Effects of Obesity on Respiratory Resistance*

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To assess the effects of obesity on pulmonary function, 46 healthy subjects exhibiting various degrees of obesity underwent lung function tests. Subjects were divided into three groups according to body mass index (BMI): 13 had minimal obesity (BMI, 25 to 29 kg/m², group 1); 24 had a BMI in the 30 to 40 range (group 2); and 9 displayed to morbid obesity (BMI >40, group 3). Respiratory resistance was estimated by the forced random oscillation technique and airway resistance was determined by body plethysmography. Lung volumes and expiratory flows were also determined and significant negative correlations with BMI were found. Expiratory flows diminished in proportion to lung volumes, and the ratio of forced expiratory volume in 1 s to forced vital capacity was within normal limits. Although expiratory flows did not suggest bronchial obstruction, both respiratory resistance and airway resistance rose significantly with the level of obesity (p<0.005 and p<0.025, respectively), from 3.2 (± 0.02) and 3.2 (± 0.02) cm H₂O/τL⁻¹, respectively, in group 1, to 5.5 (± 0.06) and 5.0 (± 0.05), respectively, in group 3. Evaluation of the factors responsible for this increased resistance disclosed a significant linear correlation between airway conductance and functional residual capacity (r = 0.70, p<10⁻⁵), but specific airway conductance was found to be independent of the degree of obesity. The difference between respiratory resistance and airway resistance did not widen significantly according to the level of obesity, suggesting that chest wall resistance was not a factor enhancing these resistances. Taken together, these findings suggest that in addition to the elastic load, obese subjects have to overcome increased respiratory resistance resulting from the reduction in lung volumes related to being overweight.

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beseity is known to induce respiratory mechanical impairment that may be combined with gas exchange abnormalities. The mass loading of the ventilatory system induced by obesity, particularly on the abdominal component of the chest wall, modifies the static balance within the respiratory system. Previous studies of lung function have shown a restrictive pattern with a reduction in lung volumes that in massive obesity amounts to 20 to 30 percent for total lung capacity (TLC) and vital capacity (VC).¹ The overweight implies a heavy load for the respiratory muscles that may be involved in obesity-related respiratory complications such as exercise-induced dyspnea.²

Although a great deal of energy may be spent in overcoming abnormal chest elasticity, it only accounted for one third of the increase in the mechanical work of breathing in the study of Naimark and Cherniack.³ Several factors, in fact, may explain the resulting increase in nonelastic work. In obese subjects, the diaphragm is in the upper position, which results in a low functional residual capacity (FRC). Such modifications in resting end-expiratory lung volume may result in a passive change in airway resistance (Raw) related to an increase in transmural pressure across the bronchial wall. In addition, chest wall resistance may be increased due to obesity. An additional cause of increased respiratory resistance could also be the existence of upper airway obstruction in these obese subjects because of fat deposition or lax pharyngeal muscle tone.

The aim of the present study was to characterize the resistive properties of the respiratory system in a large number of healthy subjects who exhibited various degrees of obesity. Body plethysmography was used to determine nasal and oral airway resistance whereas the forced oscillation technique was used to determine the resistance of the respiratory system and parameters as compliance, inertance, and frequency dependence of resistance.

METHODS

Patients

Quelet's index (or body mass index [BMI]), wt/ht², in which wt is weight (kg) and ht is height (m), was the inclusion criterion for the subjects participating in this study. Obesity was defined by a BMI larger than 25.⁴

Forty-six patients were selected. They comprised 31 women and 15 men, aged from 16 to 63 years (38.7 ± 14.4 years; mean ± SD), with a height of 165 ± 6 cm (range, 154 to 180 cm). All these subjects were healthy, with no cardiac or major metabolic disorders. They did not complain of respiratory symptoms, except for the five who experienced mild dyspnea on strenuous exercise. None was a habitual snorer and none complained of diurnal or nocturnal symptoms relating to a sleep apnea syndrome (excessive daytime

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sleepiness, daytime fatigue, presence of snoring, and stopping breathing during sleep). Whenever possible, patients' bedmates were interviewed and asked to confirm the absence of snoring, restless sleep, or episodes of apnea. All were nonsmokers. Each subject was aware of the goals of the study and gave informed consent.

