**Simulated Obesity-related Restrictive Ventilatory Load Impairs Moderate Exercise Sustainability in Nonobese Men**

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This study aims in investigating the effects of simulating obesity-related restrictive ventilatory load (RVL) with external thoracic restriction (ETR) on sustaining moderate exercise and on associated cardio-respiratory responses, perceived breathlessness sensation and ventilatory muscle fatigue in nonobese men. Seven male adults with body fat <15% performed two identical exhaustive constant-load moderate cycle exercises with their resting total respiratory elastance either increased twofold by the ETR, with concomitant reduction in functional residual capacity in the RVL trial, or not manipulated in the control trial. In RVL condition, the ETR induced inspiratory muscle fatigue (as indicated by the reduction in post-exercise static maximum mouth pressure) and shortened the exercise time to exhaustion in all subjects (p < 0.05). At exhaustion, hyperventilation composed of rapid and shallow breathing pattern and decreased end-tidal CO₂ tension occurred, while arterial O₂ saturation and whole-body O₂ consumption were maintained in the control group. Subjective rating of perceived breathlessness sensation (RPB) was increased in the RVL condition (p < 0.05). The increase in RPB was correlated to the reduction in exercise sustainability (r = −0.83). Such findings implied that the perceived sensation of breathlessness in obesity mediated by obesity-related RVL during moderate exercise might contribute to their inferior exercise tolerance.

**Keywords:** breathlessness, inspiratory muscle fatigue, total respiratory elastance

**Introduction**

It was reported that the excessive adipose tissues in the thorax of subjects characterized as morbidly obese could cause more than twice the increase in total respiratory elastance (ERS) concomitant with reduction in functional residual capacity compared with normal values (Laghi & Tobin 2003; Weiner et al. 1998). The increase in ERS in obesity implies that inspiratory muscle force for performing resting inhaled chest wall movements associated with each breath might be greater in obese compared with normal subjects. This difference could be exaggerated during exercise with increasing ventilatory demand. Such mechanical constraints on the inhaled chest wall movement could be considered as the extra thoracic restrictive ventilatory load (RVL) specifically found in obesity. This has been simulated in a recent study by imposing mass loading on nonobese subjects’ chest for investigating the
related ventilatory responses to exercise (Wang & Cerny 2004).

Recent studies on healthy subjects have demonstrated that an increase in inspiratory muscle force output by augmenting airway flow-resistive load could impair sustainability in continuous and intermittent type exercises at moderate and supra-maximal intensities, respectively (Tong et al. 2003; 2001). In these studies, the impairment of exercise sustainability was partly attributed to the loaded breathing-induced noxious breathlessness sensation and the occurrence of ventilatory muscle fatigue. In obesity, mild exercise can induce a sensation of severe breathlessness. This is an obesity-related syndrome which is directly related to the excessive adipose tissues and the resultant impaired ERS (Lawrence & Kopelman 2004). Moreover, exercise tolerance in obese subjects is generally lower than that of their normal counterparts (Salvadori et al. 1991). It is not clear whether the severe breathlessness sensation induced by the obesity-related RVL during exercise in obesity accounts for the inferior exercise tolerance.

Tong et al. (2004; 2003) reported that exercise-induced breathlessness sensation is related to the magnitude of inspiratory muscle force output relative to the dynamic force output capacity of the muscles. It has also been noted that the force output capacity of the inspiratory muscles and associated exercise-induced breathlessness sensation could be improved significantly with the pressure threshold type inspiratory muscle training (Romer & McConnell 2003). Such specific training on inspiratory muscle function may potentially improve the ability of obesity to sustain moderate exercise provided that the obesity-related RVL-mediated noxious breathlessness sensation during the exercise, isolated from other obesity-related complications, is a factor in impairing their sustainability. However, the independent contribution of the obesity-related RVL to the inferior sustainability in moderate exercise in obesity was ambiguous. For clarifying the ambiguity in this study, chest compression-induced external thoracic restriction (ETR) was used to simulate the obesity-related RVL in obese individuals. The effects of the ETR on the ability of nonobese healthy individuals to sustain moderate exercise and on associated cardio-respiratory responses, perceived breathlessness sensation and ventilatory muscle fatigue were examined.

