Respiratory muscle power and lung function in healthy obese Sudanese medical students

Abstract

There is an increasing prevalence of obesity worldwide (Body Mass Index≥30kg/m²). Obesity may cause changes in pulmonary mechanics, as measured by spirometric values: mild reduction in vital capacity and proportionate reduction in Forced Expiratory volume in the first second (FEV1) depending upon the age, type of body fat distribution and severity of obesity. Changes in respiratory muscle strength in healthy obese individuals have not been widely investigated. The aim of this study is to assess the effect of obesity on respiratory muscle strength and pulmonary function in healthy obese subjects. A cross sectional study was carried out at National Ribat University, Faculty of Medicine, among ninety-five healthy male and female medical students aged 17-25 years (52 obese with BMI equals or above 30kg/m², 43 non-obese with BMI<0.05). These results were also found to be consistent in female subjects. Significant negative correlation was also observed between % fat and lung function parameters (FVC, FEV1). Increased strengthening of respiratory muscles in healthy obese adults (as measured by elevated MEP and MIP) might be a compensatory effect to bring lung function to comparable values of the non-obese. Percent of body fat could be a good predictor for decline in lung function in obese individuals.

Keywords: pulmonary, respiratory muscle power, lung function, sleep related disorders

Introduction

There is an increasing prevalence of obesity (Body Mass Index≥30kg/m²) worldwide. Data from the National Health and Nutrition Examination Surveys (NHANES ) showed that the prevalence of obesity has increased among adults aged 20-74 years in the United States from 15% (1976 to 1980 survey) to 32.9% (2003 to 2004 survey).³

Respiratory effects of obesity outside of sleep related disorders are less well known. The total respiratory compliance may be reduced to one third normal.² Although spirometric values are usually normal in patients who are obese, there may be mild reduction in vital capacity and proportionate reduction in Forced Expiratory volume in the first second (FEV1) depending upon the age, type of body fat distribution (with central fat distribution having a relatively greater effect) and degree of severity of obesity.³,⁴ The most common pulmonary function abnormality in patients who are obese is a reduction in Expiratory Reserve Volume (ERV) due to presence of adipose tissue around the rib cage and abdomen and in the visceral cavity that causes displacement of diaphragm in the thorax.⁵,⁶ Reduciton in Functional Residual Capacity (FRC) is attributed to reduced ERV. In severely obese patients there is also a decrease in Maximal Voluntary Ventilation (MVV) that may be explained by increased upper airway resistance, flow resistance and respiratory muscle inefficiency.⁴ Another study also showed that obesity can promote considerable changes in respiratory function: reduction in FRC, decrease in Tidal Volume (TV), reduced lung compliance, alveolar hypoventilation, CO₂ retention, increase in respiratory air flow resistance, alveolar hypoventilation, disturbed ventilation perfusion ratio (V̇/Q), changes in respiratory mechanics of the rib cage and diaphragm and increasing respiratory muscle work load.⁸

Evaluation of respiratory muscle strength as measured by maximal expiratory pressure (MEP) and maximal inspiratory pressure (MIP) has been used since the 1960s and 70s because of its important diagnostic and prognostic role in neuromuscular, lung and cardiovascular diseases.⁹,¹⁰ It is a quick, simple, practical, low cost and non invasive method for evaluation of both healthy individuals and those with certain chronic diseases.¹¹-¹⁵ Changes in respiratory muscle strength in healthy obese individuals have not been widely investigated.

Methods

This is cross sectional study carried out at the Faculty of Medicine, National Ribat University in Khartoum city, Sudan. Data were collected between October and December 2012. A signed and informed consent was obtained from all subjects. Fifty-two healthy obese medical students aged 17-25 years (20 males, 32 females) with body mass index (BMI)≥30kg/m² and 43 healthy non-obese (controls) aged 17-24 years (21 males and 22 females) with BMI<25kg/m² were included in the study. Both groups of subjects were clinically well, had no cardio respiratory symptoms and were non-smokers. The two groups were matched in terms of age, sex and height. Height (ht) and weight (wt) were measured for all subjects using height measures and weight scales respectively. BMI was calculated as weight in kilogram divided by height in meters squared. Percent of body fat (%fat) was measured by skin fold calibre, skin folds taken from the triceps and subcapular regions for males and from the triceps and the thighs for females. The two skin fold measures were added together and from special table schedule percent of adiposity was calculated.