Different degrees of obesity were observed in these 46 subjects. According to the classification of the Panel on Energy, Obesity, and Body Weight Standard, 13 had minimal obesity (BMI, 25 to 29 kg/m², group 1), 24 had a BMI in the 30 to 40 range (group 2), and 9 exhibited morbid obesity (BMI >40, group 3).

**Spirometry**

Lung volumes were measured in the morning, in the sitting position, using a closed circuit spirometer (VG 2000, Mijnhardt BV Bunnik, Netherlands). The FRC was measured by the helium dilution method, as the mean of two determinations. Forced expiratory flows were measured using a pneumotachograph (Fleisch No. 3) and a pressure transducer (Validyne, MP45 ± 2.5 cm H₂O. Validyne Co, Northridge, Calif) connected to a microcomputer (Apple IIe). The highest values of three technically satisfactory forced expirations were taken and expressed as percentage of predicted values.⁶

**Resistances**

Respiratory resistance (Rrs) and airway resistance (Raw) were determined in each patient by forced oscillation and body plethysmography, respectively.

The forced oscillation method used has been detailed previously. Briefly, a random noise signal of 3 to 25 Hz was generated by loudspeakers and superimposed on the spontaneous breathing of the subject who was equipped with a mouthpiece and a nose-clip while firmly holding the cheeks. Mouth flow was measured with a screen pneumotachograph (Jaeger, Wurzburg, Germany) connected to a differential pressure transducer (Sensym LX 06001D, Sunyvale, Calif). An identical transducer was used to measure mouth pressure. The signals were lowpass filtered to prevent aliasing, i.e., to eliminate the possible influence of high on low frequencies, and sampled at a frequency of 128 Hz. The signals were fed into a microcomputer on which spectral analysis was performed using a 512-point Fast Fourier Transform algorithm. The real part (which is related to the resistive properties of the system) and the imaginary part (which corresponds to inercance and compliance properties) of the respiratory impedance were computed every 0.25 Hz from 3 to 25 Hz and displayed as a function of frequency. For each of these frequencies, a coherence function ranging from 0 to 1, which enables us to evaluate the reproducibility of impedance measurement, was calculated and 0.9 was chosen as the lower limit of data acceptance. The real part of impedance was submitted to linear regression analysis that yielded the Rrs extrapolated at zero frequency (Rrs), and the slope (S) of the linear relationship of resistive impedance vs frequency. As previously described, the fitting of the real part of impedance by a linear model enabled us to use the impedance value extrapolated to zero frequency as an index of Rrs during spontaneous breathing. Respiratory compliance (Crs) and inercance (Irs) were estimated by multilinear regression analysis of the imaginary part of impedance.

Airway resistance (Raw) was measured using a flow pressure-compensated plethysmograph, according to the panting method of Dubois et al.⁷ modified by Lorino et al.⁸ To determine Raw, the subjects panted at a level close to their FRC. Functional residual capacity was measured in order to compute Raw from the plethysmographic specific Raw determination.

A first plethysmographic measurement of resistance was performed with the subject wearing a nose-clip and breathing through a mouthpiece (oral Raw). A second measurement (nasal Raw) was made with the subject breathing through a nasal mask (SEFAM, France) connected to the pneumotachograph, while he was instructed to close his mouth according to the method described by Nolte and Luder-Luhr.⁹

**Statistics**

Data were expressed as means±SE. For comparison of the groups, an analysis of variance (ANOVA) was performed. When a significant difference was found, we compared individual means by a modified t test (taking into consideration the variance of the whole sample). For all comparisons, p<0.05 was considered significant. Correlations between variables were analyzed with least-square linear regression techniques.

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**Table 1—General Characteristics of the Subjects Included in the Study**

<table>
<thead>
<tr>
<th>Group</th>
<th>BMI, kg/m²</th>
<th>BW, kg</th>
<th>Height, cm</th>
<th>Age, yr</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>27±1</td>
<td>77±3</td>
<td>167±2</td>
<td>36±3</td>
</tr>
<tr>
<td>2</td>
<td>34±1</td>
<td>93±2</td>
<td>165±1</td>
<td>39±3</td>
</tr>
<tr>
<td>3</td>
<td>46±2</td>
<td>128±6</td>
<td>167±2</td>
<td>43±3</td>
</tr>
</tbody>
</table>

ANOVA p<0.0001 p<0.0001 NS NS

*BMI = body mass index; BW = body weight; values are expressed as means±SEM.

