HHS Public Access

Author manuscript

Int J Obes (Lond). Author manuscript; available in PMC 2016 August 12.

Published in final edited form as:

Int J Obes (Lond). 2016 June ; 40(6): 998–1004. doi:10.1038/ijo.2016.21.

Effect of weight loss on operational lung volumes and oxygen cost of breathing in obese women

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Abstract

Background—The effect of moderate weight loss on operational lung volumes during exercise and the oxygen (O_2) cost of breathing are unknown in obese women but could have important implications regarding exercise endurance.

Methods—In twenty-nine obese women (33 ± 8yr, 97 ± 14kg, BMI: 36 ± 4, body fat: 45.6 ± 4.5%; means ± SD), body composition, fat distribution (by MRI), pulmonary function, operational lung volumes during exercise, and the O_2 cost of breathing during eucapnic voluntary hyperpnea ($\dot{V}O_2$ vs. $\dot{V}_E$ slope) were studied before and after a 12-week diet and resistance exercise weight loss program.

Results—Participants lost 7.5 ± 3.1kg or ≈8% of body weight ($p < 0.001$), but fat distribution remained unchanged. After weight loss, lung volume subdivisions at rest were increased ($p < 0.05$) and were moderately associated ($p < 0.05$) with changes in weight. End-expiratory lung volume (%Total Lung Capacity) increased at rest and during constant load exercise ($p < 0.05$). O_2 cost of breathing was reduced by 16% (2.52 ± 1.02 to 2.11 ± 0.72ml/L; P=0.003). As a result, O_2 uptake of the respiratory muscles ($\dot{V}O_{2Resp}$), estimated as the product of O_2 cost of breathing and exercise

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Conflict of Interest: None of the authors have any competing financial interests in relation to the work described. Dr. Bhammar has no conflicts of interest to disclose. Dr. Stickford has no conflicts of interest to disclose. Dr. Bernhardt has no conflicts of interest to disclose. Dr. Babb has no conflicts of interest to disclose.

Supplementary information is available at International Journal of Obesity's website.

$\dot{V}_E$ during cycling at 60W, was significantly reduced by 27 ± 31ml (P<0.001), accounting for 46% of the reduction in total body $\dot{V}O_2$ during cycling at 60W.

Conclusions—Moderate weight loss yields important improvements in respiratory function at rest and during submaximal exercise in otherwise healthy obese women. These changes in breathing load could have positive effects on the exercise endurance and adherence to physical activity.

Keywords

EXERCISE; BREATHING; OBESITY

INTRODUCTION

The effects of obesity on the respiratory system and lung function at rest are well-reported[1]. Hallmarks include a reduction in functional residual capacity (FRC), expiratory reserve volume (ERV), and, to a smaller extent, residual volume (RV)[1]. These changes are mainly the result of mechanical loading of the chest wall by excess fat weight. As a consequence, resting and exercise tidal breathing in obesity tends to occur at low operational lung volumes where expiratory flow reserves are low[2, 3]. In addition, excess fat weight on the chest wall increases the oxygen (O_2) cost of breathing[4, 5]. While it is known that modest weight loss can alter *resting* lung volume subdivisions, the effects of weight loss on operational lung volumes during exercise and the O_2 cost of breathing in obese women are unknown. Both these factors are important determinants of breathing mechanics during exertion.

With the onset of exercise, tidal volume (V_T) expansion occurs, in part, by reductions in end expiratory lung volume (EELV) in healthy nonobese individuals[6–10]. This change in lung volume at the end of expiration optimizes inspiratory muscle length for the subsequent inspiration. Rarely, though, does the decrease in EELV negatively affect the ventilatory capacity in healthy nonobese individuals because substantial ventilatory reserves remain. In contrast, since obese individuals already start out with considerably lower lung volume levels at rest, most V_T expansion must be accomplished by inspiratory muscles, thus contributing to the unique breathing pattern commonly observed in obese individuals (i.e. smaller V_T and higher breathing frequency (Bf))[11, 12].

The mechanical effects of obesity on respiratory function at rest and during exercise depend, in part, on the mass and anatomical distribution of chest wall fat. Chest wall fat includes adipose tissue on the rib cage, as well as subcutaneous abdominal and visceral fat deposits[3, 13]. The amount of chest wall fat appears highly correlated to FRC at rest[3]. We have previously shown that weight loss improves lung function at rest as well as breathing mechanics (i.e., operational lung volumes and respiratory pressures) during exercise in obese men[13]. To our knowledge, the effect of moderate weight loss on operational lung volumes during exercise has not been studied in obese women. The potential alteration of operational lung volume as a result of weight loss is clinically relevant because obese women have smaller respiratory reserves and potentially greater total chest wall fat when compared with obese men[3, 12, 14].

In addition to operational lung volumes, excess fat can affect chest wall compliance and resistance, static lung compliance[15], abdominal viscera compliance, and airway resistance, which could increase the mechanical work of breathing (Wb) and coinciding O_2 cost of breathing in obese adults[4, 15, 16], especially during exercise[17]. Reduced FRC is in part responsible for the increase in airway resistance observed in obese individuals[18], although other factors such as proinflammatory cytokines may also play a role[1, 19]. It is unclear whether moderate weight loss can reduce the O_2 cost of breathing or O_2 consumption by respiratory muscles $(\dot{V}O_{2Resp})$ during exercise. Reductions to the O_2 cost of breathing and $\dot{V}O_{2Resp}$ could be particularly beneficial to obese women because less O_2 would be required of the respiratory muscles during exercise, thus decreasing the overall Wb at a given exercise intensity. Moreover, respiratory muscles require substantial blood flow and oxygen supply in order to meet ventilatory requirements during exercise. Respiratory muscle work at maximal exercise can lead to diversion of 14 – 16% of the cardiac output to respiratory muscles[20, 21]. Increased respiratory muscle work also reduces blood flow to locomotor skeletal muscles through sympathetically mediated vasoconstriction, thus potentially limiting exercise tolerance[22].

The purpose of this study was two-fold: 1) to investigate the impact of weight loss and changes in chest wall fat distribution on operational lung volumes during exercise and 2) to investigate whether weight loss decreases the O_2 cost of breathing and $\dot{V}O_{2Resp}$. We hypothesized that weight loss would alter operational lung volumes during exercise via an increase in FRC at rest and EELV during submaximal exercise, with the greatest changes being observed in women with the largest changes in body fat, particularly chest wall fat. Additionally, we hypothesized that the O_2 cost of breathing and $\dot{V}O_{2Resp}$ in obese women would be reduced after a weight loss program, with the greatest changes being observed in women with the largest changes in body fat.