Methods

Subjects

Seven healthy young men who were asymptomatic for cardiovascular or respiratory disease volunteered for the study (Table 1). All were nonobese with body fat <15%. After being explained of the purposes and constraints of the study, and the potential benefits and risks involved in the exercise tests, subjects gave their written consent. This study was approved by the Committee of Hong Kong Baptist University on the Use of Human and Animal Subjects in Teaching and Research.

Procedures

Preliminary testing and familiarization. Forced spirometry and body fat percentage were assessed before experimental trials. The spirometry assessment followed the standard procedures recommended by Wanger (1992). Body fat measurement (in percent) was estimated by leg-to-leg bio-impedance analysis (Lu et al. 2003).

Prior to the experimental trials, the subject was required to perform three 4-min bouts of exercise on a cycle ergometer (Monark 818, Monark Exercise AB, Vansbro, Sweden) at constant power outputs of 60, 90, and 120 W, respectively, to determine the loading for the subsequent constant-load exercise tests. Oxygen uptake (VO2) was measured during the last minute of

| Table 1. Physical characteristics of the subjects (n = 7) |
|-------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|
| Age (years) | Height (cm) | Weight (kg) | FVC (L)    | FEV1 (L)   | FEV1/FVC (%)| BF (%)     | VO2peak (mL kg⁻¹ min⁻¹) |
| Mean ± SD  | 20.9        | 170.2       | 60.3       | 4.43       | 3.91        | 88.3       | 13.8       | 46.7        |
| 0.7        | 3.0         | 2.1         | 0.54       | 0.57       | 7.6         | 2.1        | 5.5         |

FVC = forced vital capacity. FEV1 = forced expiratory volume in 1s; BF = body fat (%); VO2peak = peak oxygen consumption.
each bout. After a linear relationship between the power output and the steady-state VO\textsubscript{2} had been determined, the subject performed a graded exercise test on the same cycle ergometer for VO\textsubscript{2peak} measurement. During the test, the subject started at 150 W for 2 min. Thereafter, 30 W was added every second minute until heart rate >90\% of the predicted maximum value (220 W) was reached. The addition of 30 W was then made for each minute until the point of volitional exhaustion. The highest VO\textsubscript{2} in 1 min was the VO\textsubscript{2peak}. Following the graded exercise test, a load that would entail ~70\% VO\textsubscript{2peak} of the subject was selected from the linear relationship of steady-state VO\textsubscript{2} versus power output for the exercise tests that followed.

Before the official testing, a familiarization trial identical to control trial (CON) exercise was undertaken to familiarize the subject with the sensation of exercising to exhaustion. It also enabled confirmation of the subject’s moderate workload.

**Experimental trials**

The subject was required to perform two constant-load moderate exercises on the same cycle ergometer with intensity at 70\% of the measured peak oxygen uptake (VO\textsubscript{2peak}) until exhaustion. During the exercise tests, the inhaled chest wall movement of the subjects was either loaded with imposition of the external thoracic restriction (ETR) or not manipulated (CON). The effects of the simulated obesity-related RVL on sustaining moderate exercise and on associated ventilatory muscle function, cardio-respiratory responses and perceived breathlessness sensation in subjects were revealed by the differences in the related parameters between the CON and RVL trials. Three of the seven subjects were assigned to perform the CON exercise prior to RVL, while the rest performed the tests in reverse order. The two tests were arranged on two separate days with at least 2 days apart in an air-conditioned laboratory with a temperature and relative humidity set at 22°C and 70\%, respectively. Prior to the exercise tests, the resting sub-divisions of lung volume and ERS in CON and RVL breathing conditions were assessed. The subject was refrained from eating at least 2 h before the sub-divisions of lung volume and ERS assessments and from participation in strenuous physical activity at least 1 day before exercise trials.