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**Figure 1.** Changes in lung volumes observed in three groups of obese subjects. Obesity was defined by the body mass index (BMI).

Group 1: subjects with minimal obesity (BMI, 25 to 29 kg/m²); group 2: subjects with a BMI of 30 to 40; and group 3: subjects with morbid obesity (BMI >40). Lung volumes are expressed in percent predicted values. TLC = total lung capacity; VC = vital capacity; ERV = expiratory reserve volume; IC = inspiratory capacity. The result of the analysis of variance is given for each panel, together with the between-group comparisons. Note the larger scale for the variations in ERV.
RESULTS

The general characteristics of the patients are given in Table 1. Patients in all three groups were comparable for age and height (ANOVA, ns), and constituted a representative group of middle-aged obese subjects.

Lung Volumes

We found a clear change in most lung volumes as the BMI increased. Figure 1 shows that TLC, VC, FRC, and expiratory reserve volume (ERV) decreased with increasing obesity. Analysis of variance demonstrated particularly highly significant reductions for FRC and ERV. A restrictive pattern, defined as a 20 percent decrease in TLC, was observed in 8 percent of the patients in group 1, 25 percent in group 2, and 56 percent in group 3. In general, the changes in lung volumes (TLC, VC, FRC, and ERV) were significantly correlated with each other: for instance, the correlation coefficient between FRC and ERV was 0.84 (p = 0.008).

Flows and Resistances

Values for the forced expiratory volume in one second (FEV₁) diminished when obesity rose, but in proportion to the reduction observed for the VC, so that the FEV₁/VC ratio was about normal (Fig 2).

Analysis of the flow-volume loop showed that expiratory flow rates at 50 percent and 25 percent of VC were significantly below normal, and decreased with increasing obesity (Fig 2). However, due to the large standard deviation, no significant difference was found between the expiratory flow rates of the three groups (ANOVA, ns).

We also observed significant increases in resistance in the obese subjects (Fig 3): thus, in subjects with morbid obesity (group 3), oral Raw was 56 percent higher than in those with minimal obesity (group 1). This increase was found to be related to the decrease in FRC, so that when the inverse of airway resistance, ie, airway conductance (Gaw), was plotted against FRC, a linear relationship was found, as shown in Figure 4. Specific conductance (sGaw) is the conventional way of taking into account the volume dependence of Raw. We found that sGaw was not dependent on the degree of obesity (Fig 4).

Nasal Raw was systematically higher than oral Raw. However, both parameters showed a similar increase with the degree of obesity: the difference in nasal Raw minus oral Raw was 1.8 ± 0.3 cm H₂O·s·L⁻¹ for group 1 and 1.7 ± 0.4 for group 3 (Fig 3).
Table 2—Effects of Increasing Obesity on Respiratory Compliance (Crs), Inertance (Ir), and the Frequency-Dependence of Resistance (Slope)

<table>
<thead>
<tr>
<th>Group</th>
<th>Crs, 1cm H₂O⁻¹</th>
<th>Ir, cm H₂O·L⁻¹·s⁻¹</th>
<th>Slope, 10⁻³ cm H₂O·L⁻¹·s⁻¹/Hz</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>27 ± 1</td>
<td>15 ± 1</td>
<td>29 ± 8</td>
</tr>
<tr>
<td>2</td>
<td>27 ± 3</td>
<td>12 ± 1</td>
<td>-21 ± 13</td>
</tr>
<tr>
<td>3</td>
<td>14 ± 4</td>
<td>10 ± 1</td>
<td>-70 ± 28</td>
</tr>
<tr>
<td>ANOVA</td>
<td>p&lt;0.025</td>
<td>p&lt;0.05</td>
<td>p&lt;0.005</td>
</tr>
</tbody>
</table>

Respiratory resistance was significantly correlated to Raw (r = 0.82, p<10⁻⁵) (Fig 5). It increased with the degree of obesity, but the trend was identical to that of Raw and no significant change in the difference between the two parameters was seen in relation to the level of obesity (ANOVA, ns): thus, the difference between Rrs and Raw, which evaluates chest wall tissue resistance, was 0.1 ± 0.3 cm H₂O.s.L⁻¹ for group 1, 0.2 ± 0.2 for group 2, and 0.4 ± 0.2 for group 3. The frequency dependence of resistance clearly increased as obesity became more marked. This parameter, the slope of the relationship between Rrs and frequency of oscillation, which has been shown to be an early marker of small airway obstruction,⁷ correlated closely with the expiratory flow rate at 50 percent of VC (FEF50): r = 0.65, p<0.0001. Analysis of the imaginary part of respiratory system impedance disclosed that as obesity increased, Crs and Ir diminished significantly (Table 2).