PARTICIPANTS AND METHODS

The UT Southwestern Institutional Review Board approved this study (approval number 122010-108) and all individuals provided written, informed consent. Participants were non-smokers and were excluded if they had a history of asthma, cardiovascular disease, musculoskeletal abnormalities, or if they had engaged in a vigorous physical activity routine (i.e. exercise more than two times a week with a specific training goal) in the last 6 months.

Participants reported to the laboratory on four occasions. Measures of body composition and pulmonary function were collected during the first visit. All cycle exercise and the O_2 cost of breathing tests were performed during the second and third visits, respectively. The fourth visit included MRI imaging for quantifying fat distribution. All visits were repeated following a 12-week diet and resistance training protocol.

These data were collected as part of a larger project examining the effects of weight loss on breathlessness in obese women[23]. Some of these data have been previously published in abstract form[24, 25].

Body Composition, Circumferences, and Fat Distribution

Measurements of weight, body circumferences (chest, waist, and hip), and percent body fat (underwater weighing) were determined using standard techniques. Multiple magnetic resonance imaging (MRI) scans through the chest and abdomen were used to assess fat distribution as described previously[3, 17]. For assessment of chest fat, three axial images were obtained through the upper rib cage (one through the sternal notch, one through the xiphoid process, and one halfway between the two). For abdominal fat, nine axial views were obtained through the abdomen and pelvis (one at the xiphoid process, T12 vertebra, each lumbar level, S1 vertebra, and symphysis pubis). MRI images (10-mm slice thickness) were analyzed with custom interactive software (Wafter, v1.3, Dallas, Texas). Subcutaneous fat area was equal to the difference between the outer boundaries of adipose tissue (i.e., skin) and the start of the viscera, and was divided into anterior subcutaneous abdominal fat (SQ) and posterior SQ compartments through the mid-coronal plane. Visceral fat was automatically detected by the software. The sum of the anterior SQ and visceral fat area yielded the total abdominal fat. Chest fat area was determined in a similar manner, except that fat mass was not divided into SQ and visceral fat, or anterior and posterior fat. Total chest wall fat was calculated as the sum of chest fat, anterior SQ, posterior SQ, and visceral fat.

Pulmonary Function

All participants had spirometry, lung volume, and diffusing capacity determinations in a body plethysmograph (Sensormedics Vmax Auto Box) according to ATS/ERS guidelines[26–28]. Maximal expiratory pressure (MEP) was determined at total lung capacity (TLC) and maximal inspiratory pressure (MIP) at FRC[29].

Operational Lung Volumes during Exercise

Operational lung volumes were determined while seated at rest on a cycle ergometer, during the last minute of constant load exercise at 60W, and at peak exercise. Total body O_2 uptake ($\dot{V}O_2$), expired carbon dioxide ($\dot{V}CO_2$), and minute ventilation ($\dot{V}_E$) were measured using the Douglas bag technique where expired gases were collected in polyurethane bags. Gas fractions were analyzed by mass spectrometer (Marquette Electronics, model 1100), and ventilatory volume was measured with a 200L Tissot spirometer. Resting measurements were made with the participants seated upright on a cycle ergometer (Lode Corival, The Netherlands) for 6 minutes. Subsequently, the participants cycled at a constant load of 60W for 6 minutes. Following a rest period (15–20 minutes), participants performed a peak exercise test. Participants pedaled at 60–65 rpm with an initial work rate of 20W, which was increased by 20W each minute until volitional exhaustion.

Flow was measured continuously using an inspiratory pneumotachograph (Hans Rudolph, Model 4813) and a heated expiratory pneumotachograph (Hans Rudolph, model 3850A) connected to a Hans Rudolph valve (Model 2700) via large-bore tubing. Flow signals were combined into a single bidirectional flow signal (Validyne Buffer Amplifier, model BA112) and digitally integrated to yield volume. Inspiratory capacity (IC) was measured at rest and

during the last 20s of exercise to determine placement of tidal flow-volume loops within the maximal flow-volume loop corrected for gas compression artifact[30].

EELV was estimated by subtracting IC from TLC and was expressed as a percentage of TLC. End-inspiratory lung volume (EILV) was calculated as the sum of EELV and V_T and was expressed as a percentage of TLC. Maximal flow-volume loops were determined at rest, and within 2 min following termination of the peak exercise test to determine if exercise had induced bronchodilation or bronchoconstriction, which none of the participants experienced. Expiratory flow limitation (EFL) was defined as the percentage of V_T where tidal expiratory flow impinged on maximal expiratory flow[30].

O_2 Cost of Breathing

The O_2 cost of breathing was determined from measurements of $\dot{V}O_2$ and $\dot{V}_E$ at rest and during 5 – 6 minute eucapnic voluntary hyperpnea (EVH) at 40 and 60 L/min. Bf, V_T, and $\dot{V}_E$ were held constant during EVH trials before and after weight loss, allowing for within-participant comparisons. The order in which the levels of voluntary hyperpnea were conducted was randomized, except for rest, which was always measured first. Participants breathed from a 1,000 L inspiratory reservoir bag containing 4% or 5% CO_2 (21% O_2 and balance nitrogen) to maintain eucapnia. Bf at each target ventilation (i.e., 40 and 60L/min) was set with a metronome at 30 and 35 bpm, respectively. Expired breath by breath $\dot{V}_E$ was monitored in real-time at the mouth with a turbine flow device and a custom computerized gas-exchange system, which was calibrated prior to each test. $\dot{V}_E$ from the breath by breath system was called out to give the participant volume feedback every 3–4 breaths to ensure attainment of the target $\dot{V}_E$ (i.e. 40 or 60L/min). $\dot{V}O_2$ and $\dot{V}_E$ were analyzed using the Douglas bag technique and averaged over 4 minutes for each target $\dot{V}_E$. Inspiratory capacity (IC) was measured at rest and approximately 5 minutes into each target $\dot{V}_E$ to determine placement of tidal flow-volume loops within the maximal flow-volume loop corrected for gas compression artifact[30]. The $\dot{V}O_2$ vs. $\dot{V}_E$ relationship during EVH trials was linear for all participants before and after weight loss (Pre R^2: 0.98 ± 0.03, Post R^2: 0.96 ± 0.04). O_2 cost of breathing was calculated as the slope of the linear regression between $\dot{V}O_2$ (ml/min) vs. $\dot{V}_E$ (L/min) at rest and during the two voluntary hyperpnea levels of EVH.

