20±2
Obese ($n=10$)	102±10	96±10	79±7	108±15	94±9	94±10	120±13	94±8	70±19	21±6
P-value	NS	NS	NS	NS	NS	<0.05	NS	NS	NS	NS

Values are mean±s.d. FVC = forced vital capacity; FEV$_1$ = forced expiratory volume in 1s; PEF = peak expiratory flow; MVV = measured maximal voluntary ventilation; DLco = diffusing capacity of the lung; VA = alveolar volume; TLC = total lung capacity; RV = residual volume; and NS = nonsignificant.

Table 3 Peak exercise

	Exercise time (min)	*Maximum workload (W)*	$\dot{V}O_2$ *(l/min)*	$\dot{V}O_2$ *(%Pred)*	$\dot{V}O_2$ *(ml/kg/min)*	*RER*	$\dot{V}_E$*/MVV (%)*	*HR (bpm)*	*HR (%Pred)*	*RPE (0–20)*	*RPB (0–10)*	P$_{ET}$*CO$_2$ (torr)*
Lean ($n=9$)	7.02±0.72	217±25	2.61±0.37	89±17	36±4	1.36±0.14	67±13	177±13	91±6	18±1	8±2	37±3
Obese ($n=10$)	6.58±0.45	198±15	2.72±0.36	97±18	24±4	1.22±0.08	68±13	171±15	91±7	18±1	8±2	37±4
P-value	NS	NS	NS	NS	<0.001	<0.05	NS	NS	NS	NS	NS	NS

Values are mean±s.d.; W = watts; $\dot{V}O_2$ = oxygen uptake; RER = respiratory exchange ratio; $\dot{V}_E$ = minute ventilation; MVV = measured maximal voluntary ventilation; HR = heart rate; bpm = beats per minute; RPE = ratings of perceived exertion; RPB = ratings of perceived breathlessness; $P_{ET}CO_2$ = end-tidal CO$_2$, and NS = nonsignificant.

values. Relative to the lean subjects, lung volume subdivisions as a percent of predicted were not significantly different in the obese group (ie, total lung capacity, functional residual capacity, residual volume as % predicted). The normal predicted values for functional residual capacity are based on age, gender, height, and body weight and thus as a percent predicted were not different between groups.[22] In absolute terms, functional residual capacity (3.20±0.53 and 2.65±0.28 L) and expiratory reserve volume (1.78±0.38 and 1.24±0.51 L) were significantly lower ($P<0.05$) in the obese men despite them being slightly taller. These variables were also correlated ($P<0.05$) with weight, waist, and hip circumference, weight-to-height ratio, BMI, % body fat, and fat weight (correlation coefficients range from −0.50 to −0.67). Diffusing capacity of the lung for carbon monoxide ($D_{\rm Lco}$) as a percent of predicted was significantly ($P<0.05$) reduced in the obese subjects. When $D_{\rm Lco}$ was corrected for alveolar volume ($V_{\rm A}$), diffusing capacity was not different between groups ($D_{\rm Lco}/V_{\rm A}$ ratio as percent of predicted).

Exercise capacity

Values obtained during peak exercise are shown in Table 3. Comparison with predicted values for absolute $\dot{V}O_2$ and heart rate (HR), and the respiratory exchange ratio demonstrated maximal effort during exercise testing. Work rate, exercise time, and HR were not significantly different between groups at peak exercise. Ratings of perceived exertion and breathlessness were also similar between groups at peak exercise. American Heart Association norms for $\dot{V}O_2$ max (ml/kg/min) demonstrated average fitness for the lean subjects and low fitness for the obese subjects, which was significantly less than in the lean men ($P<0.001$). However, as a percent of predicted $\dot{V}O_2$ max based on ideal body weight, cardiorespiratory functional capacity was not different between groups.