**Protocol**

Resting sub-divisions of lung volume and ERS measurements. During the measurements, the subject was seated on the cycle ergometer. The height of the seat and the handle of the cycle ergometer were exactly the same as those adjusted for exercise tests. The residual volume was measured by using the closed-circuit oxygen-dilution method (Wilmore 1969). The other sub-divisions of lung volume including inspiratory vital capacity, inspiratory reserve volume, inspiratory capacity, expiratory reserve volume were recorded from a slow vital capacity curve measured by using the SensorMedics Cardio-pulmonary Exercise Testing Instrument (Vmax 229 d, Yorba Linda, CA, USA). The slow vital capacity measurement was repeated until at least three acceptable curves were produced (American Thoracic Society 1995). The curve of the highest slow vital capacity was selected for the determination of the sub-divisions of lung volume. Functional residual capacity was the sum of residual volume and expiratory reserve volume. Total lung capacity was the sum of residual volume and inspiratory vital capacity. After a 15-min rest from the slow vital capacity measurements, ERS was measured with the weighted-spirometer method which is a non-invasive technique for measuring the relationship between the increase in functional residual capacity resulting from weighted spirometer and the corresponding increase in end-tidal mouth pressure during spontaneous breathing (Figure 1). This procedure has been described previously (Berger & Burki 1982). In the RVL trial, the ETR was imposed on the subject prior to all measurements.

Constant-load moderate exercise tests. After reporting to the laboratory, the subject was weighed. Following the standardized warm-up exercise consisting of 5-min cycling exercise at 60\% of the prescribed power output and 5-min stretching exercise, maximum pre-exercise static inspiratory (P\textsubscript{Imax}) and expiratory (P\textsubscript{Emax}) mouth pressures were measured. After the measurements, the subject began to cycle 60 revolutions per minute at the prescribed power output until volitional exhaustion. During the exercise, no indication of the time elapsed or verbal encouragement was given to the subject. Exercise was terminated when the subject failed to maintain the required pedal frequency twice.
within 30 s. The exercise sustainability was defined as the time to exhaustion. After the exercise, post-exercise $\Pi_{\text{max}}$ and $\Pi_{\text{Emax}}$ were measured within 5 min. The differences between pre- and post-exercise $\Pi_{\text{max}}$ and $\Pi_{\text{Emax}}$ were used to indicate the change in ventilatory muscle function. In the RVL trial, an identical exercise test was performed with the ETR imposed on the subject immediately after the pre-exercise $\Pi_{\text{max}}$ and $\Pi_{\text{Emax}}$ measurements. The ETR was released when the exercise was terminated and the measurements of the post-exercise $\Pi_{\text{max}}$ and $\Pi_{\text{Emax}}$ were followed.

**Measurements and instruments**

The ETR in RVL trial was imposed on the subject’s thorax with an inexpandable corset composed of two layers of inelastic denim, between which five semi-elastic pneumatic cuffs with different sizes and shapes were fixed. The total weight of the corset was 0.8 kg. The subject sat on the cycle ergometer. The front and back pieces of the deflated corset secured at the pre-exercise end-tidal lung volume stage and tightened by buckling up the lateral straps. An adjustable electric air pump was used to inflate the cuffs until the pressure inside the cuffs checked with a mercury-filled U-tube manometer at 160 mmHg. This was shown to be twice the resting ERS in normal individuals in pilot test. The cuff pressure of 160 mmHg was maintained with the electric air pump and monitored with the mercury manometer throughout RVL trials.