O_2 Cost of Breathing during Exercise $(\dot{V}O_{2Resp})$

We anticipated that the $\dot{V}_E$ measured during the submaximal constant load exercise test at 60W would be similar to the 40 L/min target ventilation of the EVH protocol. Since the mechanical Wb measured during exercise and during an EVH protocol is similar for a matched $\dot{V}_E$ below approximately 60L/min even with differences in breathing pattern[31], there was reasonable justification to extend our measurement of the O_2 cost of breathing to estimate $\dot{V}O_{2Resp}$ during exercise at 60W. Thus, $\dot{V}O_{2Resp}$ was estimated during constant load exercise at 60W as the product of the O_2 cost of breathing and exercise $\dot{V}_E$ and was

expressed as ml/min and as a percentage of the total body $\dot{V}O_2$ measured during exercise at 60W.

12-week Weight Loss Intervention

Participants underwent a supervised 12-week diet and resistance exercise program. Each participant met with a registered dietician and received dietary counseling and an individualized diet plan. All participants performed resistance exercises (i.e., weight-lifting exercises), three days per week, under the guidance of a personal trainer, who also monitored weight and compliance with the diet. The resistance exercises were utilized to increase caloric expenditure and minimize loss of muscle mass during the program. Participants were encouraged to lose one to two pounds per week.

Data Analysis

Based on previous data[13], it was estimated that 24 participants would need to complete the weight loss program to detect a significant improvement in EELV at an effect size of 0.69 (two-tailed test, $\alpha = 0.05$, $\beta = 0.90$).

Data were checked for normality using the Shapiro-Wilk test. Differences in variables after weight loss were tested by Student's paired t – tests. Relationships among variables were investigated with Pearson correlation coefficients. A P value < 0.05 was considered significant. Data were expressed as means ± SD.

RESULTS

Body Composition, Circumferences, and Fat Distribution

Participants were 29 obese women. All body composition measurements and body circumferences were significantly reduced after weight loss (Table 1). Participants lost approximately 8% of total body weight as a result the intervention ($p<0.001$). Most of the weight loss was driven by loss in fat weight (~15% reduction in total fat mass), however, 1.33±1.76kg of lean body mass was also lost despite the resistive exercise used in the weight loss program ($p<0.001$). Participants lost significant amounts of fat from all body regions, thus fat distribution (i.e., fat as a % of total body fat) remained unchanged after weight loss (Table 1). Participants lost a total of 3.53±1.76kg of fat from the chest wall ($p<0.001$) and a sum of over 18 cm from their cumulative chest wall circumferences (i.e., sum of chest, waist, and hip circumferences; 323±24 to 304±25 cm; $p<0.0001$).

Pulmonary Function

Spirometry was normal and unchanged with weight loss (Table 2). In contrast, TLC, FRC, and ERV were significantly increased ($p<0.05$) while IC was decreased after weight loss (Table 2). The change in FRC was moderately associated ($p<0.05$) with changes in BMI, visceral fat, the sum of chest, waist, and hip circumferences, and weight (e.g., correlations ranged from $r=-0.51$ with change in weight to $r=-0.59$ with change in BMI).

There were significant ($p<0.05$) but small improvements in MIP and maximal voluntary ventilation (MVV) as a percent of predicted after weight loss (Table 2 and supplementary

Table 1). The increase in MIP is most likely explained by the increase in FRC, which puts the diaphragm at a more mechanically advantageous length. Single-breath carbon monoxide diffusing capacity of the lung (DLco) relative to alveolar volume (VA) as a percent predicted decreased with weight loss due to a slight increase in VA ($p<0.05$).

Operational Lung Volumes during Exercise

Seated on the cycle ergometer, total body $\dot{V}O_2$ and $\dot{V}CO_2$ were significantly reduced at rest following weight loss, while $\dot{V}_E$ remained similar (Table 3). Resting breathing pattern was not altered, but EELV and EILV were significantly ($p<0.05$) increased after weight loss (Figure 1), which was consistent with the changes noted in the FRC measured during pulmonary function testing. No participant had EFL before or after weight loss at rest.

During constant load exercise at 60W, total body $\dot{V}O_2$ and $\dot{V}CO_2$ were reduced after weight loss (Table 3). Additionally, $\dot{V}_E$ was lower (– 4.8 ± 5.5 L/min; $P < 0.05$), accomplished via a reduction in Bf. The increase in EELV at rest as a result of weight loss was sustained during constant load exercise at 60W ($p<0.05$). However, there was no increase in EILV, in part due to the reduction in V_T. Six participants had EFL (n=6, 21±6%V_T) before weight loss, but only two participants (n=2, 21±12%V_T) had EFL after weight loss.

At peak exercise, total body $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}_E$, and EELV were unchanged as a result of weight loss (Table 3). However, EILV increased, which was the result of an increase in V_T at peak exercise. Twenty participants had EFL (19±9%V_T) before weight loss and 11 participants had EFL (23±11%V_T) after weight loss at peak exercise. Only 8 of the 11 women had EFL both before and after weight loss. There was no meaningful relationship between the change in EELV and change in EFL at peak exercise. There were no correlations between the change in EELV during constant load exercise or during peak exercise and changes in body composition or pulmonary function, not even the change in FRC at rest.

O_2 Cost of Breathing

Total body $\dot{V}O_2$ was reduced significantly at rest and at each level of EVH after weight loss (Figure 2). There was also a significant reduction in the O_2 cost of breathing slope from 2.52 ± 1.02 ml/L to 2.11 ± 0.72 ml/L ($P<0.010$). Measurements during the EVH bouts of 40 and 60L/min are reported in Table 3. There were significant differences after weight loss in $P_{ET}CO_2$ at rest, 40L/min EVH, and 60L/min EVH; however, these differences were very small in magnitude (< 1mmHg). Similar to changes observed during cycling at 60W, EELV during 40L/min EVH was significantly higher after weight loss (Table 3). In addition, EILV was also significantly higher after weight loss (Table 3), with no change in V_T, which was controlled before and after weight loss. There were no differences after weight loss in $\dot{V}_E$, V_T, Bf, or inspiratory duty cycle.