Ventilation and ventilatory response to exercise

Ventilation during exercise is shown in Figure 1. $\dot{V}_{\rm E}$ was not significantly different between groups at rest or peak exercise but was significantly greater in the obese men at VTh. Neither $V_{\rm T}$ nor $f_{\rm b}$ was significantly greater in the obese men, although both were slightly larger at VTh. Additionally, the ventilatory response to exercise (ie, the slope of $\dot{V}_{\rm E}$ *vs* work rate) was not different below (0.26±0.10 *vs* 0.32±0.08 l/min/W) or above (0.64±0.12 *vs* 0.64±0.17) VTh between the lean and obese men, respectively. This finding suggests

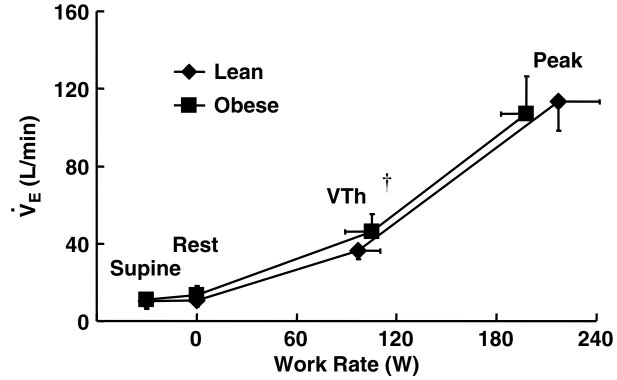

Figure 1 Minute ventilation ($\dot{V}_E$) plotted against work rate (watts) for supine and seated postures (Rest), and during exercise at ventilatory threshold (VTh) and peak exercise. Values are mean ± s.d. *$P<0.05$; †$P<0.001$.

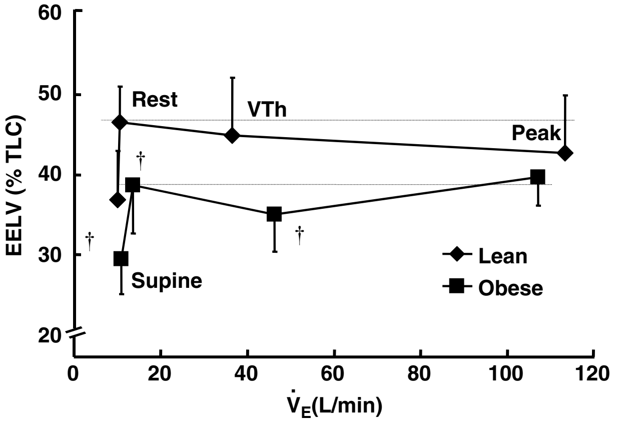

Figure 2 End-expiratory lung volume (EELV) expressed as percent total lung capacity (%TLC) plotted against ventilation ($\dot{V}_E$) for supine and seated postures (Rest), and during exercise at ventilatory threshold (VTh) and peak exercise. Small dashed line indicates resting EELV level. Values are mean and s.d. †$P<0.01$.

that body size had little influence on the overall ventilatory response to exercise.

Cardiorespiratory values obtained at VTh are shown in Table 4. Relative $\dot{V}O_2$ (ml/kg/min) and $P_{ET}CO_2$ were lower ($P<0.01$) and $\dot{V}_E$/MVV and relative HR were higher ($P<0.05$) in the obese group at VTh. Other variables were similar between the obese and lean men.

Breathing mechanics

EELV at rest and during exercise is shown in Figure 2. EELV was significantly lower ($P<0.01$) in the obese men in the supine position, upright-seated position, and during exercise at VTh, but not during peak exercise. During heavy-to-peak exercise, the obese subjects increased EELV back to resting levels, whereas the lean subjects continued to decrease EELV (Figure 2). The change in EELV from rest to VTh was not significant in either group. However, the difference from rest to maximal exercise was significant for the lean men ($P<0.05$) but not for the obese men. Neither group of subjects decreased their 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 ($r=-0.66$, $P=0.002$), percent body fat ($r=-0.56$, $P=0.01$), waist circumference ($r=-0.68$, $P=0.001$), hip circumference ($r=-0.59$, $P=0.008$), weight-to-height ratio ($r=-0.65$, $P=0.002$), BMI ($r=-0.64$, $P=0.004$), and waist-to-hip ratio ($r=-0.60$, $P=0.007$). All these relationships were weaker but remained correlated with EELV during exercise at VTh, whereas during peak exercise none of the indicators of body size and fat distribution were correlated with EELV. Relationships between the measures of body size listed above and supine EELV were similar to those observed during exercise at VTh.

EILV as a percentage of TLC exhibited a similar pattern to EELV (Figure 3). EILV was significantly lower ($P<0.05$) in the obese subjects in the supine position and at rest, but not at VTh and peak exercise. Both groups approached their TLC during maximal exercise (87 ± 9% TLC in lean and 83 ± 7%TLC in obese).