Measures

Lung function (Forced Vital Capacity FVC, Forced Expiratory...
Volume in the first second FEV1, Peak Expiratory Flow Rate PEFR) and respiratory muscle pressures measured by maximal expiratory pressure (MEP) and maximal inspiratory pressure (MIP) were taken for all subjects using Digital Spirometer and Micro Respiratory Pressure Meter respectively.

**Analysis**

Variables analyzed were age, gender, ht, wt, BMI, % fat, FVC, PEFR, MEP and MIP. Data was analyzed using Statistical Package for Social Sciences version 19 (SPSS 19). Means of variables were analyzed and compared using an unpaired t-test. Level of statistical significance was set at P≤ 0.05. Pearson correlation was used for correlation of variables.

**Results**

Fifty-four females and forty-one males were included in the study (figure 1). The anthropometric measurements in obese subjects for age, height, weight, BMI and % fat were: 20.09±1.94 years, 168.08±8.78cm, 102.08±19.57kg and 35.40±5.12 respectively compared to 19.26±1.80 years, 168.16±8.22cm, 60.67±9.90kg, 21.53±4.22 and 18.64±11.10 respectively in non obese subjects (Table 1). Mean, SD for FVC, FEV1, PEFR, MEP and MIP in obese (males and females) were: 3.58±0.85L, 3.22±0.72L, 445.77±134.95L/sec, 109.02±29.76cmH2O and 78.86±17.15cmH2O respectively compared to 3.63±0.74L, 3.30±0.63L, 460.33±123.40L/sec, 94.60±27.71cmH2O and 67.81±18.70cmH2O respectively in non-obese subjects (Table 2) (Table 3). Correlation between % fat and FVC, FEV1 were -0.0332, -0.381 respectively (Table 4). Correlation between weight and MEP was 0.284 (Table 4).

**Table 1** Anthropometric parameters in the study group

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Obese (mean±SD)</th>
<th>Non-obese (mean±SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in years</td>
<td>20.09±1.94</td>
<td>19.26±1.80</td>
</tr>
<tr>
<td>Height in cm</td>
<td>168.08±8.78</td>
<td>168.16±8.22</td>
</tr>
<tr>
<td>Weight in kg</td>
<td>102.08±19.57</td>
<td>60.67±9.90</td>
</tr>
<tr>
<td>BMI</td>
<td>35.40±5.12</td>
<td>21.53±4.22</td>
</tr>
<tr>
<td>% Fat</td>
<td>28.15±11.93</td>
<td>18.64±11.10</td>
</tr>
<tr>
<td>Total (N)</td>
<td>52</td>
<td>43</td>
</tr>
</tbody>
</table>

**Table 2** Lung function parameters (FVC, FEV1, PEFR) and respiratory muscle pressures (MEP, MIP) in the study group

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Obese (mean±SD)</th>
<th>Non-obese (mean±SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC in liter</td>
<td>3.58±0.85</td>
<td>3.63±0.74</td>
</tr>
<tr>
<td>FEV1 in liter</td>
<td>3.22±0.72</td>
<td>3.30±0.68</td>
</tr>
<tr>
<td>PEFR in liter/sec</td>
<td>445.77±134.95</td>
<td>460.33±123.40</td>
</tr>
<tr>
<td>MEP in cm H2O</td>
<td>109.02±29.76</td>
<td>94.60±27.71*</td>
</tr>
<tr>
<td>MIP in cm H2O</td>
<td>78.86±17.15</td>
<td>67.81±18.70*</td>
</tr>
</tbody>
</table>

**Table 3** Lung function parameters (FVC, FEV1, PEFR) and respiratory muscle pressure pressures (MEP, MIP) in male and female subjects