The reduction in total body $\dot{V}O_2$ at 40L EVH following weight loss was moderately correlated with changes in sum of waist, hip, and chest circumferences ($r = 0.58$; $P = 0.001$), body mass ($r = 0.49$; $P = 0.007$), fat mass ($r = 0.46$; $P = 0.013$), and FRC ($r = −0.49$; $P =$

0.007). In contrast the reduction in total body $\dot{V}O_2$ at 60L EVH or O_2 cost slope was not associated with changes in weight or pulmonary function. Individual data for selected variables during EVH before and after weight loss are shown in supplementary figure 1.

O_2 Cost of Breathing during Exercise $(\dot{V}O_{2Resp})$

Before weight loss, $\dot{V}O_{2Resp}$ during cycling at 60W was 110 ± 50 ml/min, which accounted for 8.5% of total body $\dot{V}O_2$. Following the weight loss, $\dot{V}O_{2Resp}$ was significantly reduced by 27 ± 31 ml/min ($P < 0.001$; Figure 3). As a result, $\dot{V}O_{2Resp}$ represented 6.7% of total body $\dot{V}O_2$ after weight loss.

DISCUSSION

The major findings of this study are that moderate weight loss improves obesity-related alterations in operational lung volumes during cycle exercise, and reduces O_2 cost of breathing in otherwise healthy obese women. These findings suggest that moderate weight loss reduces the ventilatory load of breathing, which in turn reduces the overall metabolic demand at rest and during exercise for obese women. Of note, the improvements do not appear to be strongly associated with changes in fat distribution because weight loss occurs equally from all regions of the body.

Body Composition, Circumferences, and Fat Distribution

Overall, the participants in this study lost approximately 16% of their fat weight. The changes in body composition are in agreement with the findings of other studies of obese men[32–34] and women[35, 36], who participated in diet and/or exercise programs. Additionally, the participants lost a substantial amount of fat weight from the chest wall (~47% of total fat weight lost). Fat distribution was not altered by weight loss in the obese women of the current study just as in the obese men we studied earlier[13]. These findings strongly support the conventional wisdom that weight loss occurs equally over the entire body and not preferentially from any particular body region.

Pulmonary Function

As anticipated, lung volume subdivisions increased after weight loss, especially FRC, which is very sensitive to the effects of decreased fat on the chest wall[1]. The improvement in resting pulmonary function with weight loss was also consistent with the findings reported by other studies[33, 37, 38].

Since no one measure of fat distribution was related to the change in FRC, this suggests that it could be the cumulative effect of weight loss from the chest wall in obese women that improves lung volume subdivisions. This is similar to what we observed in obese men previously[13]. However, the women only lost 3.53±1.76kg of weight from the chest wall, suggesting that lung volume subdivisions can be improved with fairly minimal weight loss from the chest wall.

Operational Lung Volumes during Exercise

Moderate weight loss improved operational lung volumes during exercise, as demonstrated by the increase in EELV during constant load cycling. In non-obese women, EELV is usually greater than 50% of TLC and drops during exercise into the upper forties[2]. In obese women, resting EELV is typically around 40% of TLC[2, 3] and may either remain there[6] or reduce below resting levels even during submaximal exercise[2]. At peak exercise, EELV returns near levels observed at rest (i.e., increase above submaximal levels) in non-obese women but could go even higher in obese women (i.e. dynamic hyperinflation)[2, 12].

It was unclear if weight loss would change exercise EELV in obese women since weight on the chest wall and other unknown factors could affect EELV in obese adults[2, 39]. In other words, the increase in EELV at rest could have allowed for a greater change in EELV from rest to exercise. We observed that the increase in EELV at rest with weight loss does not significantly change the response of EELV to submaximal exercise (i.e. the decrease in EELV from rest to exercise is similar before and after weight loss) (Figure 1). Moreover, the significant increase in EILV at peak exercise was not due to the increase in EELV but to an increase in V_T. The increase in EILV at peak exercise was weakly associated with the reduction in $\dot{V}O_{2Resp}$ at 60W, although this relationship did not reach statistical significance ($r = -0.284$, $P = 0.135$). It is plausible that the increase in EILV and V_T could be due to a decreased fat load on the chest wall and decreased elastic Wb rather than an increase in respiratory drive since peak $\dot{V}_E$ was not changed after weight loss.

While the reduction in EELV from rest to exercise did not change in magnitude, the overall improvement in EELV during exercise after weight loss placed the tidal flow-volume loop higher in the maximal flow-volume loop (i.e., ERV sparing), thus minimizing the potential for developing EFL[40, 41]. Nevertheless, EFL was affected little by the increase in EELV in the current study since only 4 out of 29 participants experienced EFL before weight loss during constant load exercise.

O_2 Cost of Breathing

We are the first to show that moderate weight loss reduces the O_2 cost of breathing by approximately 16% in obese women. The O_2 cost of breathing before weight loss in this study (2.52 ± 1.02 ml/L) was similar to values reported in previous studies in obese men and women[17, 42, 43]. However, it is substantially higher than what is observed in normal weight men and women (~1.2 ml/L)[4, 44, 45]. Potential mechanisms for increased O_2 cost of breathing due to obesity include increased fat load on the chest wall[46, 47], decreased efficiency of respiratory muscles[4, 48], reduced chest wall compliance, reduced lung compliance[15], and increased airway resistance[46, 47].

By estimating the O_2 cost of breathing[47, 49, 50] certain factors that cannot be detected within a volume-pressure loop such as breathing inertia, chest wall distortion, gas compressibility, antagonistic activity of respiratory muscles, and work on the abdominal viscera can be captured. Thus, O_2 cost of breathing represents the *total* energy required by the respiratory muscles to move the lung and chest wall, overcoming some of the limitations with measuring the mechanical Wb[46, 47]. Nonetheless, there is a strong relationship between the

mechanical Wb and the O_2 cost of breathing[31, 50, 51]. Accordingly, measurement of one will provide a robust index of the other.

As opposed to a frank reduction in the mechanical Wb, it is possible that the energetic requirements of the respiratory muscles were reduced due to an improvement in mechanical and/or metabolic efficiency. Typical efficiency values are reportedly between 1 and 10%[31, 44, 50, 52], but may be as high as 25%[53]. Efficiency of the respiratory muscles depends on the specific task and on shortening velocity. While we did not directly measure efficiency, since parameters of the voluntary hyperpnea task (i.e., minute ventilation, breathing pattern, and respiratory duty cycle) were similar before and after weight loss, we believe it is unlikely that efficiency was altered in our participants.