Expiratory airflow limitation was calculated at rest, VTh, and peak exercise. Expiratory airflow limitation was absent at rest in both groups. At VTh, two obese and lean men experienced expiratory airflow limitation (Obese 19, and 3% V_T; and Lean 19, and 3% V_T; $P=0.93$). During peak exercise, six obese men had flow limitation (13 ± 16% V_T for $n=10$; individually 13, 49, 13, 11, 23 and 22% V_T), whereas four lean men experienced flow limitation (8 ± 12% V_T for $n=9$; individually 25, 2, 29, 18% V_T; $P=0.45$). When supine, one lean subject exhibited flow limitation (8% V_T), while three of

Table 4 Submaximal exercise at ventilatory threshold

	Exercise time (min)	*Workrate (W)*	$\dot{V}O_2$ *(l/min)*	$\dot{V}O_2$ *(%Pred max)*	$\dot{V}O_2$ *(ml/kg/min)*	*HR (bpm)*	*HR (%Pred max)*	*RPE (0–20)*	*RPB (0–10)*	$\dot{V}_E$/*MVV (%)*	$P_{ET}CO_2$ *(torr)*
Lean ($n=9$)	3.22 ± 0.44	97 ± 13	1.44 ± 0.20	49 ± 10	20 ± 2	115 ± 10	59 ± 5	11 ± 2	2 ± 1	20 ± 4	47 ± 2
Obese ($n=10$)	3.50 ± 0.53	105 ± 16	1.60 ± 0.23[a]	57 ± 11[a]	15 ± 2[a]	127 ± 19	68 ± 9	12 ± 1	2 ± 2	27 ± 5[a]	44 ± 3
P-value	NS	NS	NS	NS	<0.001	NS	<0.05	NS	NS	<0.01	<0.05

Values are mean ± s.d.; W = watts; $\dot{V}O_2$ = oxygen uptake; HR = heart rate; bpm = beats per minute; RPE = ratings of perceived exertion; RPB = ratings of perceived breathlessness; $\dot{V}_E$ = minute ventilation; MVV = maximal voluntary ventilation; $P_{ET}CO_2$ = end-tidal CO_2, and NS = nonsignificant. [a]$n=9$.

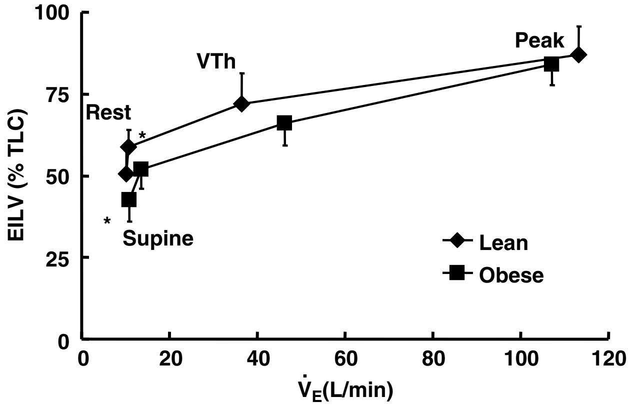

Figure 3 End-inspiratory lung volume (EILV) expressed as percent total lung capacity (%TLC) plotted against ventilation ($\dot{V}_E$) for supine and seated postures (Rest), and during exercise at ventilatory threshold and peak. Values are mean ± s.d. *$P<0.05$.

the obese subjects had flow limitation (55, 31, and 30% V_T). The occurrence of expiratory flow limitation was independent of smoking history.

Respiratory pressures and mechanical work of breathing

The total mechanical work of breathing against the lung was not significantly different between the two groups at rest and peak exercise, but was significantly higher ($P<0.05$) at VTh (data not shown). This was due to the greater $\dot{V}_E$ at VTh in the obese men.

Respiratory pressures (P_{ga} and P_{TP}) were measured continuously throughout exercise and were analyzed at end-inspiration, peak expiratory pressure, and end-expiration of the breathing cycle. P_{ga} during the different phases of breathing is shown in Figure 4. At end-inspiration, P_{ga} was increased at VTh and during peak exercise in obese men ($P<0.05$). Peak expiratory P_{ga} was increased when supine, at rest, and during exercise at VTh ($P<0.05$). End-expiratory P_{ga} was increased in the obese men at VTh ($P<0.05$).