In the experimental trials, $\Pi_{\text{max}}$ and $\Pi_{\text{Emax}}$ were determined by measuring the maximum negative and positive mouth pressures developed against an occluded airway at residual volume and total lung capacity, respectively. The measurement was performed with the same equipment and procedure reported in our previous study (Tong et al. 2001). During exercise, subjects’ ratings of perceived intensities of breathlessness sensations (RPB) were assessed with the aid of a modified Borg category scale that was described in detail in our previous studies (Tong et al. 2004; 2003). Their heart rate and arterial $O_2$ saturation during the exercise were monitored using a heart rate monitor (Polar, Finland) and a Sat-Trak Pulse Oximeter with a finger sensor (SensorMedics, Yorba Linda, CA, USA), respectively. RPB, heart rate and arterial $O_2$ saturation were recorded at the last 5 s of every second minute. The last recorded data prior to exhaustion in CON and RVL and that recorded at the iso-time point of RVL exhaustion in CON (ISO) were used for the analysis. Moreover, subject’s ventilatory and metabolic responses including minute ventilation, tidal volume, breathing frequency, inspiratory and expiratory times, duty cycle, peak and mean inspiratory flow rates, $V_{O_2}$, and end-tidal $CO_2$ tension at exhaustion in CON and RVL and at ISO were recorded for analysis. The parameters were recorded with the same SensorMedics cardio-pulmonary measuring instrument at 1-min interval. Calibrations of the $O_2$ and $CO_2$ analyzers, and the flow-volume integration system of the instrument have been reported previously (Tong et al. 2001). The data of the parameters at exhaustion were the values recorded in the last completed minute of the exercise.

**Fig. 1** The changes in the volume-pressure tracings in a typical subject during the measurement of the resting total respiratory elastance by applying a weight of 3 kg on the spirometer bell in (a) CON and (b) RVL trials.
Statistical analysis

Paired *t* tests were used to determine the difference in sub-divisions of lung volume, ERS and exercise sustainability between CON and RVL. One-way analysis of variance (ANOVA) with repeated measures was performed to examine the differences among RPBs and cardio-respiratory variables at exhaustion in CON and RVL, and at ISO. Two-way ANOVA with repeated measures was used to examine the differences in PImax and PEmax between pre- and post-exercise, and across CON and RVL conditions. *Post hoc* analyses using Newman–Keuls were performed when main effects of the above ANOVA were significant. Relationships between variables were determined from simple regression. All tests for statistical significance were standardized at an alpha level of *p* > 0.05, and all results were expressed as mean ± SD.

Results

In RVL, the ETR increased the CON ERS at rest almost twofold in all the subjects (Figure 2). The increase in the CON ERS was concomitant with the reductions in the resting expiratory reserve volume and residual volume (*p* > 0.05), while there was no significant change in the inspiratory reserve volume and inspiratory capacity (*p* < 0.05). As a result, the CON total lung capacity was reduced (Table 2).

In this study, the cycling work rate of subjects during the exercise test in both CON and RVL trials ranged from 96 to 144 W. The average value of the corresponding steady-state O2 consumptions measured at different periods of the exercise in CON, interpreted as percent VO2peak, ranged from 65.8% to 73.4% VO2peak in the subjects and the group mean was 70.0% ± 3.2% VO2peak. In CON, the group mean of exercise sustainability was 48.2 ± 15.5 min. The CON exercise sustainability was reduced dramatically with the ETR in all subjects to the group mean of 18.1 ± 4.2 min (*p* < 0.05) in RVL (Figure 3).

For the PImax, the main effect for the CON and RVL conditions was not significant, while the main effects

<table>
<thead>
<tr>
<th></th>
<th>CON</th>
<th>RVL</th>
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<tbody>
<tr>
<td>ERS (cmH2O L⁻¹)</td>
<td>9.31 ± 0.94</td>
<td>20.52 ± 1.12*</td>
</tr>
<tr>
<td>IVC (L)</td>
<td>4.38 ± 0.44</td>
<td>3.74 ± 0.56*</td>
</tr>
<tr>
<td>ERV (L)</td>
<td>1.85 ± 0.22</td>
<td>0.84 ± 0.26*</td>
</tr>
<tr>
<td>IRV (L)</td>
<td>1.79 ± 0.32</td>
<td>1.96 ± 0.33</td>
</tr>
<tr>
<td>IC (L)</td>
<td>2.53 ± 0.31</td>
<td>2.62 ± 0.33</td>
</tr>
<tr>
<td>RV (L)</td>
<td>1.47 ± 0.22</td>
<td>1.04 ± 0.21*</td>
</tr>
<tr>
<td>FRC (L)</td>
<td>3.33 ± 0.36</td>
<td>1.89 ± 0.42*</td>
</tr>
<tr>
<td>TLC (L)</td>
<td>5.85 ± 0.62</td>
<td>4.51 ± 0.71*</td>
</tr>
</tbody>
</table>

Values are mean ± SD. IVC = inspiratory vital capacity; ERV = expiratory reserve volume; IRV = inspiratory reserve volume; IC = inspiratory capacity; RV = residual volume; FRC = functional residual capacity; TLC = total lung capacity.