Discussion

Although lung volumes are known to decrease in obesity,¹ⁱ the resistive properties of the respiratory system have seldom been investigated in subjects with this disorder.¹²,¹³ In the present study, we demonstrated that Rrs increases in obese subjects and showed that this increase was mainly due to the decrease in lung volumes.

Lung Volumes

As in previous studies,¹¹,¹²,¹³ we observed that FRC, TLC, and VC dropped significantly when obesity rose. The drop in FRC was combined with a low expiratory reserve volume, as previously reported.¹⁵,¹⁶ We confirmed that the decrease in ERV is the most sensitive lung function test in obesity. Lung volume abnormalities are related to changes in chest wall compliance, since the resting end-expiratory position of the lungs, chest wall, and diaphragm is determined by the balance of the elastic recoil forces of these structures. Chest wall compliance has been measured in obese subjects and, except in one study,¹⁷ it has been shown that the pressure-volume curve of the chest wall was flattened, with a lower resting volume (FRC) than normal.³,¹²,¹⁸

By contrast, we found no abnormalities in inspira-
tory capacity. This capacity was also previously found to be normal in the obese, and a lower than normal inspiratory capacity in these patients was attributed to an intrinsic respiratory disease. 1

Residual volume (RV) has rarely been measured in the obese. Ray et al1 showed that it decreased proportionally to TLC when the obesity was minimal, but tended to be equal or even to exceed its predicted value as the obesity became more marked. Similarly, Sharp et al22 reported that in massive obesity, RV was higher than in a group of normal subjects. It may be assumed that as obesity increases toward massive overweight, the chest wall pressure-volume curve below FRC becomes flatter, which limits the action of the expiratory muscles and helps to increase RV; thus, in morbid obesity, RV does not decrease as much as TLC. In addition, a high closing volume has been found in obesity, 16,19 which may also help to account for the absence of reduction of RV. The results of this study confirmed that there is no significant reduction in RV values with increasing obesity. Our results also indicate that in obese subjects with a BMI below 40, an increase in RV may be indicative of an associated disease.

Airway Resistance

Emirgil and Sobol13 reported a decrease in Raw after weight reduction in a study of four obese patients. We found no other studies in which Raw was evaluated in obesity. Our results clearly indicate that Raw increases in obese subjects, as we observed that it was 56 percent higher in patients with massive obesity than in those with minimal obesity. In order to explain such an increase in Raw, it is tempting to hypothesize that the large decrease in resting lung volume (FRC) is responsible for these abnormalities. Indeed, Briscoe and Dubois14 showed that airway conductance (Gaw) was linearly related to lung volume. We found a highly significant correlation between Gaw and FRC in our subjects, which suggests that low lung volume is crucial in determining the increase in resistance. Airway resistance, in fact, depends on the elastic recoil pressure of the lung, which tends to increase the airway caliber at high lung volumes and to reduce it at low lung volumes when this pressure diminishes. In addition, closure of small peripheral airways16 may participate to the increase in resistance.

In this study, we also addressed the question of whether obesity in itself is a factor that increases the resistance of the nasopharyngeal airway. The anatomic patency of this segment has been clearly documented as a major variable in the pathophysiology of obstructive sleep apnea (OSA), and elevated nasopharyngeal resistance has been demonstrated in patients with OSA.20 Shepard and Burger21 recently showed that nasal flow-volume loops were highly abnormal in such patients. However, these authors pointed out that they had been unable to determine the role of obesity in these abnormalities as a factor independent of OSA, because no control obese subjects were studied. Since in the present investigation we observed no change in the difference between nasal and oral airway resistances with increasing obesity, it can be concluded that obesity alone is not responsible for increasing upper airway resistance, for instance in the retropalatal segment, which is frequently involved in OSA.22

Analysis of forced expiration showed that FEV1/VC was normal in all subjects, but that expiratory flow rates were significantly below normal, with a nonsignificant trend toward lower values as obesity increases. This points to the conclusion that in massive obesity, airway abnormalities involve a predominant increase in proximal airway resistance but only minimal distal obstruction.