A similar plot is shown in Figure 5 for P_{TP}. End-inspiratory pressure was only different at rest ($P<0.05$) in the obese men corresponding to the similar EILV and lung elastic work of breathing between groups. Peak expiratory P_{TP} was increased ($P<0.05$) only at VTh, where V_T/T_E was increased in the obese men. In the obese subjects, end-expiratory P_{TP} was increased at rest ($P<0.01$), VTh ($P<0.001$), and peak ($P<0.05$) exercise, which corresponds with a downward shift in EELV.

Discussion

The results of this study demonstrate that EELV is reduced in young mild-to-moderately obese men in the supine position, upright-seated position at rest, and during moderate-intensity cycling exercise. Furthermore, during low- and moderate-intensity cycling exercise, EELV decreased in both the lean and obese men; however, unlike the lean men in this study, EELV increased during heavy-to-peak exercise in the obese men. This qualitative difference in the EELV response during heavy exercise (ie, increase in EELV or lung hyperinflation) indicates the presence of respiratory mechanical limitations during exercise in obese men.[5] The lower EELV in the obese men is reflected by the higher P_{TP} and P_{ga} at rest and throughout exercise. To our knowledge, this is the first study to demonstrate the mechanical limitations imposed by mild-to-moderate obesity on pulmonary function at rest and breathing mechanics during

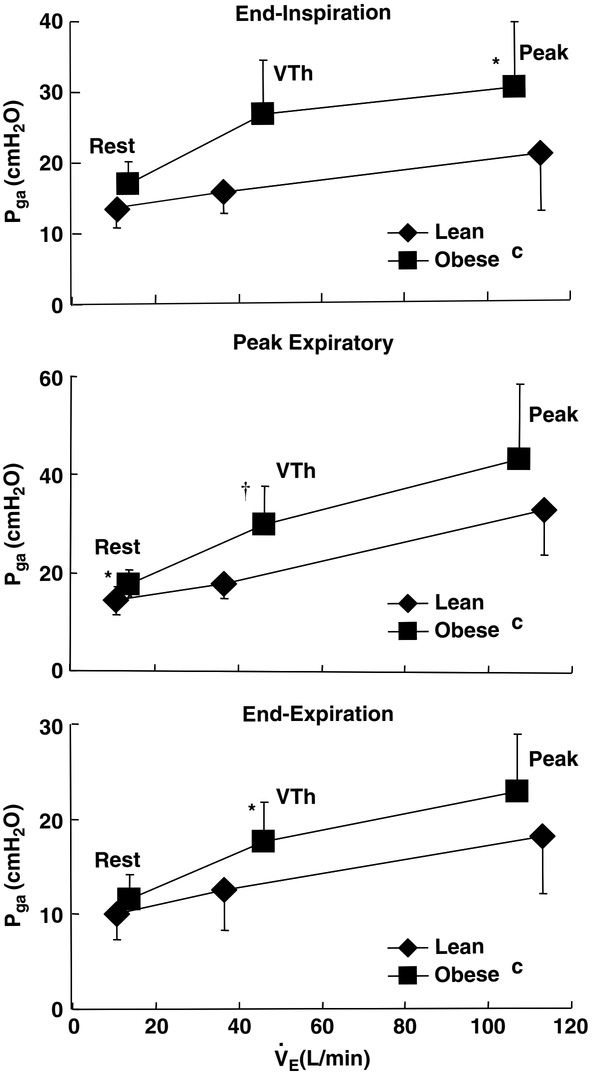

Figure 4 Gastric pressure (P_{ga}) at end-inspiration (top panel), peak expiratory pressure (middle panel), and end-expiration (bottom panel) plotted against ventilation ($\dot{V}_E$) for seated posture (Rest), and during exercise at ventilatory threshold (VTh) and peak. Values are mean ± s.d. *$P<0.05$, $^{\dagger}P<0.01$. $^{c}n=6$.