*Significantly different from the CON (*p* < 0.05).

Table 2. Total respiratory elastance (ERS) and sub-divisions of lung volume at rest in CON and RVL trials (*n* = 7)

Fig. 2 Individual subject’s total respiratory elastance (ERS) at rest under CON and RVL conditions are shown.

Fig. 3 Individual subject’s exercise sustainability (Ex) under CON and RVL conditions are shown.
that pre-exercise PImax was reduced after the exercise percentage of the ISO value, the percent increase in RPB at RVL exhaustion in subjects was expressed in values were higher than the ISO value (Table 4). While the sessions showed that the RPB at CON exhaustion values were significant (Table 3). For the PEmax, the main effects for the pre- and post-exercise, the CON and RVL conditions and their interaction were not significant (p > 0.05).

For the RPB, one-way ANOVA revealed that the main effect for the values at CON exhaustion, at RVL exhaustion and at the iso-time point of RVL exhaustion in CON (ISO) was significant (p < 0.05). Post hoc analyses showed that the RPB at CON exhaustion values was similar to that at RVL exhaustion, while both values were higher than the ISO value (Table 4). While the RPB at RVL exhaustion in subjects was expressed in percentage of the ISO value, the percent increase in the RPB at RVL exhaustion from that at ISO was negatively inter-individual correlated to the percent reduction in the CON exercise sustainability in RVL trial (r = -0.83, p < 0.05).

The cardio-respiratory variables during the exercise in CON and RVL at exhaustion and at ISO are shown in Table 4. A comparison of these variables at exhaustion in RVL with those in ISO, found that the values of minute ventilation, breathing frequency, and peak and mean inspiratory flow rates were higher, while the values of tidal volume, inspiratory and expiratory times, and end-tidal CO2 tension were lower in RVL trial, but not in CON (Table 3). For the PEmax, the main effects for the pre- and post-exercise, the CON and RVL conditions and their interaction were not significant (p > 0.05).

Values are mean ± SD. PImax = static maximum inspiratory mouth pressure; PEmax = static maximum expiratory mouth pressure.

*Significantly different from the Pre-ex value in RVL trial (p < 0.05).

Table 4. The RPB and cardio-respiratory variables in CON and RVL trials at exhaustion and in CON trial at the iso-time point of RVL exhaustion (ISO) (n = 7)