O_2 Cost of Breathing during Exercise ($\dot{V}O_{2Resp}$)

Methodological concerns: A recent paper by Dominelli et al[50] concluded that detailed flow-volume-pressure feedback during EVH was essential towards obtaining valid estimates of exercise $\dot{V}O_{2Resp}$. However, the study was not designed to examine whether detailed flow-pressure-volume feedback during EVH versus the absence of this feedback would significantly change the $\dot{V}O_{2Resp}$ estimates. To our knowledge, there are no studies to date that provide support for the concept that the absence of detailed flow-pressure-volume feedback results in inaccurate estimates of $\dot{V}O_{2Resp}$. Therefore we believe that experiments performed by Coast et al[31], which provided only $\dot{V}_E$ feedback while controlling V_T, Bf and T_i/T_{tot}, are more applicable when deciding the methodological validity of this study for estimation of $\dot{V}O_{2Resp}$. Coast et al[31] showed that at $\dot{V}_E$ below 60L/min (i.e. Wb < 10 kgm/min), there was a strong, almost linear relationship between $\dot{V}_E$ and Wb for both exercise and hyperventilation trials. Moreover, there were no differences in slope or mean values of $\dot{V}_E/Wb$ between exercise and hyperventilation, suggesting that $\dot{V}O_{2Resp}$ can be predicted either from $\dot{V}_E$ or from Wb measurements. Even individual results for exercise and hyperventilation trials for participants in Coast et al show a strong relationship ($r^2 > 0.97$) between $\dot{V}_E$ and Wb, especially when $\dot{V}_E$ is < 60L/min. Similarly, Aaron et al[51] showed that Wb between exercise and hyperpnea trials is strongly correlated at an intensity of 70% $\dot{V}O_{2max}$.

$\dot{V}O_{2Resp}$ during constant load exercise for the obese women in this study (106 ± 49 mL/min) is substantially higher than that reported by Coast et al (~50 mL/min) in nonobese men exercising at a similar absolute $\dot{V}O_2$[31]. The elevated $\dot{V}O_{2Resp}$ in our participants is likely due to the effect of obesity on the Wb and the O_2 cost of breathing. Following weight loss, $\dot{V}O_{2Resp}$ in obese women was reduced by 26%, coming closer to what has been previously reported in nonobese adults[31]. The reduction in $\dot{V}O_{2Resp}$ accounted for 46% of the reduction in $\dot{V}O_2$ during cycling at 60W.

Of note, 38% of the reduction in $\dot{V}O_{2Resp}$ during constant load exercise at 60W could be explained by the reduction solely in $\dot{V}_E$ due to decreased whole body O_2 uptake. The mechanism of the remaining 62% reduction cannot be determined using our measurement protocol but likely is a result of the reduced mass load placed on the chest wall and improved chest wall compliance or lung compliance[15]. The change in $\dot{V}O_{2Resp}$ during cycling at 60W was moderately associated with change total body $\dot{V}O_2$ at peak exercise following weight loss ($r = -0.351$), although this relationship did not reach statistical significance (P = 0.062), possibly due to our small sample size. Future studies may consider pursuing this hypothesis since it provides a plausible mechanistic explanation for an improvement in aerobic fitness, a finding that has important physiological and clinical implications for individuals with obesity.

CONCLUSION

In conclusion, moderate weight loss (~15 lb) can improve operational lung volumes during exercise and reduce the O_2 cost of breathing in obese women. Moreover, after moderate weight loss, $\dot{V}O_{2Resp}$ constitutes a smaller fraction of whole body $\dot{V}O_2$ during submaximal exercise, suggesting that weight loss could play a significant role in increasing submaximal exercise tolerance through favorable improvements in operational lung volumes and the O_2 cost of breathing, even in the absence of endurance exercise training. These data have important clinical implications for obese women who may find it difficult to engage in exercise and are thus struggling to meet the recommended physical activity guidelines. Our results suggest that losing a moderate amount of weight could reduce the breathing load during exercise, which could have a positive effect on exercise tolerance and adherence to physical activity, although this requires further investigation.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

The authors wish to thank Todd Bassett, Raksa Moran, Dr. Santiago Lorenzo, Dr. Matthew Spencer, Joseph Genovese, Jessica Pineda, and Sarah Haller-Martineau for their assistance in various stages of this project, and to Susan Rodder and the staff members of the Texas Health Finley Ewing Cardiovascular & Fitness Center Dallas for their expertise in implementing the diet and exercise program.

Sources of Support: Funding was provided by NIH R01 HL096782-01A2, King Charitable Foundation Trust, Cain Foundation, and Texas Health Presbyterian Hospital Dallas. The sponsors had no input or contributions in the development of the research and manuscript.