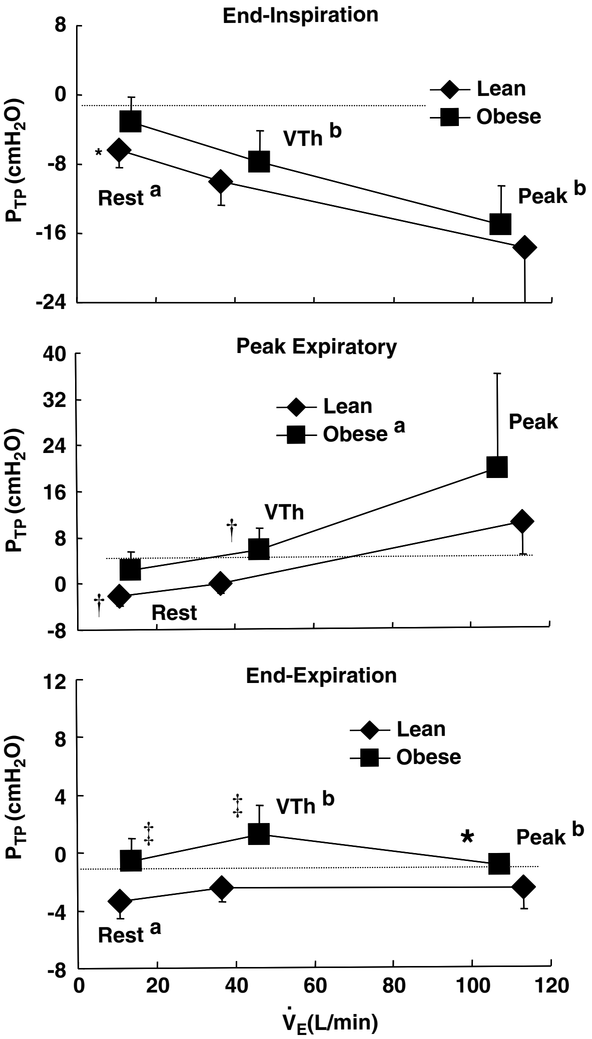

Figure 5 Transpulmonary pressure (P_{TP}) at end-inspiration (top panel), peak expiratory pressure (middle panel), and end-expiration (bottom panel) plotted against ventilation ($\dot{V}_E$) for seated posture (Rest), and during exercise at ventilatory threshold (VTh) and peak. Values are mean±s.d. $^*P<0.05$, $^\dagger P<0.01$, $^\ddagger P<0.001$. $^a n=8$; $^b n=7$.

exercise in young obese men who are otherwise healthy and symptom free during exertion. Furthermore, these mechanical limitations are not reflected in the standard measurements of $\dot{V}_E$ and exercise capacity.

The lower resting EELV has not been shown before for young mild-to-moderately obese men, but is consistent with other reports on resting pulmonary function in obesity.[3,34,35] The reduction in resting EELV has been attributed to expiratory abdominal forces on the diaphragm (ie, forces that push upward on the diaphragm), in contrast to inspiratory abdominal forces found in leaner individuals (ie, forces that pull downward on the diaphragm). In this study, P_{ga} was higher in the obese men at rest, suggesting an increased abdominal load, which displaces the diaphragm upward. The critical influence of central fat distribution on P_{ga} and diaphragm position remains unclear,[12,10,14] but the importance of central fat distribution on lung function will be essential to address more closely in future investigations. The significant correlations between several indicators of body size and EELV further suggests a relationship between obesity and a lower EELV. However, many of the usual indicators of body weight or body size were rather low predictors of the decrease in EELV, which suggests that more specific indicators of fat distribution could be better predictors of the reduced EELV (eg, percent body fat has lower correlation than waist or hip circumference). These findings suggest that many aspects of thoracic fat distribution (ie, rib cage, anterior abdominal subcutaneous, and visceral fat) could be important to the EELV adopted at rest.

The EELV adopted in the supine position by the obese men was lower than the EELV adopted in the upright-seated position and throughout exercise and is consistent with our previous observations in obese women.[4] A decline of EELV below resting values is typical of grossly obese subjects upon assumption of the supine posture,[36] but has not been shown specifically for mild-to-moderately obese men. In the supine posture, all static mechanical forces of the rib cage and abdomen are expiratory in nature (ie, pushes the diaphragm upward) and drives EELV to its lowest static position.[11] Thus, EELV adopted in this posture theoretically represents the static mechanical limit or lowest possible EELV attainable without expiratory muscle recruitment. This finding suggests that factors other than the static mechanical limits imposed by obesity influence the EELV adopted during cycling, which is also supported by the lack of relationship between indicators of obesity and EELV at peak exercise.