<table>
<thead>
<tr>
<th>Variable</th>
<th>CON</th>
<th>ISO</th>
<th>RVL</th>
</tr>
</thead>
<tbody>
<tr>
<td>RPB</td>
<td>8.4 ± 2.1*</td>
<td>6.3 ± 1.9</td>
<td>9.5 ± 1.1*</td>
</tr>
<tr>
<td>V̇e (L·min⁻¹)</td>
<td>61.1 ± 6.8</td>
<td>59.1 ± 7.8</td>
<td>70.4 ± 10.8*†</td>
</tr>
<tr>
<td>Vt (L)</td>
<td>1.6 ± 0.4</td>
<td>1.7 ± 0.2</td>
<td>1.3 ± 0.2*†</td>
</tr>
<tr>
<td>fᵢ (breaths min⁻¹)</td>
<td>39.8 ± 8.9</td>
<td>35.4 ± 7.0</td>
<td>56.3 ± 10.3*†</td>
</tr>
<tr>
<td>Tᵢ (s)</td>
<td>0.76 ± 0.20</td>
<td>0.79 ± 0.16</td>
<td>0.54 ± 0.12*†</td>
</tr>
<tr>
<td>Te (s)</td>
<td>0.87 ± 0.17</td>
<td>0.93 ± 0.21</td>
<td>0.58 ± 0.15*†</td>
</tr>
<tr>
<td>Tᵢ/Ttot (%)</td>
<td>46.5 ± 0.1</td>
<td>46.2 ± 0.1</td>
<td>48.3 ± 0.1</td>
</tr>
<tr>
<td>PIF (L·s⁻¹)</td>
<td>3.57 ± 0.45</td>
<td>3.29 ± 0.47</td>
<td>4.16 ± 0.43*†</td>
</tr>
<tr>
<td>V₃/Tₖ (L·s⁻¹)</td>
<td>2.11 ± 0.30</td>
<td>2.15 ± 0.18</td>
<td>2.41 ± 0.16*†</td>
</tr>
<tr>
<td>VO₂ (mL·kg⁻¹·min⁻¹)</td>
<td>35.2 ± 6.0*</td>
<td>31.8 ± 4.6</td>
<td>31.8 ± 3.6*</td>
</tr>
<tr>
<td>PetCO₂ (mmHg)</td>
<td>38.5 ± 3.9</td>
<td>39.7 ± 3.7</td>
<td>36.4 ± 3.7*†</td>
</tr>
<tr>
<td>HR (beats min⁻¹)</td>
<td>176.6 ± 10.6*</td>
<td>166.0 ± 10.0</td>
<td>172.1 ± 13.2</td>
</tr>
<tr>
<td>SaO₂ (%)</td>
<td>96.4 ± 1.1</td>
<td>95.6 ± 1.8</td>
<td>95.4 ± 0.6</td>
</tr>
</tbody>
</table>

Values are mean ± SD. RPB is rating of perceived intensities of breathlessness sensations; V̇e = minute ventilation; fᵢ = breathing frequency; Vt = tidal volume; Tᵢ = inspiratory time; Te = expiratory time; Tᵢ/Ttot is duty cycle; PIF = peak inspiratory flow rate; V₃/Tₖ = mean inspiratory flow; VO₂ = oxygen uptake; PetCO₂ = end-tidal CO₂ tension; HR = heart rate; SaO₂ = arterial O₂ saturation.

*Significantly different from the ISO (p < 0.05).
†Significantly different from the CON (p < 0.05).

for the pre- and post-exercise and for their interaction were significant (p < 0.05). Post hoc analyses revealed that pre-exercise PImax was reduced after the exercise in RVL, but not in CON (Table 3). For the PEmax, the main effects for the pre- and post-exercise, the CON and RVL conditions and their interaction were not significant (p > 0.05).
to those at ISO except that CON heart rate and \( \text{VO}_2 \) at exhaustion were higher \((p < 0.05)\).

**Discussion**

Recently, the effect of obesity-related RVL independent of other obesity-related complications on individuals’ maximal exercise performance was studied (Wang & Cerny 2004). They found that the maximal work rate of the nonobese subjects during the graded cycle exercise was reduced when mass loading was imposed on the chest to simulate obesity-related RVL. However, the related changes in the ERS and breathlessness sensation intensity were not reported. In the present study, with the ETR imposed on seven nonobese subjects in RVL trial, the resting ERS values were increased almost twofold from the CON, while the expiratory reserve volume and residual volume were reduced. The twofold increase in ERS concomitant with expiratory reserve volume and residual volume reductions were the typical ventilatory mechanical impairments in morbid obesity reported earlier (Laghi & Tobin 2003). Further, when the subjects exercised at an intensity of 70% \( \text{VO}_{2\text{max}} \) in RVL, the ETR augmented the intensity of breathlessness sensation and impaired the global inspiratory muscle function and exercise sustainability in all subjects.

In RVL, the ETR reduced the CON exercise sustainability to the group mean of 40.7% \( \pm 16.7\% \) (ranged from 30.1% to 76.8%). The detrimental effect of the ETR on exercise sustainability was consistent with those reported in previous studies where similar RVL were imposed on subjects’ chests during various exercises either for simulating obesity-related chest loading (Wang & Cerny 2004) or for other purposes (Hussain et al. 1985; Ghesquiere et al. 1979). Further, when the subjects exercised at an intensity of 70% \( \text{VO}_{2\text{max}} \) in RVL, the ETR augmented the intensity of breathlessness sensation and impaired the global inspiratory muscle function and exercise sustainability in all subjects.