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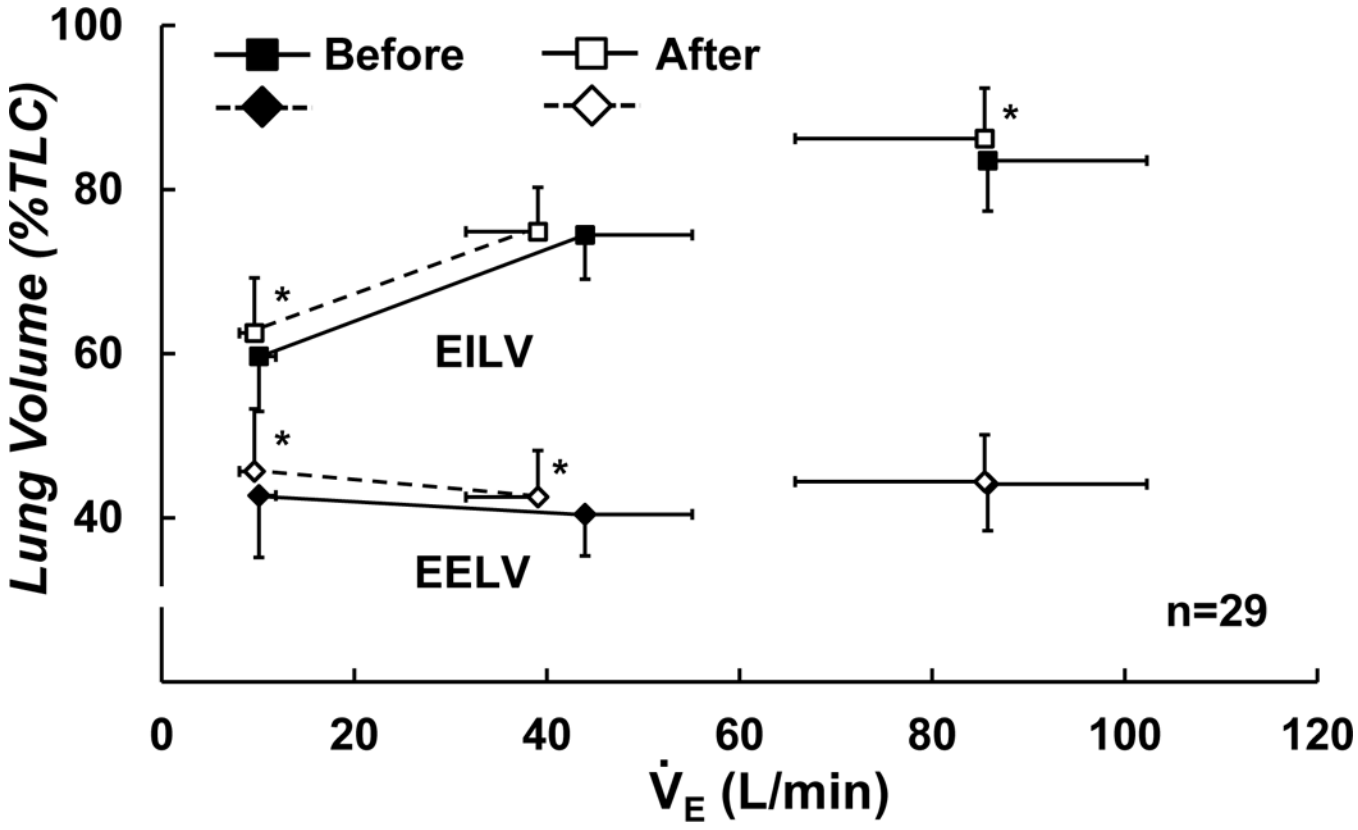
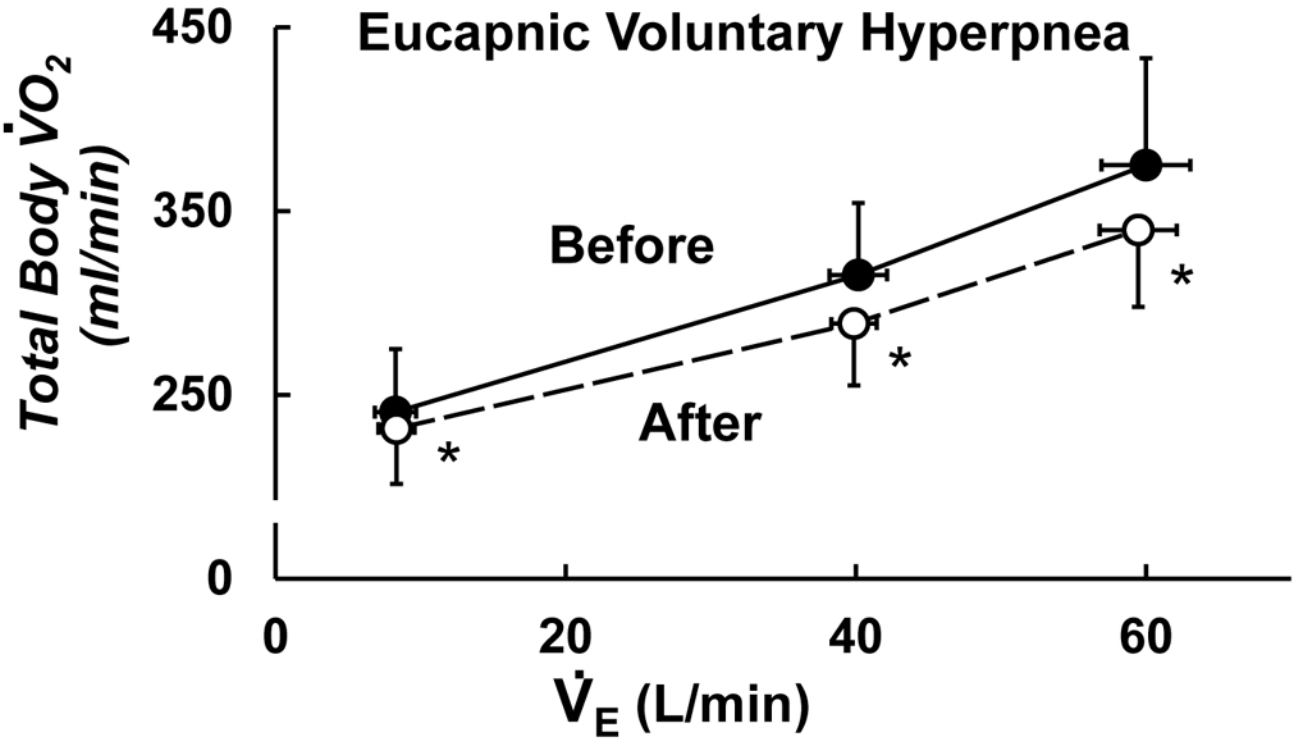


Figure 1.

End-expiratory lung volume (EELV) as a percent of total lung capacity (%TLC) and end-inspiratory lung volume (EILV, %TLC) at rest, during the 6 minute constant load exercise test at 60 Watts, and at peak exercise before and after a 12-week weight loss program. Values are means ± SD. *P < 0.05.



O_2 cost of breathing	Before		After	
Slope (ml/L)	2.52	1.02	2.11	0.72+
Y Intercept (ml/min)	218.4	36.8	210.9	32.9

Figure 2.

Total body oxygen uptake $(\dot{V}O_2)$ was reduced significantly at rest and each voluntary hyperpnea level. Oxygen (O_2) cost of breathing was also significantly reduced after weight loss. Values are means ± SD. *P < 0.05.