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Obese (mean±SD)</th>
<th>Non-obese (mean±SD)</th>
<th>Obese (mean±SD)</th>
<th>Non-obese (mean±SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC (L)</td>
<td>4.40±0.72</td>
<td>4.07±0.70</td>
<td>3.06±0.40</td>
<td>3.21±0.49</td>
</tr>
<tr>
<td>FEV1 (L)</td>
<td>3.90±0.60</td>
<td>3.76±0.58</td>
<td>2.79±0.36</td>
<td>2.86±0.43</td>
</tr>
<tr>
<td>PEFR (L/sec)</td>
<td>551.40±142.58</td>
<td>332.12±114.17</td>
<td>379.75±76.18</td>
<td>391.77±89.21</td>
</tr>
<tr>
<td>MEP (cm H2O)</td>
<td>128.60±36.34</td>
<td>105.05±31.61*</td>
<td>96.78±15.68</td>
<td>84.64±19.26*</td>
</tr>
<tr>
<td>MIP (cm H2O)</td>
<td>82.17±16.94</td>
<td>72.21±19.45</td>
<td>72.60±11.90</td>
<td>59.00±13.01*</td>
</tr>
<tr>
<td>Total (N)</td>
<td>20</td>
<td>21</td>
<td>32</td>
<td>22</td>
</tr>
</tbody>
</table>

**Table 4** Correlation of weight & MEP, % fat & FVC and % fat & FEV1 in the study group

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Pearson correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight &amp; MEP</td>
<td>0.284**</td>
</tr>
<tr>
<td>% fat &amp; FVC</td>
<td>-0.332**</td>
</tr>
<tr>
<td>% fat &amp; FEV1</td>
<td>-0.381**</td>
</tr>
</tbody>
</table>

**Discussion**

The present study demonstrates the relationship of pulmonary function and respiratory muscle strength with obesity in a group of young healthy Sudanese medical students. In this study a significant increase was observed in both MEP and MIP measurements in obese subjects when compared to non-obese control subjects (P value < 0.05). The results were also found to be consistent in female subjects, but in male subjects only MEP showed a significant increase. Controversy exists in different studies where some have found a positive correlation of MEP and MIP with obesity, while others have not.

The results obtained in the present study agreed with some previous reported studies. Queiroz conducted a study on obese and non-obese subjects; the author found that MIP was significantly increased in obese groups when compared to non-obese individuals, indicating that obese subjects had higher respiratory muscle strength. Cordoso studied the apparent correlation of respiratory muscle strength with normal weight in obese women, observing that respiratory muscle strength was higher in obese women, but noting that the difference was insignificant when compared to normal weight subjects. Neder et al., Domingos-Bernicio et al., found similar values of respiratory muscle strength (MEP/MIP) in groups of obese and normal weight subjects. Magnani & Cataneo verified in their study that obesity does not impair respiratory muscle strength in obese individuals aged 20-60 years, since MIP values did not achieve significant differences when compared with reference values established by Neder et al., in normal subjects.
Tanner et al.,24 investigated the type of muscle fibers present in obese subjects by means of biopsy from rectus abdominis muscle. The author found a higher percent of type II fibers that related to high muscle strength.24 Shonberg et al.,25 stated that weight affects maximal respiratory pressures (MEP, MIP) variables due to an increase in the size and volume of respiratory muscles that is seen with an increase in body weight, consequently improving the strength of the muscles.26 It has been found that muscles of obese individuals have specific and metabolic characteristics due to daily physical effort to move the loaded chest, for that obese individuals have higher proportion of skeletal muscle mass and type II fibers.24

This study reports that there was no difference in lung volumes (FVC, FEV1, PEFR) between obese and non-obese subjects, a fact which can be explained by the compensation with the increased respiratory muscle power in obese subjects. This observation is also supported by the positive correlation of MEP with body weight, however, a negative significant correlation was detected between lung function parameters (FVC and FEV1) and % of body fat (P value <0.05). This may imply that the amount of body fat might be related to lung function through a mechanical impact on the diaphragm (impeding its descent into the abdominal cavity) and on the chest wall due to changes in compliance and work of breathing. There was no correlation of body mass index with lung function in the present study and this may imply that evaluation of the change in lung volumes in obese subjects might best be achieved by estimation of body fat percent rather than body mass index. Therefore, percent and distribution of body fat can be a good predictor of decline in lung function more than body mass index in overweight subjects.

Conclusion

The significant increase in MEP and MIP in obese subjects observed might be a compensatory effect, in order to align lung function to comparable values of non-obese individuals. Additionally, the percent of body fat could may be a good predictor for decline in lung function in obese individuals. Further study is warranted.

Acknowledgments

My deepest gratitude and appreciation to Chairman of Board of Trustees and all workers and administration of Elsheikh Abdallah Elbadri University and to Prof. Omer A/Aziz Musa.

Conflict of interest

Medical Physiology.

References