It was reported that the reduction in tidal volume with restrictive load was partly due to the mechanical restriction, while the increase in breathing frequency might be the result of extravagal-reflex response that was modulated by the cerebral cortex through the fusimotor-muscle spindle system (Nishino et al. 1992). In obesity, the similar breathing pattern of rapid breathing frequency and lessened tidal volume were generally observed during exercise and such breathing pattern has been considered to lead to inadequate alveolar ventilation and therefore low exercise tolerance (Sakamoto et al. 1993). However, this was not the case in RVL when subjects exercised with the ETR. It was found that CON end-tidal \( \text{CO}_2 \) tension was lowered, while arterial \( \text{O}_2 \) saturation was maintained during the exercise even at exhaustion under the loaded breathing condition. Ventilatory failure therefore should not be the major attribution of the impairment of the CON exercise sustainability in RVL. Such findings further implied that the adapted breathing pattern in obesity in response to obesity-related RVL alone during moderate exercise might not lead to unavailable ventilatory capacity and the resultant limitation in exercise tolerance. Although arterial hypoxemia has been reported previously in obesity during exercise, it might not be directly related to the obesity-related mechanical constraint on the inhaled chest wall movement. It would be a result of impaired ventilation at the lung bases due to the upward shift of the abdominal contents and diaphragm that diminished the volume change at the lower portion of the lung (Holley et al. 1967).

Wang and Cerny (2004) reported that the imposition of extrathoracic mass in nonobese subjects for stimulating obesity-related RVL could increase overall oxygen cost during graded exercise. The increased demand in whole-body oxidative metabolism was attributed to the increased work necessary for moving the extra weight on the chest. In contrast, in the present study, although the cost of breathing in subjects during exercise was being increased with the ETR, it was not revealed in the whole-body \( \text{VO}_2 \) measurement. The impairment of CON exercise sustainability in RVL therefore should not be attributed to the augmented oxidative metabolic demand. The nonsignificant change in \( \text{VO}_2 \) in RVL was in agreement with that reported previously in various types of exercise including continuous, incremental and intermittent exercise at moderate to supra-maximal intensities under either elastic or resistive loaded breathing conditions (Tong et al. 2003; 2001; Marciniuk...
et al. 1994; D’Urzo et al. 1985; Flook & Kelman 1973). It might be a result of activation of neural load-compensating mechanism involving high motor center for meeting ventilatory demand during exercise with minimal additional work of breathing (Caretti & Whitley 1998). Further, it was noted that those external ventilatory loads that did not entail significant increase in whole-body VO₂ during exercise were not composed of mass loading. The increase in whole-body VO₂ in subjects with mass loading on chest during graded exercise reported in the study of Wang and Cerny (2004) may have resulted from the additional muscular work in least relation to the ventilatory movement of the chest wall. Such findings implied that the sole factor of obesity-related mechanical constraint on inhaled chest wall movement in obesity might not cause a marked increase in oxidative metabolic demand during exercise to lessen their exercise capacity. During the moderate exercise in RVL, the ETR augmented the intensity of breathlessness sensation reflected by the RPB in subjects. It was noted previously that when the inhaled portion of chest wall excursion was impeded in a conscious person, the misalignment between the demand for the shortening of the external intercostal and accessory muscles and the actual degree of their shortening would increase the afferent spindle activity. As a result, the inspiratory drive in the motor area of the cerebral cortex was heightened, while the sensation of breathlessness in the sensory area was accentuated (Shannon 1975). In addition, afferent signals derived from stretch receptors in the lungs due to the rapid reduction in end-tidal lung volume with extrathoracic RVL might also contribute to the load-induced breathlessness sensation (Harty et al. 1999). Killian (1986) defined that the sensation of breathlessness is an internal subjective perception of ventilatory muscle effort. Fink et al. (1996) observed that the greater the efferent signal transmitted from the high motor cortex to the ventilatory muscles, the greater the neuronal corollary discharge being transmitted to sensory areas in CNS. This suggested that the conscious awareness of the enhanced motor output to the ventilatory muscles by means of collateral discharge might account for the augmented breathlessness sensation perceived in individuals under loaded breathing condition.