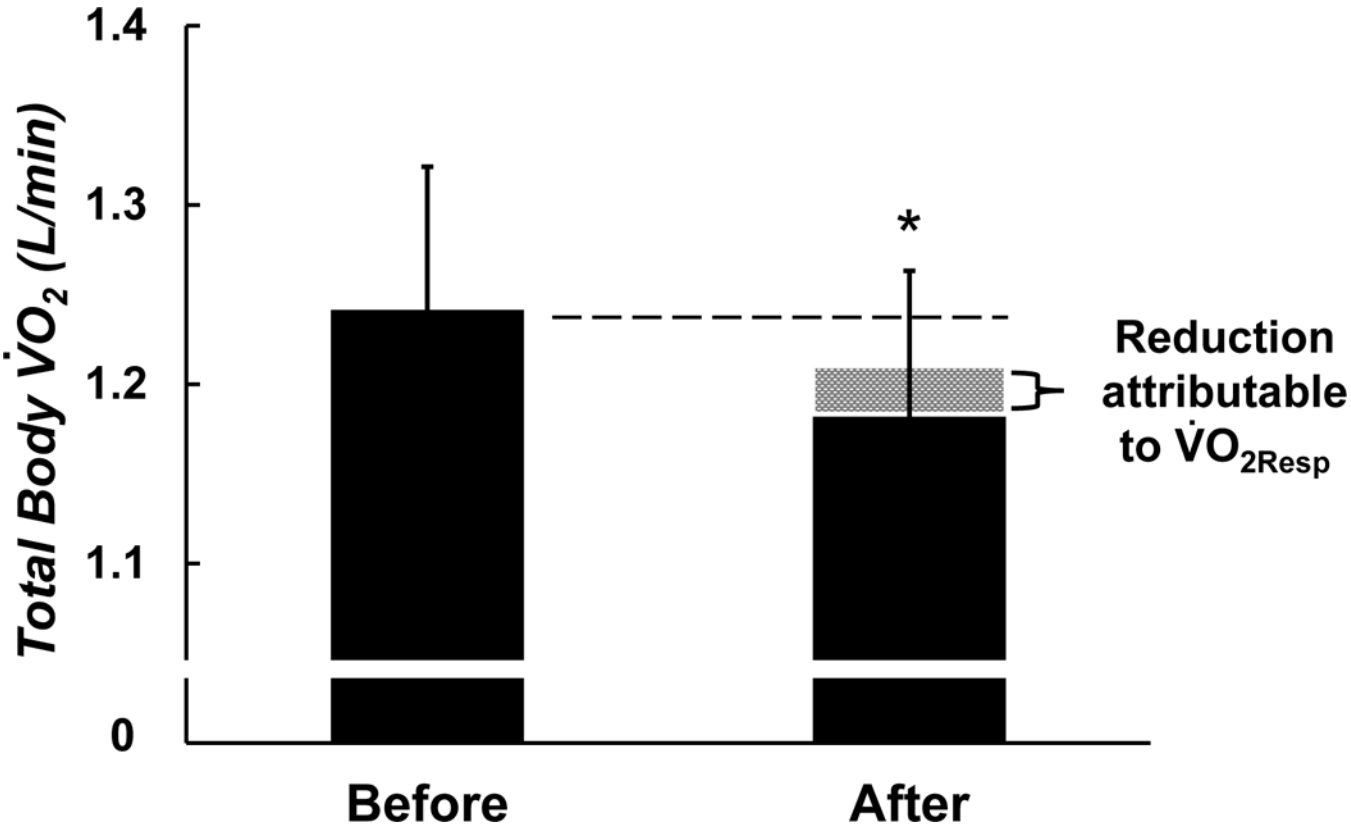


Figure 3.

Total body oxygen uptake ($\dot{V}O_2$) during the 6 minute constant load exercise test at 60 Watts before and after weight loss. The shaded region of the area between the dashed line and filled column indicates the reduction in $\dot{V}O_2$ that is attributable to the reduction in $\dot{V}O_2$ of the respiratory muscles ($\dot{V}O_{2Resp}$). Values are means ± SD. *P < 0.05.

Table 1

Participant characteristics, body composition, circumferences, and fat distribution via magnetic resonance imaging before and after a 12 week weight loss program in obese women (n=29).

	Before	After
Characteristics		
Age (yr)	33 ± 8	33 ± 8
Height (cm)	163.2 ± 7.1	163.1 ± 7.0
Weight (kg)	96.8 ± 14.0	89.2 ± 14.3^{+}
BMI (kg/m^2)	36 ± 4	33 ± 4^{+}
Body Composition		
Percent Body Fat (%)	46 ± 4^{a}	43 ± 6^{+}
Total Fat Mass (kg)	45 ± 10^{a}	38 ± 10^{+}
Lean Body Mass (kg)	52 ± 6^{a}	51 ± 6^{+}
Circumferences		
Chest (cm)	98 ± 6	93 ± 6^{+}
Waist (cm)	103 ± 13	96 ± 11^{+}
Hip (cm)	122 ± 9	115 ± 10^{+}
Neck (cm)	35 ± 2	34 ± 2^{+}
Fat Distribution		
Chest (kg)	5.5 ± 1.2^{a}	4.7 ± 1.2^{a*}
Anterior SQ (kg)	6.5 ± 1.8^{a}	5.6 ± 1.8*
Posterior SQ (kg)	8.4 ± 1.8^{a}	7.2 ± 1.9^{a*}
Visceral (kg)	4.0 ± 1.3^{a}	3.4 ± 1.2^{a*}
Chest Wall (kg)	24.4 ± 5.0^{b}	20.8 ± 5.3^{a*}
Peripheral (kg)	19.5 ± 5.1^{b}	17.0 ± 5.7^{a*}

Values are means ± SD. +P < 0.001 significant change with weight loss.

*P < 0.0001 significant change with weight loss. an=28 and bn=27. BMI, body mass index; SQ, subcutaneous abdominal fat; Chest Wall = Anterior SQ + Posterior SQ + Visceral; Peripheral = Total Fat – Anterior SQ – Visceral – Posterior SQ – Chest.

Table 2

Pulmonary function before and after weight loss in obese women (n=29).