Forced Oscillation Mechanics

The resistive properties of the respiratory system include not only the resistance of the airways, but also that of the chest wall. In our subjects, we measured Rrs in order to evaluate the repercussions of the alterations in chest wall mechanics resulting from obesity.

We found that Rrs increased with obesity, since in subjects with morbid obesity (group 3) it was 72 percent higher than in those with minimal obesity (group 1). This is in agreement with the results reported by Sharp et al22 that showed an increase in total Rrs in a small group of obese subjects. In obese subjects who displayed high respiratory impedance, Rrs could have been underestimated due to the upper airway artifact corresponding to airway wall motion during the forced oscillation maneuver.25 However, Peslin et al23 showed that for an index such as the resistance extrapolated at 0 Hz, this artifact has little effect.

The average values found here for Rrs were only slightly higher than the values for Raw. In the group with minimal obesity, Rrs was 0.1 cm H2O·s·L−1 greater than Raw. If this difference is considered as representative of chest wall and lung flow resistance, this value was lower than the previous estimate of 0.5 cm H2O·s·L−1 for chest wall resistance in seated conscious normal subjects.24 The discrepancy might be due to a difference in methods of measurement, since the previous evaluations were made using the esophageal balloon technique in order to partition the Rrs. In the present group with morbid obesity, the difference between Rrs and Raw was not significantly greater than for the subjects with minimal obesity, although the values tended to be larger (0.4 ± 0.2 vs 0.1 ± 0.3 cm H2O·s·L−1. These results indicate that chest wall resistance cannot explain the increase in

Effects of Obesity on Respiratory Resistance (Zarah et al)
Rrs observed in obesity, and are in keeping with the observation by Van Noord et al., who used abdominal strapping, which simulates obesity, and showed that in normal subjects, this did not alter chest wall resistance.

The change in FRC therefore appears to be the main factor explaining the increase in Rrs. The influence of lung volume on the resistance of the respiratory system has been studied in several investigations both in normal subjects and patients. In the supine position, in which FRC is lower than in the sitting position, an increase was observed in Rrs, accompanied by a decrease in its frequency dependence. Reduction in volumes also follows rib cage restriction and its effect on Rrs and reactance has been studied in healthy subjects. Rib cage strapping raised resistance, mainly at low frequencies, and this rise was reversed when the subjects breathed 1 L above the actual FRC, which corresponded to FRC in the free condition, thus demonstrating that reduced lung volume is involved in the increase in resistance.

In massive obesity, we observed a negative frequency-dependence for Rrs. Although upper airway shunt properties (upper airway wall motion during forced oscillations) might explain some of this frequency-dependence, it is mainly attributable to mechanical inhomogeneities inside the lungs. In fact, we observed a highly significant correlation between the parameters describing the flow-volume curve, such as the flow at 50 percent of VC, and the frequency dependence of resistance, indicating that airway obstruction and/or the closure of small peripheral airways occurred in these obese subjects. Subjects with airway obstruction indeed exhibit a negative frequency-dependence for Rrs, unlike healthy subjects who display no such frequency-dependence.

Measurement of respiratory compliance by the forced oscillation technique takes into account tissue compliance, airway distensibility, and gas compressibility. In our subjects, Crs dropped significantly with increasing obesity, probably because of changes in chest wall compliance. The decrease in FRC may also have been involved in this drop, since obesity causes cephalad displacement of the diaphragm that reduces FRC; this diminution induces a concomitant decrease in lung compliance, due to the shape of the pressure-volume curve, and a decrease in gas compressibility, due to the reduced pulmonary gas volume. Similar effects of lung volume on Crs have been reported when the FRC was lowered artificially.

Finally, the significant decrease in inductance observed herein with increasing obesity might be due to changes in FRC. The Ir5 takes into account not only airway and tissue inductance, but also gas inductance. The decrease in Ir5 may therefore be attributable to changes in gas inductance reflected by the decrease in FRC.

In summary, our obese subjects exhibited marked increases in Rrs that were related to the decrease in their FRC. Such increases in resistance add a breathing load to the extra load induced by the chest wall abnormalities, and might be involved in the pathogenesis of respiratory symptoms such as breathlessness described by obese patients without any clearly identified respiratory or bronchial disease.

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REFERENCES