In RVL, mean inspiratory flow rate was increased with no change in duty cycle during the exercise. This suggested that the central inspiratory drive was enhanced for increasing the inspiratory muscle force output to work against the ETR for preserving the tidal volume in each breath (Shannon 1975). Based on the previous findings of restrictive loaded breathing, inspiratory muscle effort of the subjects in RVL might have increased to overcome at least the following:

1. The RVL-induced hyperventilation.
2. The net increase in elastic work during inspiration.
3. The additional negative work that involved eccentric contraction during the early phase of each inspiration to counteract the excess recoil force in chest generated in last expiration.
4. The increased magnitude of diaphragmatic displacement at unfavorable muscle length.
5. The augmented inspiratory muscle recruitment for the maintenance of the force generated at increased shortening velocity of the muscles reflected by the increased peak inspiratory flow rate (Wang & Cerny 2004; Hussain & Pardy 1985).

As a result, RPB in subjects was increased. Further, it was found that the Pimax in subjects measured following the exercise was generally reduced from pre-exercise value in RVL, but not in CON. The reduced post-exercise Pimax revealed that global inspiratory muscle fatigue occurred in the subjects working against the ETR during the exercise, but it did not result in ventilatory failure. It was likely that additional accessory muscles may have been recruited to maintain the inspiratory force during the exercise (Bradley et al. 1986). Therefore, subjects’ RPB increased further and at exhaustion, the RPB of all subjects were almost at the level of maximal tolerance. These values increased to 160.8% ± 35.9% (ranging from 105.3% to 200%) from the ISO.

It was further found that the increase in RPB in subjects was negatively correlated to the reduction in exercise sustainability. This accounted for >60% of the variance in the reduction in exercise sustainability. The impairment of exercise sustainability with the RVL-induced noxious breathlessness sensation was consistent with the consequences of the flow-resistive ventilatory loading in sustaining different types of
exercise reported previously (Tong et al. 2003; 2001). Gibson (2000) reported that the unpleasant breathlessness sensation on mild physical exertion is a very common symptom in the obesity. The present findings further suggested that the augmented intensity of breathlessness sensation mediated by the sole factor of obesity-related RVL in obesity during moderate exercise might contribute to the inferior moderate exercise tolerance. We cannot exclude the possibility that the rapid increase in ERS in nonobese subjects might have exaggerated the intensity of breathlessness sensation perceived. In obesity, the increase in ERS is adopted gradually and in parallel with natural adjustments in the breathing pattern (Harty et al. 1999). However, one should note that the ventilatory muscle function in obesity seems likely to be compromised both by the RVL of excessive adipose tissues and by the reduction in their functional capacity (Gibson 2000; Weiner et al. 1998). The adverse effects of such ventilatory muscle dysfunction in obesity on the augmentation of RPB and the impairment of exercise sustainability have not been counted in the present findings.

In conclusion, when a stimulated obesity-related RVL was imposed on nonobese subjects, expiratory reserve volume and residual volume were reduced, with concomitant twofold increase in the ERS. During moderate exercise with the RVL, the intensity of breathlessness sensation was dramatically augmented while the post-exercise inspiratory muscle function and exercise sustainability were impaired. The RVL-induced augmentation of intensity of breathlessness sensation, which was partly due to the increase in inspiratory effort for working against the ETR on inhaled chest movement that eventually caused occurrence of inspiratory muscle fatigue, could explain >60% of the variance in exercise sustainability impairment in subjects. Such findings implied that the perceived noxious breathlessness sensation in obese individuals mediated by the obesity-related RVL during moderate exercise, isolated from other obesity-related disorders, might contribute to their inferior exercise tolerance. Whether the enhancement of inspiratory muscle strength through specific training can benefit adherence to physical activity in obesity by improving moderate exercise tolerance and minimizing the intense sensation of breathlessness awaits further study.

References