The regulation of EELV during exercise was similar during mild and moderate exercise in the lean and obese men, with both groups decreasing EELV from rest to VTh. This response is in agreement with earlier work from our laboratory on obese and nonobese women[4] and suggests that during mild-to-moderate cycling exercise, the ability to decrease EELV is not affected by mild-to-moderate obesity in men just as observed in women. However, during heavy to maximal exercise, the lean men continued to decrease EELV from VTh to peak exercise, while the obese men increased EELV. While the levels of expiratory flow limitation at peak exercise were modest and similar between groups, it is important to recognize that the lean subjects were able to decrease their EELV and tolerate these modest levels of expiratory flow limitation. On the other hand, the obese subjects had to increase EELV to avoid significant levels of expiratory flow limitation and were unable to completely escape being flow limited without further increasing EILV, which was already approaching TLC, and most likely producing large increases in the oxygen cost of breathing. The impact of the increase in EELV during heavy exercise on functional capacity or shortness of breath in obesity remains unknown.

P_{TP} at end-expiration was also significantly increased in the obese group at rest, VTh, and peak exercise, suggesting that these obese men encountered increased expiratory resistance as a consequence of breathing at lower lung volumes. However, the lack of correlation between indexes of body size and EELV at peak exercise would suggest that the effects of obesity are only one of several factors including expiratory flow limitation and expiratory resistive work that are involved in the regulation of EELV before and during peak exercise.

P_{ga} progressively increased throughout exercise and during all phases of breathing in both the lean and obese men. P_{ga} was slightly higher in obese men during all phases of breathing, reaching significance ($P<0.05$) at VTh for all phases of breathing and at end-inspiration during peak exercise. P_{ga} increased throughout exercise. The implications of the increased P_{ga} in regards to the work of breathing to displace the abdomen during exercise was not addressed in this study, but this unrecognized source of impedance must be considered a potentially important contributor to the overall oxygen cost of breathing in obesity. At end-inspiration, the diaphragm has to displace the abdominal contents downward, which appears to also contribute to the increased P_{ga}. With expiration during exercise, the recruitment of expiratory muscles increases the force on the abdominal contents and pushes the diaphragm upward. This also drives the decrease in EELV during exercise.

RPE and RPB ratings were not different between groups throughout the exercise protocol. This is in contrast to other reports,[37–39] which have reported frequent complaints of dyspnea in obese subjects during exercise. In this study, EELV was lower and $\dot{V}_E$, P_{TP}, and P_{ga} were all higher in the obese subjects during submaximal exercise; however, these changes did not result in a greater perception of effort or breathlessness in the obese men. This may indicate that in otherwise healthy mild-to moderately obese men, the level of mechanical ventilatory constraints may be too low to manifest a greater sensation of perceived exertion or breathlessness. Or, the obese men may have become accustomed to the mass load of obesity over time. Breathlessness on exertion observed in the clinical setting may be the result of a more exaggerated form of the same constraints resulting from a greater level of obesity or a difference in fat distribution. Additionally, it is possible that the obese subjects have adapted to a certain level of breathlessness over time and may rate perceived exertion and breathlessness differently than lean subjects. At peak exercise intensities, all subjects were working at high ventilatory and metabolic demands, thus the RPE and RPB ratings were near maximal in both groups and a difference between groups would not be expected.

In conclusion, mild-to-moderate obesity in men does not appear to limit the decrease in EELV during mild-to-moderate intensity cycling exercise. However, the reduced EELV at rest associated with obesity appears to influence the regulation of EELV during heavy-to-peak exercise. This occurs by placing the obese subject at a lung volume that predisposes him to high expiratory resistance and expiratory flow limitation, which necessitates an increase in EELV in order to increase $\dot{V}_E$ in concert with exercise intensity. Increased respiratory pressures during exercise could be a potentially important factor in the oxygen cost of breathing in obesity. The lower EELV, higher $\dot{V}_E$, and respiratory pressures during submaximal exercise in the obese men did not result in increased ratings of perceived exertion and breathlessness during exercise in these subjects. Thus, the importance of the alterations in respiratory pressures during exercise in obese men deserves further investigation, especially given the epidemic of obesity and the importance of exercise in weight maintenance and weight loss programs. Exercise interventions are important in lowering the risk of developing obesity-related comorbidities.

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