	Before	After
Spirometry		
FVC (L)	3.71 ± 0.62	3.71 ± 0.64
FVC (%pred)	105 ± 14	104 ± 15
FEV_1 (L)	2.98 ± 0.50	2.97 ± 0.46
FEV_1 (%pred)	100 ± 14	99 ± 14
FEV_1 / FVC (%)	81 ± 6	80 ± 6
PEF (%pred)	106 ± 14	109 ± 18
MVV (%pred)	107 ± 16	114 ± 15^{+}
Lung Volumes		
TLC (L)	4.85 ± 0.74	4.91 ± 0.76*
TLC (%pred)	98 ± 14	99 ± 14
IC (%TLC)	58 ± 6	54 ± 7^{+}
FRC (%TLC)	42 ± 6	46 ± 7^{+}
ERV (%TLC)	17 ± 6	22 ± 8^{+}
RV (%TLC)	22 ± 5	22 ± 4
Respiratory Pressures		
MIP (%pred)	129 ± 31	135 ± 28*
MEP (%pred)	102 ± 25	105 ± 26
Diffusing capacity		
DLco (%pred)	81 ± 11	82 ± 11
DLco / VA (%pred)	119 ± 16	115 ± 17*
VA (L)	4.28 ± 0.68	4.38 ± 0.71*

Values are means ± SD.

*P < 0.05 significant change with weight loss and +P < 0.01 significant change with weight loss. FVC, forced vital capacity; %pred, percent predicted; FEV1, forced expiratory volume in one sec; PEF, peak expiratory flow; TLC, total lung capacity; IC, inspiratory capacity; FRC, functional residual capacity; ERV, expiratory lung volume; RV, residual volume; MIP, maximal inspiratory pressure; MEP, maximal expiratory pressure; MVV, maximal voluntary ventilation; DLco, diffusing capacity; and VA, alveolar volume.

Int J Obes (Lond). Author manuscript; available in PMC 2016 August 12.

Table 3

Gas exchange and breathing parameters measured at rest on the cycle ergometer, during the 6 minute constant load exercise test at 60 Watts (W), peak exercise, and during the eucapnic voluntary hyperpnea trials before and after weight loss.

	Exercise						Eucapnic Voluntary Hyperpnea					
	Rest on Cycle		Exercise at 60W		Peak Exercise		Rest on Chair		40 L/min EVH		60 L/min EVH	
	Before	After	Before	After	Before	After	*Before*	*After*	*Before*	*After*	*Before*	*After*
Power Output (Watts)	0	0	60	60	142 ± 24	148 ± 25 *	–	–	–	–	–	–
VO_2 (L/min)	0.28 ± 0.05	0.27 ± 0.04 *	1.24 ± 0.08	1.18 ± 0.08 *	1.84 ± 0.32	1.85 ± 0.33	0.24 ± 0.03	0.23 ± 0.03 *	0.32 ± 0.04	0.29 ± 0.03 *	0.38 ± 0.06	0.34 ± 0.04 *
VCO_2 (L/min)	0.24 ± 0.04	0.22 ± 0.04 *	1.27 ± 0.16	1.17 ± 0.13 *	2.33 ± 0.38	2.33 ± 0.41	0.19 ± 0.04	0.18 ± 0.03	0.25 ± 0.03	0.22 ± 0.04 *	0.24 ± 0.05	0.21 ± 0.04 *
V_E (L/min)	10.1 ± 1.7	9.6 ± 1.6	42.1 ± 10.0	37.3 ± 7.4 *	85.8 ± 16.5	85.5 ± 19.7	8.3 ± 1.4	8.3 ± 1.2	40.2 ± 2	39.9 ± 1.6	60 ± 3.1 ¶	59.5 ± 2.7¶
V_T (L)	0.82 ± 0.28	0.83 ± 0.31	1.66 ± 0.36	1.57 ± 0.28	1.9 ± 0.4	2.0 ± 0.4 *	0.69 ± 0.24	0.63 ± 0.14	1.35 ± 0.08 ¶	1.38 ± 0.10¶	1.71 ± 0.08	1.69 ± 0.12
Bf (bpm)	13 ± 4	13 ± 4	28 ± 10	26 ± 7 *	46 ± 11	44 ± 13	13 ± 4	14 ± 3	30 ± 1	30 ± 1 ¶	36 ± 0 ¶	37 ± 2 ¶
$P_{ET}CO_2$ (mmHg)	39 ± 3	39 ± 5	39 ± 4	40 ± 4	32 ± 4	32 ± 5	41 ± 3	40 ± 4	38 ± 2	38 ± 1 *¶	43 ± 1 ¶	43 ± 1 *¶
T_i/T_{tot}	0.39 ± 0.04	0.38 ± 0.05	0.47 ± 0.03	0.47 ± 0.03	0.48 ± 0.03	0.46 ± 0.05	0.43 ± 0.03	0.43 ± 0.03	0.48 ± 0.02	0.48 ± 0.03	0.49 ± 0.04	0.48 ± 0.03
V_T/T_i (L/s)	0.44 ± 0.09	0.42 ± 0.08	1.44 ± 0.28	1.28 ± 0.22	3.01 ± 0.58	3.10 ± 0.65	0.32 ± 0.06	0.32 ± 0.05	1.38 ± 0.10	1.38 ± 0.11	2.08 ± 0.17 ¶	2.06 ± 0.17 ¶
V_T/T_e (L/s)	0.28 ± 0.05	0.26 ± 0.05	1.31 ± 0.38	1.17 ± 0.27	2.74 ± 0.57	2.68 ± 0.73	0.24 ± 0.04	0.24 ± 0.04	1.29 ± 0.07	1.27 ± 0.07	1.91 ± 0.10¶	1.90 ± 0.11¶
EELV (%TLC)	42.7 ± 7.5	45.6 ± 7.6 *	40.4 ± 5.1	42.5 ± 5.7 *	44.1 ± 5.7	44.4 ± 5.7	41.6 ± 5.5	44.4 ± 7.7 *	42.6 ± 6.0 ¶	44.8 ± 5.0 *¶	42.2 ± 7.1	44.0 ± 5.5
EILV(%TLC)	59.7 ± 6.7	62.5 ± 7.8 *	74.5 ± 5.4	74.9 ± 5.9	83.5 ± 6.2	86.2 ± 5 *	56.7 ± 5.6	58.3 ± 8.0	70.9 ± 7.8¶	73.4 ± 6.1 *	77.4 ± 6.9	78.7 ± 7.1¶

Values are means ± SD.

* P < 0.05 significant change after weight loss.

¶ P<0.05 significant difference between EVH level and Exercise at 60W

VO2, Oxygen uptake; VCO2, carbon dioxide production; VE, minute ventilation; VT, tidal volume; Bf, breathing frequency; PETCO2, end tidal CO2; Ti/Ttot, ratio of inspiratory time to total time; Te, expiratory time.